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Arrhythmias

Leonard S. Dreifus, M.D. | Guest Editor

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Editor's Commentary

The diagnosis and management of arrhythmias have always commanded a prominent interest among clinicians. However, the advances made during the past decade probably outstrip those of any prior 10-year interval. Our understanding of the physiology of atrioventricular and intraventricular conduction has been expanded immeasurably by the development of electrode catheter techniques for recording bipolar electrograms of the A-V node, bundle of His, and the right bundle branch. Concomitantly, the diagnosis and clinical significance of bilateral bundle branch block as well as divisional blocks of the left bundle have won new recognition. The advent of intensive coronary care has, of course, played a prominent role in focusing attention on the nuances of diagnosis and the intricacies (and rewards) of selective management. Pacemakers have achieved an integral role in the therapy of both brady- and tachyarrhythmias; and important new antiarrhythmic drugs have emerged during the past decade. Each of these important advances, and others as well, are elaborated in the present volume. I know that the readers of this issue of CARDIOVASCULAR CLINICS will share my gratitude to the authors and Guest Editor for their lucid clinical reports.

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Contents

Anatomy Related to Atrioventricular Block*

Raymond C. Truex, Ph.D.

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A structural basis for the cardiac arrhythmias and heart block has been extensively investigated in the last 50 years, and a voluminous literature has accumulated on this subject. As one might suspect, this accrued information contains many established facts, a fair number of misconceptions, and a sprinkling of morphological errors. The present paper is designed to elucidate some of the current anatomical knowledge of the conduction system and to correlate the pertinent aspects with normal and abnormal cardiac conduction.

One essential point is emphasized: namely, that the intrinsic conduction system is composed of modified cardiac muscle. The initiation and propagation of the cardiac impulse are properties residing in these specialized muscle units. The conduction system of the heart is *not* made up of nervous tissue. There are intimate relations between the autonomic ganglia and the sinoatrial (S–A) and atrioventricular (A–V) nodes, and numerous fascicles of nerve fibers accompany the atrioventricular bundle and its branches. The central and peripheral neural elements of the autonomic nervous system can and do exercise profound influence on heart rate, conduction velocity, myocardial contraction, and even the genesis of cardiac arrhythmias. However, such neural regulation is only superimposed on the inherent or autonomous functional properties of the muscle elements that comprise the conduction system. Indeed, the muscle fibers of this unique system can maintain synchronized conduction and cardiac function after complete experimental autonomic denervation and heart transplantation.[1–4] More will be said about cardiac innervation after some of the gross and microscopic features of the conduction system are considered.

The conduction system is indicated in black in Figure 1. Histologically,

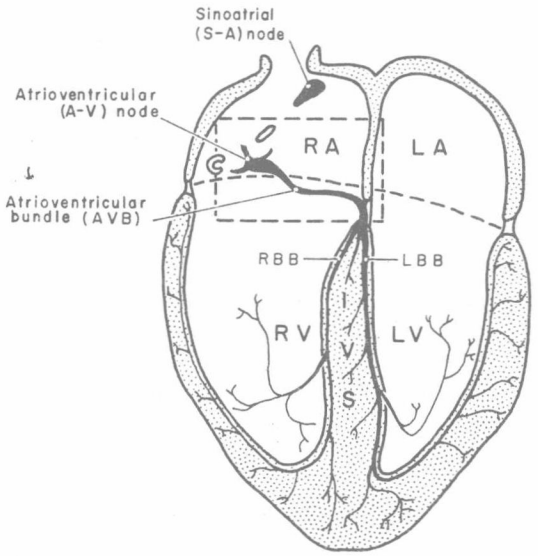

Figure 1. Diagram of cardiac conduction system. Specialized cardiac muscle of the nodes and bundle branches (LBB, RBB) are black. Note endocardial to epicardial distribution of terminal bundle branches in the ventricles (LV and RV). Rectangular dotted lines indicate area enlarged in Figure 2.

