individuals is evidently extremely small. It can be shown to be impossible for any individual to be nearest neighbor for more than 5 other individuals.

These proportions of nearest neighbor relationships prove not to be restricted to random distributions. They may also occur in populations marked by a considerable degree of aggregation or clumping. For example, as is shown in Table 1, similar proportions were found to exist in populations of the plants *Liatris aspera* Michx., *Lespedeza capitata* Michx., and *Solidago rigida* L. occurring in an old field on the E. S. George Reserve, Pinckney, Mich. All of these have been shown to exhibit marked clumping tendencies (*4*). It is evident, therefore, that these nearest neighbor relationships are not affected by all types of departure from randomness. The distributional forces responsible for the aggregation in these plant populations seem not to have influenced their nearest neighbor properties. The most important factors in their distribution appeared to be the patchiness of environmental conditions and the particular methods of reproduction and dispersal of each species.

It is clear, however, that the nearest neighbor properties of populations will respond readily to social forces affecting distribution. Any social force that promotes the formation of pairs or of other relatively small groups will tend to increase not only the proportion of reflexive relationships but also the proportion of individuals that serve as nearest neighbor to 1 or more other individuals. When these proportions are greater than expected, one can conclude that in some way the individuals have responded positively to one another.

These observations suggest a way of separating the consequences of social forces from other factors affecting the spatial patterns of distribution in biological populations.

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Direct Recording from the A-V Conducting System in the Dog and Monkey

Allen M. Scher

Department of Physiology and Biophysics, University of Washington School of Medicine, Seattle

The functional properties of the cardiac purkinje tissue, like those of many anatomical entities, are known mainly from deduction and indirect methods. Older physiologists recognized that there is a delay between auricular and ventricular activity, and also that a bridge of special tissue connects these two divisions of the heart. They rightly concluded that the electric impulse is confined within this tissue during at least part of the delay. Lewis (*1*) in his work on

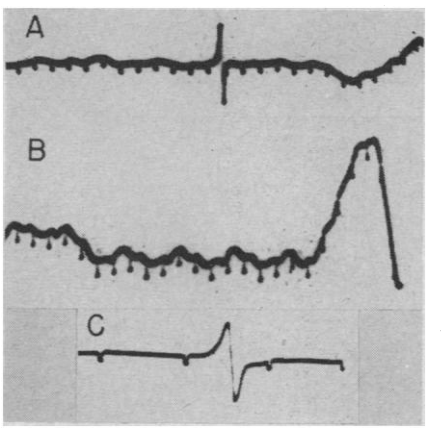

Fig. 1. Electric activity in conducting bundle of dog. (*A*) Records from electrode high in mid-interventricular septum under valve ring. Potential in center is from the bundle; slower potential changes at right show negative (receding) followed by positive (approaching) activity resulting from activation of ventricular myocardium. (*B*) Simultaneous Lead II ECG. P wave appears at left and QRS complex at right. It can be seen that biphasic bundle potential occurs about midway between auricular and ventricular activity. (*C*) Same potential as *A*, but faster sweep speed. Time pips were delivered simultaneously to both channels at 5-msec intervals. Bundle potential on *A* and *C* has peak-to-peak value of 3 mv.

the excitation of the heart correlated the peripheral distribution of the purkinje fibers in the ventricle with his findings relative to the sequence of cardiac excitation. Further, he and others found that cutting peripheral ramifications of the conducting system or destroying the A-V node caused changes in excitation. Recently Draper and Weidemann (*2*), using intracellular microelectrodes, have given an excellent picture of the electric characteristics of the portions of the conducting system that may be easily removed and studied, the false tendons. However, direct study of the tissue in the intact heart, particularly of the portions buried in the interauricular and interventricular septa, has not previously been available.

During a continuing study of the pathway of normal ventricular activation, we had often taken records of excitation of the false tendons which cross the ventricular cavities, and it seemed possible to record from portions of the conduction system nearer to the A-V node and from the node iself.

Available apparatus consisted of a multichannel oscilloscope and multipolar needle electrode. These and the associated techniques have been described (*3, 4*). The multichannel apparatus facilitated scanning of many loci in a search for potentials. In the open-chested dog, exploring electrodes were inserted into the interauricular septum anterior to the coronary sinus in the area where Tawara (*5*) has located the A-V node. Other electrodes were pushed into the interauricular and interventricular septa along the pathway of the conducting bundle and its branches and into the apical ventricular cavity where preter-

minal branches of the conduction system constitute the false tendons. These procedures have been successful in four dogs and one monkey.

