The revival of heart rate

See European Heart Journal Supplements Suppl. H which accompanies this issue

Nowadays, ‘modern cardiologists’ and, in general, MDs, deal with complex theories and sophisticated approaches in their clinical practice: they rely on elaborate diagnostic tools which have, through the years, succeeded in identifying and evaluating those more hidden parts of the hearts and vessels of their patients.

As a result, so-called bedside medicine is no longer a daily clinical practice when compared to ‘high tech’ medicine. Equally, evidence-based medicine has ruled out intuitive medicine.

The aim of the European Heart Journal Supplement which accompanies this issue is to amaze modern cardiologists with an ancient and apparently banal concept, i.e. the heart rate. In our daily activities, we are all concerned with using the best available technique to ensure the proper diagnosis, prognosis and therapeutic intervention. Palpation and auscultation are no longer used. Imaging is the preferred manner of diagnosis, and therefore we often forget to measure heart rate in our patients. Who does this any more?

It is a pity that this method is no longer used, as ‘taking the pulse’ has always been the first point of contact between doctor and patient and succeeds in reassuring and gaining the confidence of the patient. Besides its value as an easy patient approach, measuring the heart rate also has scientific worth, as we all know that a fast or an extremely slow heart rate is an unequivocal sign of a ‘suffering’ heart, as is frequently shown in romantic literature, paintings and music. This unsophisticated sign can be considered the means of communication chosen by the body to ‘speak’ to the doctor. Heart rate is not only the language of romantic times, it also seems to be the language of our modern high tech times, since change in heart rate is the language chosen by the body to communicate with its own cells. It has recently been suggested that the heart rate corresponds to the rate of energy needed by the body. There are theories hypothesizing that the body controls its own metabolic rate and energy needs by altering its heart rate. In fact, heart rate controls nitric oxide release from the endothelium through shear stress. Nitric oxide, in turn, regulates the degree of vasodilatation and thus the amount of blood supplied to the muscles. In this way, heart rate contributes to the metabolic needs of the muscles and, therefore, to their metabolic rate.

Not only this! Heart rate negatively affects the metabolic rate of the heart itself, thus negatively influencing its own ‘supplier’! It is surprising to realize that a reduction in heart rate of 10 beats min\(^{-1}\) day\(^{-1}\) saves 5 kg of ATP! In addition, an increase in heart rate of 5 beats min\(^{-1}\) corresponds to an increase in the atherosclerosis progression score of 0.21 and to an increase in the stenosis progression score of 0.27!

By analysing heart rate variability one can properly assess autonomic tone, better appraise the control of sinus node functionality and, in general, evaluate the function of the entire body. You may be surprised to learn that animal studies show that life expectancy is related to heart rate! Take a look at these examples: a rat from the pyramids has a heart rate of about 240 beats min\(^{-1}\) and lives no longer than 5 years, while the life expectancy of a tortoise from the Galapagos islands, which has a heart rate of 6 beats min\(^{-1}\), is 177 years!

Please, don’t take your pulse now! We know that human beings are exceptions. We have relatively fast heart rates which correspond to relatively long life expectancy. Why? Changes in the social environment, as well as an improvement in scientific and, more specifically, therapeutic tools have played a role in increasing life expectancy. We know, for instance, that drugs which reduce cardiovascular mortality,
such as beta-blockers and some calcium-antagonists with electrophysiological activity, also reduce heart rate. Therefore, could heart rate reduction be the simple explanation for the amazing results of ‘complicated’ large clinical trials on mortality?

Thus, let’s bring back heart rate — not only as a risk factor for cardiovascular patients (and the general population) — but also as a specific therapeutic target for ischaemic heart disease, heart failure and hypertension.

For this to be meaningful, the concept of heart rate is proposed to you in modern terms in all the topics of this European Heart Journal Supplement. We hope you enjoy reading this Supplement as much as we enjoyed preparing it. All the papers have been peer-reviewed and we would like to thank Roche for making this supplement possible.

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Sympathetic overactivity and exercise intolerance in heart failure: a cause–effect relationship

See page 880 for the article to which this Editorial refers

During the past 15 years a large amount of new data has been collected on the behaviour of the sympathetic nervous system in congestive heart failure. In most studies two main methodological approaches have been used to quantify adrenergic cardiovascular influences, i.e., (1) radiotracer-derived measurement of noradrenaline spillover, reuptake and clearance in the systemic circulation as well as in the main cardiovascular regional districts and (2) direct recording of efferent post-ganglionic sympathetic neural discharge to skeletal muscle vessels. These approaches have allowed the studies to establish that the state of sympathetic overactivity characterizing the heart-failure syndrome (1) is an early phenomenon in the clinical course of the disease, an increase in sympathetic nerve traffic being clearly detectable not only in patients belonging to the New York Heart Association functional class III or IV, but also in those belonging to the New York Heart Association class I or II[1,2], (2) involves cardiovascular districts such as the coronary, the renal, the cerebral and the muscular[1–4], the only exception to a rather generalized sympathetic activation being represented by an unchanged sympathetic neural outflow to the cutaneous circulation[5,6], (3) is inversely related to haemodynamic indexes of the myocardial inotropic state, such as cardiac stroke volume or stroke work, and (4) is usually coupled with, and probably caused by, a profound dysfunction of the reflex mechanisms (arterial baroreflex and cardiopulmonary reflex), which powerfully restrain sympathetic activity in physiological conditions[2,7].

Conclusive evidence has also been provided on the pathophysiological and clinical relevance of this phenomenon. Although initially representing a compensatory mechanism which allows tissue perfusion and cardiac output to be maintained, sympathetic overactivation with disease progression aggravates the heart failure condition by a number of adverse cardiovascular effects[7,8]. These include (1) an increase in the oxygen and metabolic demands of the myocardium, (2) a reduction in the myocardial oxygen supply, (3) augmented sodium and water retention, (4) a decrease in the myocardial arrhythmogenic threshold, and (5) a direct necrotic effect on the myocardium. This accounts for the evidence that plasma noradrenaline levels, and also cardiac noradrenaline spillover values, are inversely related to patients’ survival[9,10].

The study by Notarius and co-workers[11], published in this issue, expands this evidence by showing that in heart failure sympathetic overactivation also contributes to the exercise intolerance that is a common hallmark of this condition. In the study’s population, oxygen consumption at peak exercise was inversely related to resting muscle sympathetic nerve