some of the muscle fibers of this system can be identified in the posterior wall of the A–V canal in human embryos of 12 to 15 mm. C–R length. At this stage of development, closure of the interventricular septum is well advanced but still incomplete. Therefore, it is not surprising that conal and cushion derivatives from this region in the adult heart display fibers that may resemble the conduction system (e.g., large atrial fibers in the posterior leaflet of the tricuspid valve; Purkinje fibers related to the chordae tendineae). After partitioning of the heart into its four chambers, there is normally only one point of continuity between the atrial and ventricular myocardium, provided by the A–V bundle (common bundle of His). Accessory atrioventricular bundles have been demonstrated in fetal and adult human hearts, and these may play a role in the genesis of the Wolff-Parkinson-White syndrome.[5, 6] However, in the hearts of the higher mammals, only the A–V bundle normally persists.[7] The junction zone between the A–V node and A–V bundle is the site of fibrotic disruption, usually responsible for complete congenital heart block.[8–12]

GROSS RELATIONS OF THE CONDUCTION SYSTEM

Figure 1 depicts schematically the course and relations of the bundle and bundle branches within the subendocardium of the ventricles and the muscular interventricular septum (IVS). The distribution and pattern of the left and right bundle branches account for the sequential ECG activation of the interventricular septum, right ventricle, apex, lateral walls, and base of the ventricles. Note also that the smaller branches are distributed from endocardium to epicardium and that their course may be lengthened in myocardial hypertrophy.

Several macroscopic relationships at the A–V junction merit additional consideration, for this zone often proves to be the "Achilles heel" in cardiac conduction. The gross relations of the A–V node and bundle to atrial structures, the annulus fibrosus and tricuspid valve, are illustrated in Figure 2. These relations have been demonstrated in man by careful microdissection at high magnifications.[13–15] In gross dissections, the A–V node lies in the lower interatrial septum, approximately a millimeter beneath the endocardium and a thin sheet of cardiac muscle fibers (Figs. 6, 7). It appears as a thin, grayish mass of tissue (4 x 6 x 1.5 mm.) enmeshed in connective tissue, nerve fibers, and small blood vessels. In dissected specimens, extensions of the node can be followed only for short distances into the adjacent atrial myocardium. On its distal deep surface, the A–V node is firmly attached to the right fibrous trigone by the fascicles of the A–V bundle, which then pass through the trigone to reach the top of the interventricular septum along the posterior and inferior margin of the membranous septum. The relationships shown in Figure 2 become crucial indeed when intracardiac surgical procedures are anticipated, e.g., placement of a prosthetic aortic valve, closure of septal defects, or ligature of the A–V bundle for uncontrollable tachycardia.[16, 17] The endocardial

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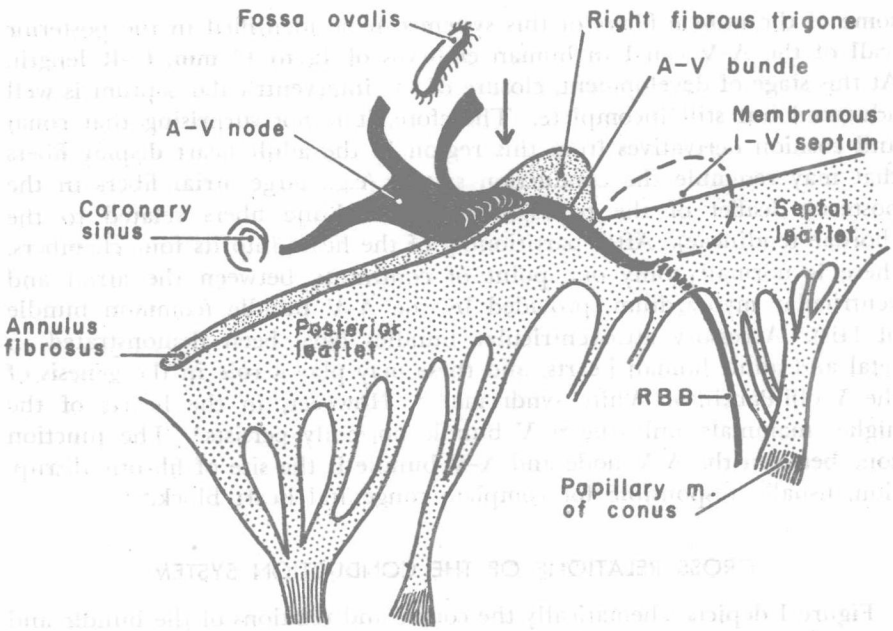

