

EDITORIALS

Atrioventricular Conduction Versus Heart Size From Mouse to Whale*

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In normal sinus rhythm, the atrioventricular (AV) conduction system introduces an appropriate delay between atrial and ventricular excitation and contraction (1,2). This delay, the AV or PR interval, increases with the size of the animal and, thus, of the heart (3,4). This increase, however, is relatively small in comparison with the difference in the sizes of the body and the heart. For instance, the PR interval is only twice as long in mammals such as the elephant or the whale as it is in human beings (4,5). Comparison of the AV nodal electrophysiologic properties with the size of the heart in a number of mammalian species raises rather interesting questions regarding the functional properties of the AV conduction system.

Comparative Function of the AV Node-His System

The behavior of the AV node-His bundle system has been studied systematically using conventional programmed atrial stimulation in rats, dogs and human beings (6-12).

1. AV conduction time or delay after atrial premature stimulation can be described by an exponential function of the coupling interval between the last normally conducted R wave and the stimulus (10,11).
2. Adaptation of AV conduction delays to stepwise atrial rate changes is a phenomenon dependent on time rather than on the number of cardiac cycles (10-12).
3. Atrial rate-induced changes in AV conduction time show a short time constant (10-12) or memory, with memory being the time the AV conduction system needs to adapt to changes in PP (AA) intervals (10). For example, in human beings, AV memory does not normally persist beyond one or two cardiac cycles.

*Editorials published in *Journal of the American College of Cardiology* represent the views of the authors and do not reflect the views of JACC or the American College of Cardiology.

From the Department of Cardiology, University Hospital, Utrecht, The Netherlands. This work was supported by the Wynand M. Pen Foundation, Utrecht. Manuscript received June 18, 1984, accepted September 6, 1984.

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4. The ratios between normal AV conduction time (atrial-His interval or PR interval) during sinus rhythm and the durations of the AV memory in rats, dogs and human beings are roughly similar (10).
5. Species-dependent differences in AV conduction delay during sinus rhythm and AV conduction adaptation after pacing-induced atrial rhythm changes are small, taking into account the differences in relative sizes of the respective animals and their hearts.

Comparative Morphology of the AV Node-His System

Despite differences in detail, the overall architecture of all mammalian hearts is essentially similar. One familiar with the anatomy of the heart of one species will have no great difficulty identifying cardiac structures in other mammals. Nature has used the same "blueprint" for all mammalian hearts, be it mouse or whale. This similarity applies generally to the morphology of the mammalian AV node-His system as well. Although differences such as the blood supply or the presence of an os cordis do exist between the AV nodes of, for instance, rabbits, dogs and human beings on the one hand and cattle on the other, the fact remains that the known similarities significantly outweigh the differences (13-15). The overall configuration of the mammalian AV node-His system from the AV region to the bundle branches is also similar among the smaller (16) and larger (17-19) mammals.

Although most studies tend to stress the differences, the microscopic and electron microscopic characteristics of the mammalian myocardial and AV nodal cells in the species studied to date show remarkable similarities (20). For the electron microscopist, it is difficult to differentiate between myocardial or AV nodal cells derived from the rabbit, dog or human subject (21). It is reasonable to expect that this is also true for the horse, elephant and whale. Thus, macroscopically and microscopically, the structural arrangement of the mammalian AV conduction system tends to be similar, if not uniform, while the size of the heart varies greatly from species to species.

Size Versus Function of the AV Node-His System

According to Schmidt-Nielsen (3), the weight of the mammalian heart is about 0.6% of the body weight. From comparative anatomic studies (13,22), it appears that the size of the mammalian AV node increases with the size of the heart, although not proportionally. For instance, the ratio between the weight of the heart of a rat, a human being and a medium-sized whale is approximately 1:300:60,000. When we accept the PR interval of the mammalian species (4,5) as a measure of AV conduction time and plot these values against the body mass (3), we find a gross disproportion between these two sets of data (Fig. 1). This presents a most interesting and still unexplained discrepancy between size and function of the mammalian AV conduction system.

Conduction Velocity in the AV Node-His System

Living systems must function at relatively uniform temperatures and pressures (23). The mammalian biologic system functions at about 37°C. Cardiac muscle is composed of individual cells that tend to be uniform in diameter, approximately 10 to 15 μm , whether the source is the mouse or the whale (24). This small range of mammalian myocardial fibers may reflect an optimal relation between cell volume and diffusion rate (25). From present knowledge, it is reasonable to assume that the same physicochemical conditions are applicable to mammalian conduction fibers.

