Request to Rat Colony Curators

To the Editor:

This letter is a plea to curators of rat colonies to supply us with a basic description of the resting heart rates and body weights of animals within your colonies. The urgency of this request is briefly summarized as follows. The results of recent studies of colonies of animals specially bred to become hypertensive have suggested that increased heart rate is an important factor in the genesis of the observed hypertension. The animals with spontaneous hypertension have resting heart rates which are significantly greater than the resting heart rates seen in other colonies of rats that do not become hypertensive. The observations are complicated, however, by the fact that normotensive control animals have body weights that are greater than the spontaneously hypertensive animals, and the results must be interpreted according to the dictum that heart rate increases with decreasing body size. There are little data available that describe resting heart rates in rats as a function of age and mature body size.

This is a request, then, to all scientists currently using rat colonies to send to us information describing: (1) the genetic background of their colony; (2) resting heart rate, body weight, and age of animal when the data were collected, for as many different ages as possible; (3) an estimate of the normal systolic blood pressure in the mature animal; and (4) comments on any extenuating circumstances that might prevent the data from being typical, such as environmental stress, unusual temperatures, or malnutrition. This information is greatly appreciated and will hopefully be used to maximum advantage. Two final comments: (1) Readers interested in helping but not having active colonies, please pass this request along to those who can respond. (2) All responders will be sent copies of any final compilations.

THOMAS G. COLEMAN, Ph.D.
Associate Professor
The University of Mississippi Medical Center
2500 North State Street
Jackson, Mississippi 39216

H-V Intervals in LBBB

To the Editor:

Several authors have indicated that in LBBB the H-V intervals appear to be longer than in absence of this conduction disturbance. The subdivision made by Rosen, Ehsani, and Rahimtoola into “normal,” “intermediate,” and “prolonged” H-V intervals is most interesting.

I believe that some of the “intermediate” H-V intervals (measured to the onset of the QRS complex in at least three surface leads) do not necessarily indicate an associated conduction delay through the right branch. We have recently been evaluating the usefulness of close (1 mm apart) bipolar (filtered) electrograms from a stable position in the right ventricular apex (RVA). When bundle-branch block was not present, the H-RVA interval gave a measure of conduction time through His bundle, right branch, and that part of the ordinary ventricular

![Diagram of electrograms](image)

**Figure 1**
Acute “complete” LBBB. H-RVA interval maintained pre-block value; H-V interval increased by 12-20 msec.
myocardium located between the site of impulse emergence (from the right branch) and the recording electrodes. In acute “complete” RBBB with normal H-V intervals the H-RVA interval was significantly prolonged since it now represented conduction time from His bundle to left ventricular endocardium, and from the latter site to the RVA (across the septum in a left-to-right direction). In some cases of acute “complete” LBBB in which the H-RVA interval maintained the preblock value, the H-V interval increased by 12–20 msec. This is shown in figure 1. The first QRS complex without a “complete” LBBB morphology had a small q wave in lead I. The corresponding H1-V1 and H1-RVA1 intervals measured 50 and 70 msec, respectively. In the second beat (with a “complete” LBBB pattern) the H-V increased to 70 msec but the H-RVA remained unchanged, indicating that a conduction delay had not occurred through the right branch.

These findings corroborate studies performed from surface electrocardiograms,6 in revived human hearts,7 and the Elizari preparation.8 For instance, the left-sided schematic in figure 2 indicates that the preblock H-V interval represented conduction time from His bundle to left ventricular endocardium. On the other hand when “complete” LBBB occurred, the H-V interval increased slightly because of the (normally) longer conduction time through the right branch, without necessarily implying the presence of an additional conduction delay within this structure.

Probably the 20-msec increment of the H-V interval is artifactualy long (due mainly to the incorrect assessment of the onset of the QRS complex). Nevertheless, the importance of the H-RVVA interval warrants further studies of the RVA electrogram as already is being done in some laboratories.9

AGUSTIN CASTELLANOS, JR., M.D.
University of Miami Section of Cardiology
Department of Medicine
School of Medicine
Miami, Florida

References


The authors reply:

To the Editor:

Dr. Castellanos’ data suggest that the prolonged H-V intervals frequently noted in patients with left bundle-branch block partially reflect a slightly longer conduction time in the right bundle-branch system. This is in keeping with Durrer’s demonstration that the onset of left ventricular activation precedes that of the right by 5–10 msec (Castellanos’ reference 7).

These observations contrast with our experience in patients with rate-dependent left bundle-branch block. In two previously reported cases,1 and in four cases studied recently (Rosen KM, Wu D, Denes P, Dhingra R: Unpublished data), H-V intervals were similar during normal conduction and left bundle-branch block. These results suggest that left bundle-branch block does not unmask a longer conduction time in the right bundle branch.

The apparent conflict between our data and that of Castellanos may reflect patient-to-patient variability in the degree of asynchrony between onset of left and right ventricular activation. Further electrophysiologic studies, with endocardial mapping, should clarify the patterns of normal and abnormal ventricular activation.

KENNETH M. ROSEN, M.D.
Professor of Medicine
Chief, Cardiology Section
The Abraham Lincoln School of Medicine
Chicago, Illinois

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AGUSTIN CASTELLANOS, JR.

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