Figure 2. Diagram of the gross relationships of the A–V node and bundle at the right atrioventricular junction. This is a highly vulnerable zone in cardiac conduction. Arrow indicates plane of sections through right fibrous trigone shown in Figures 6 and 7A.

position of the A–V node and its atrial extensions also accounts for the electrocardiographic changes so frequently encountered during atrial and coronary sinus catheterization. Attention is called to the descending course of the small right bundle branch deep to the septal leaflet. It most commonly passes posterior to the base of the papillary muscle of the conus, en route down the interventricular septum to the base of the anterior papillary muscle or to the moderator band if present.

The terminal relationship of the A–V bundle (arrow), aorta, and adjacent structures when viewed from the left side of the interventricular septum can be seen in Figure 3. Note the broad, pale sheet of left bundle branch fibers lying below the membranous septum. In this specimen, the left bundle promptly divided into a small anterior (A) and larger posterior division (P). These divisions become more superficially placed as they descend in the endocardium on the interventricular septum.[15] In Figure 3, several small fascicles of conduction fibers are shown to leave the posterior division and septal wall to span the left ventricle as free fascicles en route to the papillary muscles. It is obvious that a small patch of endocardial fibrosis might interrupt completely the right bundle branch (Fig. 2), whereas a plaque of similar size would only interrupt a portion of the broad left bundle or one of its subdivisions. Induration and shortening following disease of the adjacent aortic valve cusps or anterior

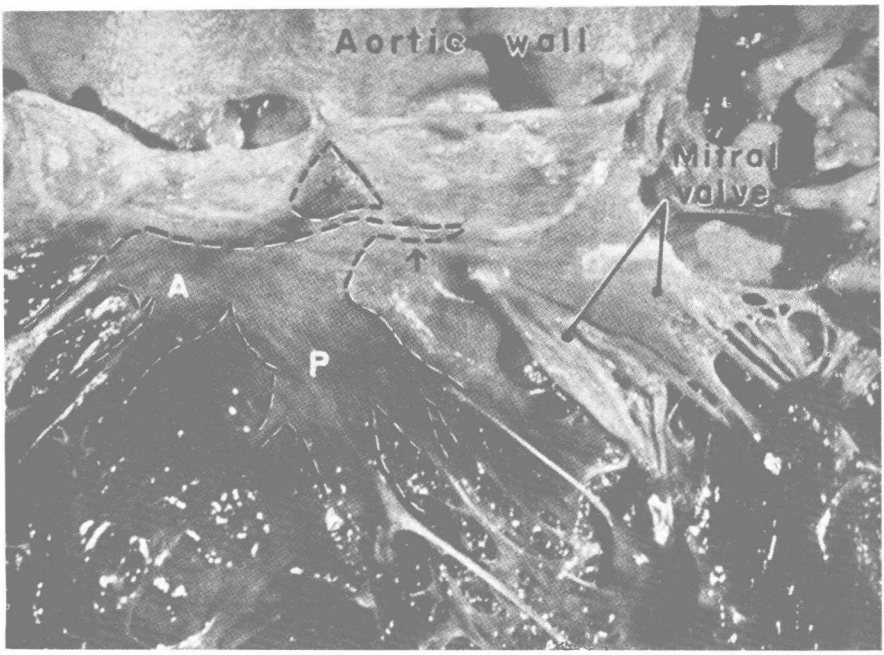

Figure 3. Left ventricular relations of the A–V bundle (arrow) and broad left bundle branch to the membranous interventricular septum (*), aortic cusps, and anterior leaflet of the mitral valve. The anterior (A) and posterior (P) divisions of the left bundle branch are identified.

mitral leaflet can also lead to conduction abnormalities by tension, compression, or encroachment on the A–V bundle and left bundle branch.