From the rabbit to the elephant, the metabolic rate per gram tissue decreases only slightly (26). Conduction velocity depends largely on cell (fiber) diameter (27,28). Assuming a more or less constant cell to cell resistance, it is unlikely that with increasing length or diameter of the His bundle and bundle branches, the known conduction velocity of approximately 2.5 m/s (29) will increase significantly.

These assumptions suggest that in a large mammalian heart, such as that in the elephant (30) or whale (31), the contribution to the AV conduction delay by each component of the AV conduction system must be different from that of the heart in smaller mammals such as the human being, dog or rabbit. For example, in the adult blue whale having bundles that are likely 1 m or more in length from their origin at the AV node to their terminal ventricular ramifications and a conduction velocity of about 2.5 m/s, it will require at least 400 ms for the impulse to cover that distance. The PR interval in the elephant or a small whale does not exceed 400 ms (30-32). Even in the largest whales, the PR interval will not be much longer than 500 ms (Fig. 1). Therefore, the AV node, although anatomically present in large mammals, would not be expected to impose a significant delay on AV conduction during normal sinus rhythm, even if the conduction velocity was greater than 2.5 m/s.

During atrial fibrillation or flutter, however, the presence of the AV node may yet be vital in large mammals to protect the heart against an unduly rapid rate of ventricular responses (33,34). This may be especially true in large mammals with large stroke volumes because they need sufficiently long RR intervals for adequate diastolic ventricular filling.

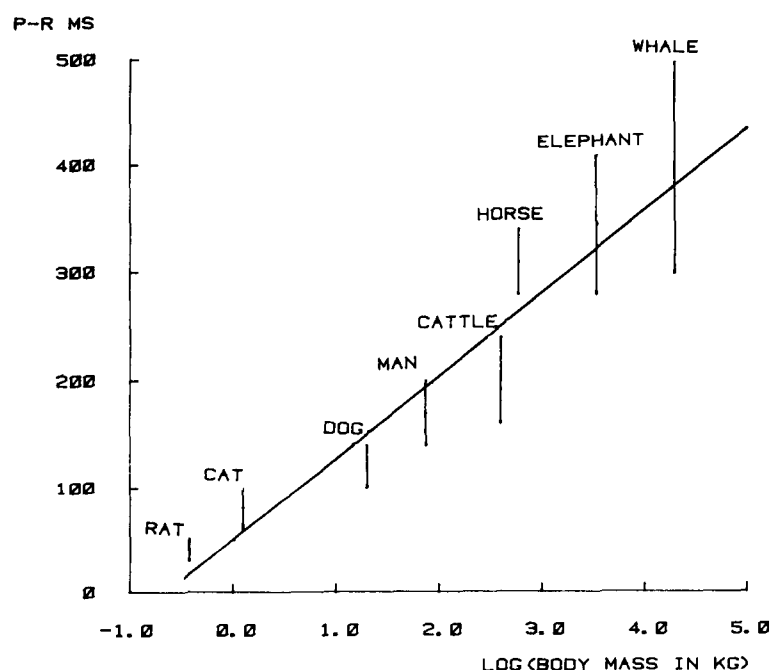


Figure 1. Approximation of the relation between body mass in kg (abscissa) and PR intervals in ms (ordinate) according to: $y = 52.5 + 77.5 \times \log(x)$. In mammals smaller than 200 g, this formula may not be applicable.

Implications

An important lesson from a comparative biologic point of view seems to be that nature has maintained in all mammals the anatomic macro- and microstructure of the heart and its conduction system, but has adapted the function to enable the heart to meet the hemodynamic demands under normal as well as abnormal conditions. The tendency has been to study biologic functions in animals smaller than human beings. Had we studied larger species, our concepts about the form and function of the human AV conduction system in health and disease might have been quite different (35). A more general conclusion may also be drawn, namely, that evolution may be represented by form and structure of the mammalian heart, but survival of the species depends on proper adaptation of the function of the heart in health and disease.

The help, encouragement and criticism of many colleagues are highly appreciated. This paper would never have materialized without their active support.

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