The potentials from the region of the A-V node proper require a detailed and critical study, which is not within the compass of the present paper. They have varied markedly in certain respects. At times an electrode in this region records a local auricular depolarization followed by nodal depolarization, but at other times the former is lacking. Moreover, the records thus far do not permit any statement regarding the nature of A-V nodal delay or the mechanism of activation of the node.

An electrode a few millimeters from the A-V node records a small potential, usually biphasic, of about 2-msec duration and 5-mv amplitude (Fig. 1). This potential appears about 10 msec after the last sign of auricular activity and precedes the earliest sign of ventricular activity by about 40 msec. The potentials from the intraventricular false tendons containing the preterminal branches of the conduction system have similar characteristics, but precede earliest myocardial ventricular activity by only a few milliseconds (Fig. 2). If the direct distance between electrodes near the node and the false tendon is divided by the time required for impulse transmission, the calculated conduction velocity falls between 1.4 m and 1.7 m/sec. Since the impulse does not follow a straight path, the actual velocity must be greater. Thus, it appears that the conduction rate in the more central part of the system is nearer the 2.0 m/sec rate calculated for transmission in the false tendon (*2*) than the 1.0 m/sec found (*4, 6*) in the lining of most of the ventricular wall. When this system is stimulated in either a forward or a retrograde direction, the transmission time is identical to that during the spontaneous beat. There is no appreciable delay between activation of the terminal portions of the conducting system and activation of the ventricular musculature.

This work (*7*) is being continued with the hope that the difficult problem of the nature of A-V nodal delay and excitation will ultimately be understood.

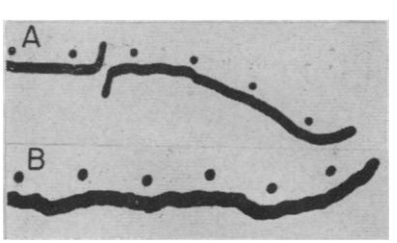

Fig. 2. Activity in false tendon of monkey. (*A*) Records from electrode that pierced false tendon in lower left ventricular cavity. Biphasic potential at left is from false tendon. Negative potential at right is caused by activity proceeding away from left cavity. (*B*) Simultaneous Lead II ECG. Time pips 5 msec. Biphasic potential on *A* has peak-to-peak value of 8 mv; it is followed in 7.5 msec by negative cavity deflection on that channel. ECG on *B*, 2.5 msec later, shows negative (Q) deflection followed by positive deflection.

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6. This velocity is found with stimulation. Normally the endocardium is excited at many points simultaneously by the branched purkinje system. Thus, the apparent velocity is high.

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Dosage Effect of Multiple *Dt* Loci on Mutation of *a* in the Maize Endosperm

M. G. Nuffer

Department of Field Crops, University of Missouri, Columbia

The mutability of certain alleles at the *A* locus on chromosome 3 in maize is affected to a remarkable degree by the gene *Dt* located on chromosome 9 (*1*). The mutations produced are changes of *a* to *A* that can be expressed, when the appropriate complementary genes are present, as long, slender, purple sectors of (*Aa*) cells surrounded by otherwise green or brown (*aa*) plant tissue and as frequent small purple or red (*Aaa*) dots on the colorless (*aaa*) aleurone of the seed.

In an effort (*2*) to find other genes similar to *Dt*, 98 strains of maize from Central and South America were tested. Included were 1 from Argentina, 5 from Bolivia, 6 from Brazil, 17 from Colombia, 2 from Costa Rica, 11 from Ecuador, 4 from Guatemala, 7 from Honduras, 4 from Mexico, 7 from Nicaragua, 31 from Peru, and 3 from Venezuela. The test consisted of backcrossing *Aa* heterozygotes, to an a^m *dt* tester and examining the ears produced for colorless seeds with red or purple dots (reversions). The a^m allele in the stock used is extremely sensitive to the action of *Dt* (*3*), and therefore it provides an excellent indicator of the presence of possible weak expressions of *Dt*. Two of the races tested gave positive dotting: a Brazilian yellow flint variety called Cateto and a purple aleurone race from the Peruvian coastal village of Huarmey.

A number of investigations were made for the purpose of locating and characterizing these two possible *Dt* genes. Tests for their allelism with Dt_1 and with each other showed that they are independent loci. They were, therefore, designated Dt_2 (from Brazil) and Dt_3 (from Peru). Preliminary linkage data from the original backcross indicate that Dt_2 is linked to *Y* (*Y* produces yellow endosperm and *y* white) on chromosome 6 with 26-percent recombination. Linkage data are not yet available for Dt_3. An effort was made, using the x-ray induced deficiency method (*4*), to confirm the location of Dt_2 and to place both Dt_2