BLOOD SUPPLY OF CONDUCTION SYSTEM

The specialized muscle fibers of the conduction system are more resistant to ischemia than are atrial and ventricular cardiac muscle, yet the former will cease to function properly during prolonged oxygen deprivation. In nerve tissue, irritative symptoms often precede complete nerve tissue destruction. In similar fashion, atrial flutter or tachycardia resulting from coronary atheroma may be followed in time by myocardial infarction. Hence, brief mention of the arterial supply to the conduction system is essential, for alterations in the electrocardiogram often reflect the region of infarction. The arterial supply of the nodes and bundles is shown diagrammatically in Figure 4. Variations in the coronary vascular pattern of man do occur, and these have been reviewed by James,[18] Truex,[19] and Baroldi and Scomazzoni.[20]

In the most frequent pattern of distribution, the S–A node receives its blood supply from a small branch that arises near the stem of the right coronary artery. According to James,[21] the S–A node artery also

provides most of the arterial supply to the preferential or internodal atrial pathways. The A–V node and initial portion of the A–V bundle receive their major blood supply from the coronary vessel that supplies the crux region. The crux is a region on the posterior surface of the heart. Here the four heart chambers and the interatrial and interventricular septi are all approximated in one small area. In over 80 per cent of male and female human hearts, the posterior interventricular artery is a continuation of the right coronary artery.[18] The A–V node artery may be a single vessel (Fig. 7A) or divide into two or three smaller branches that accompany the A–V bundle through the right fibrous trigone. As indicated in Figure 4, the A–V bundle has a dual arterial supply from both the A–V nodal artery and one or more perforating branches of the anterior interventricular artery. In its descending course, the anterior interventricular artery gives rise to 8 to 15 anterior septal branches.[20, 22] These arteries perforate the septum at right angles to the epicardial surface and traverse the middle of the interventricular septum to anastomose with shorter septal branches of the posterior interventricular artery. Smaller terminal branches of the septal arteries supply the subendocardial zone of both ventricles. In human hearts, most of the ventricular septum (65 to 100 per cent) is dependent on branches of the left coronary artery. The left and right bundle branches of the conduction system are chiefly supplied by the anterior septal arteries and their small endocardial branches (Fig. 4).

Such arterial distribution explains altered conduction in the S–A node,

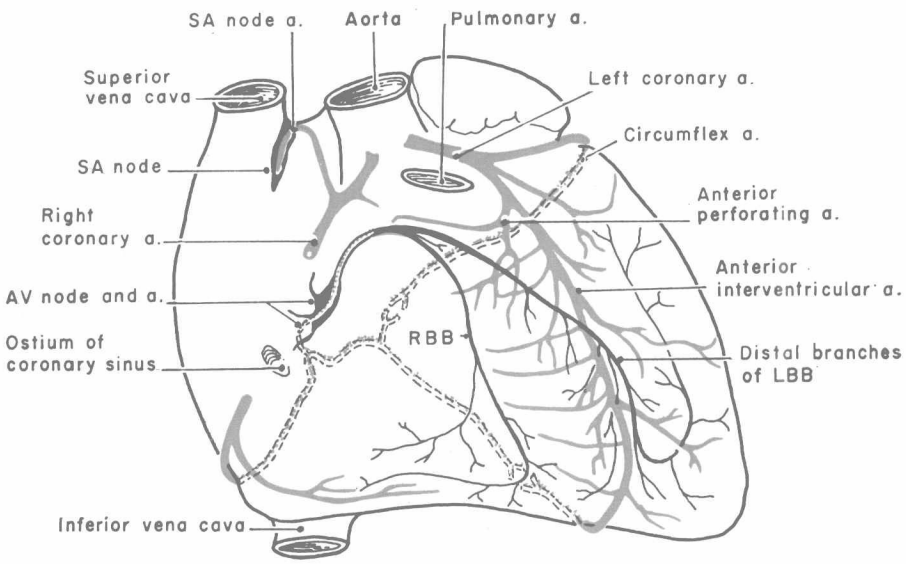

Figure 4. Diagram of anterior view of heart, showing the most common blood supply to the cardiac conduction system. An anterior segment of the right coronary artery is removed; vessels on the posterior surface of the heart are dotted.

atrium, and A–V node that often accompanies arterial disease in the right coronary artery. Arterial disease of the left coronary and its branches are most commonly associated with altered conduction in the atrioventricular bundle and its left and right bundle branches. Baroldi and Scomazzoni [20] concluded that stenosing coronary arterial disease started most frequently in both the left and right coronary arteries at the same time (58.7 per cent), less frequently in the left coronary (32.6 per cent), and only rarely in the right coronary artery (8.7 per cent). The single coronary vessel most frequently involved in atherosclerosis is the anterior interventricular artery,[23] and this observation corresponds well with the higher incidence of anterior versus posterior myocardial infarctions.

The venous drainage of the myocardium has been reviewed by Truex and Angulo [24] and Baroldi and Scomazzoni.[20] It need only be pointed out that numerous thin-walled venous channels draining the upper portion of the interventricular septum accompany the A–V bundle back to the A–V node (Figs. 5, 7A, 9). Veins from the more posterior part of the septum

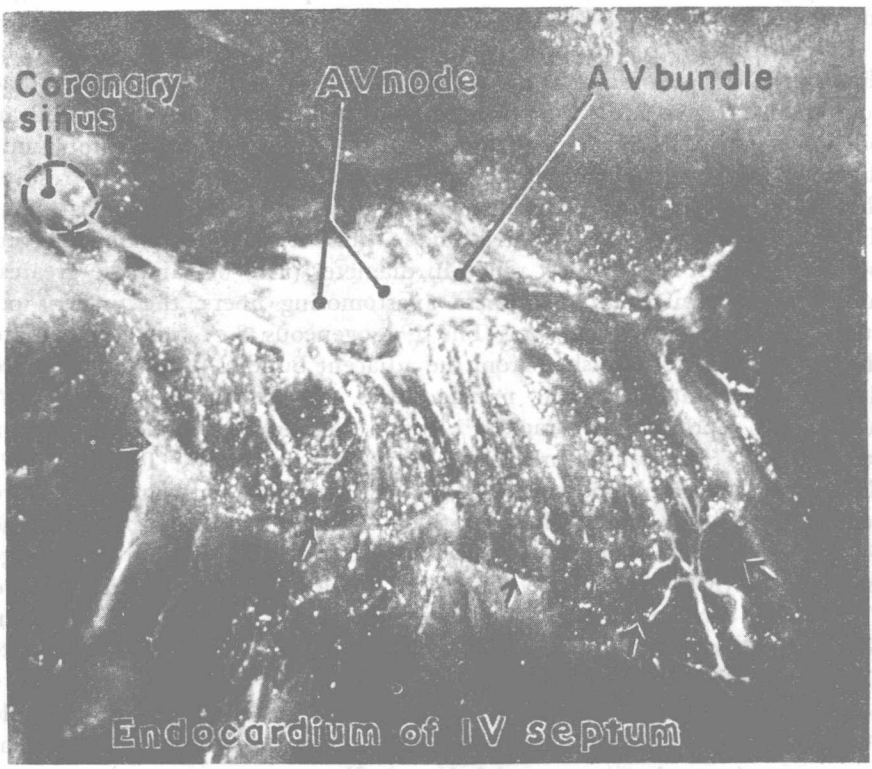

Figure 5. Dissection of human right atrioventricular junction after latex injection of the coronary sinus and its tributaries. Endocardium of the interventricular septum (arrows) and some cardiac muscle fibers were removed to expose the numerous septal veins that traverse the A–V bundle and A–V node en route to the coronary sinus.

cross the annulus fibrosus individually to join a subendocardial network in the A–V node area before terminating in the coronary sinus.[25] The tortuous and thin-walled venous radicals can be readily injected and visualized by microdissection in the human heart (Fig. 5). Conceivably, this venous network may play a role in the dissemination of blood-borne toxins and account for the unexplained hemorrhage observed in the A–V node and often associated with acute A–V block.

MICROSCOPIC CONSIDERATIONS OF THE CONDUCTION SYSTEM

The specialized muscle elements of this system vary in histological appearance, diameter, and fiber arrangement as one examines the hearts of different species.[7, 26–28] Variation in the size and arrangement of fibers also exist in individual specimens of the same species, including man. Failure to appreciate these variations has led to many contradictory statements in both the early and the current literature. It is probably fair to surmise that future investigators will perpetuate the practice by attempting to make all specimens of a given species conform to a rigid morphological mold.

In an attempt to determine the magnitude of the variations in fiber diameter, we have recently measured 100 fibers of each of the cardiac components in five adult human specimens. An ocular micrometer and an oil immersion objective ($\times 960$) were used to obtain the mean cytoplasmic diameter and nucleus-cytoplasmic ratio of each type of fiber. The results, shown in Table 1, thus represent 500 measurements in each fiber category. The small fibers of the sinoatrial node present the most homogeneous population with the smallest range in diameter (3 to 6μ) and the greatest nucleus-cytoplasmic ratio. In these anastomosing fibers, the nucleus occupies most of the cytoplasm. The homogeneous S–A nodal fibers are therefore easily distinguished from the adjacent bundles of large and more darkly stained atrial cardiac muscle fibers. Studies of the human S–A node were reported by James,[29, 30] and a human S–A node was reconstructed in a four-color, three-dimensional wax model by Truex et al.[31] These authors should be consulted for the pertinent anatomic literature related to the S–A node. Fibers of the A–V node, when compared to the S–A node, have a larger mean cytoplasmic diameter (7.5μ), a greater range in fiber size (3 to 12μ), and a somewhat smaller nucleus-cytoplasmic ratio (Table 1). The mean fiber measurements and range of the fibers in the A–V bundle and bundle branches should be noted, for they refute the often stated fact that these two segments of the conduction system are composed of a homogeneous fiber population, i.e., uniformly large, pale Purkinje fibers. The bundle and bundle branches contain small fibers that may approximate the diameter of A–V node fibers.

Some of the gross relations, as well as information in Table 1 relating to the A–V node and A–V bundle, are depicted diagrammatically in Figure 6. This figure illustrates that the conduction tissue is indeed a

Table 1. Diameter of human cardiac fibers

	Cytoplasm *	Range *	Nucleus-cytoplasmic ratio
Sinoatrial node	5.0	3.0– 6.1	0.68
Atrial cardiac muscle	15.8	7.2–30.9	0.37
Atrioventricular node	7.5	3.0–12.3	0.51
Atrioventricular bundle	10.9	5.6–21.6	0.42
Purkinje fiber of BB	23.4	11.8–49.9	0.33
Ventricle cardiac muscle	15.2	6.6–29.8	0.35

* Values of cytoplasm and range expressed in microns.

continuous system of muscle fibers, yet it consists of homogeneous and heterogeneous fiber populations in juxtaposition. A look at the mean diameters in Table 1 illustrates that as one proceeds along the conduction system, beginning in the S–A node, the fibers are small, large, small, large, large, small. No definitive study has yet been made that correlates all the specific fiber sizes with their cardiac conduction velocities. However, it is interesting to speculate, for it has been stated [32–34] that conduction velocities within these same fiber populations are slow (0.05 m. per second), faster (0.8 to 1.0 m. per second), slow (0.05 to 0.1 m. per second), faster (0.8 to 2.0 m. per second), faster (2.0 to 4.0 m. per second), slow (0.3 to 1.0 m. per second). In other words, there are junctional zones or areas of convergence between the large and small fibers of the system, e.g., nodal-atrial, atrial-nodal, nodal-nodal, nodal-A–V bundle, A–V bundle-bundle branches, and Purkinje-ventricular. The atrial-nodal junctional zones constitute areas of conduction delay and are considered to be inhibited by acetylcholine and enhanced by epinephrine. The atrionodal junction zones are also considered by some to be the site where first-degree heart block occurs.[35, 36]

Atrioventricular Node

The fiber arrangement within the human A–V node and A–V junction has been extensively studied and illustrated.[15, 27, 37–40] In brief summation, it may be stated that the A–V node is a thin, compact structure composed primarily of small anastomosing nodal fibers, arranged around one or more arteries and veins (Figs. 6, 7, 8). In the center of the node, one can usually observe cut fiber bundles oriented in longitudinal, oblique, and transverse planes (Fig. 7B). Most of the atrial fibers decrease in size and become continuous with nodal fibers along the superior and endocardial margins of the A–V node (Fig. 6). However, on occasion, a few large atrial fibers may penetrate into the compact A–V node before blending with the smaller nodal fibers (arrows in Fig. 8). It is possible that such penetrating atrial fibers with large nuclei and triangular junctional zones formed by anastomosing nodal fibers may represent the P cells described by James and Sherf.[41] These authors also described large, clear cells in and around the A–V node of man, which they believed to have Purkinje

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