Venous System of the Myocardium with Special Reference to the Conduction System

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Myocardial veins and sinusoids of the right atrioventricular junction of the heart were studied in serial section, and their relations to the common conduction bundle and nodal tissues were anatomically demonstrated by wax model reconstructions. It is suggested these venous channels may well explain the complete, but transient, bundle block of children observed in certain toxic diseases. On the other hand, the same sinusoids functioning as capillaries may nourish the bundle tissues in older decades, thus explaining the low incidence of bundle block in cases of gradual coronary occlusion. The possible role of these vessels to the intercoronary circulation is discussed.

The Authors, long interested in the conduction system of the heart, found that few recent attempts have been made to evaluate and correlate the research findings from the several scientific fields of interest. In the present study they have therefore attempted further to correlate certain well known clinical observations on the heart with lesser known facts to be found in the journals of anatomy, physiology, and pathology.

In the recent literature Glomset, Glomset and Birge have denied the morphologic existence of a conduction system in the hearts of the ungulates, dog and man, whereas the studies of Truex and Copenhaver, Nonidez, Walls, Robb, Kaylor and Turman, Stotler and McMahon, and Baird and Robb have presented convincing structural evidence that a specialized conduction system exists in the hearts of these animals.

In the pursuit of these studies on the conduction tissue in young and adult animals, the present authors were impressed by the large number of irregular venous sinusoids which either accompanied or traversed the common bundle of conduction tissue. We have observed such vessels in serial sections of the hearts of five adult dogs, 10 newborn puppies, seven human fetuses (from four to eight months of gestation), and two human newborn.

Special attention was directed toward these venous channels, for we believe they may serve as avenues for the dissemination of circulating toxins into the common bundle in certain diseases. Indeed, it is not uncommon to find reports in the clinical literature of common bundle block in diphtheria, scarlet fever, rheumatic fever, endocarditis, streptococcus infections, uremia, hemachromatosis, epidemic parotitis, and amebic hepatitis. Although Reid noted two cases of permanent heart block 19 and 42 years after diphtheria, most of the patients included in the above reports had transient common bundle blocks which lasted only a few days. These authors explained the attendant heart blocks as due to circulating toxins, myocardial pathology of a vascular nature, pathologic infiltration of the common bundle or increased vagal tone.

Brody and Smith found heart lesions of varying severity in over 90 per cent of the hearts studied after scarlet fever and related streptococcus infections. Three widespread lesions were described which were primarily associated with the endothelium of the capillaries and veins. The most common lesion was a lymphocytic and plasma cell infiltration beneath the ventricular endocardium, or about the endothelium of the coronary veins and thebesian vessels. Inasmuch as the venous channels involved are closely related to the common bundle of conduction tissue as it passes through, and courses superior to, the fibrous ring of the right atrioventricular orifice, a more detailed anatomic description of these vessels is in order.
The all-inclusive term "thebesian veins" is a most unfortunate one, for it does not denote a precise morphologic entity and is currently used by many to describe all venous channels exclusive of the cardiac veins and coronary sinus. In the latter sense it is a "system" consisting of both myocardial veins and sinusoids. Both of these venous channels are abundant and anastomose with each other freely at the right atrioventricular junction where they are intimately related to the common bundle of conduction tissue.

The sinusoids are extremely irregular in shape, with numerous dilations. Ocular micrometer measurements of the sinusoids in newborn puppies demonstrated luminal diameters ranging from 18 to 106 microns although most of the sinusoids were 40 to 50 microns in diameter. Similar measurements in the adult dog hearts demonstrated sinusoidal diameters of 40 to 75 microns (range 18 to 150 microns), while most of the sinusoids of the human fetuses were between 60 and 90 microns (range 30 to 175 microns). Although the luminal diameters of these myocardial sinusoids attest to their large caliber and capacity to transport blood, it must be remembered that they are dilated in some areas and constricted in others. For example, in an adult dog we have observed the same sinusoid in one serial section with luminal diameters of 21, 54 and 85 microns respectively.

The larger venous channels possess all the histologic features of thin-walled veins, whereas the sinusoids consist of nothing more than a single layer of endothelial cells. This is well demonstrated in figure 1, which shows a vein

**Fig. 1 (left).** Myocardial sinusoids crossing the fibrous ring of heart enroute to the coronary sinus (CS). RA, cavity of right atrium; LV, myocardium of left ventricle. Human fetus of 4.5 months cut in sagittal plane. C. R. 160 mm. Hematoxylin and eosin. × 200.

**Fig. 2 (right).** Same specimen as figure 1 with higher magnification to demonstrate endothelial walls of three myocardial sinusoids (VS). CT, conduction tissue; FR, fibrous ring; LV, left ventricle. × 420.
below the coronary sinus (CS) in contrast to three sinusoids located immediately above the fibrous ring. The endothelial nature of these sinusoids is shown under higher magnification in figure 2.

 Inferiorly the venous channels are continuous with similar vessels of the myocardium of the interventricular septum and adjacent portion into the right atrial cavity as shown in figures 3 and 4; anastomose with the epicardial veins on the posterior surface of the left ventricle; or, as is most common, empty into the coronary sinus.

 In order to demonstrate more clearly the intracardiac relations of these venous sinusoids to the conduction tissue, the authors made pro-

![Diagram](http://circ.ahajournals.org/)

**Fig. 3 (left).** Myocardial sinusoids inferior to the fibrous ring in the interventricular septum (IS). Other sinusoids are to be seen superior to the fibrous ring at its junction with the interatrial septum (IAS). RV, right ventricle; TV, tricuspid valve; CB, common bundle tissue in interventricular septum. Human fetus of 6 months cut in frontal plane. C. R. 210 mm. Azan stain. × 100.

**Fig. 4 (right).** Same specimen as figure 3 with higher magnification. Note thin endothelial walls and variation in size of the myocardial sinusoids (VS) which are partly or completely filled with blood. After anastomosing with the larger sinusoid in this field the sinusoid in the upper right area is seen to open into the right atrial cavity immediately superior to the tricuspid valve. CT, conduction tissue lying above the fibrous ring. × 350.

of the left ventricle. These vessels ascend in the inner myocardium or subendocardial region to cross the fibrous ring in intimate relationship with the common bundle as pointed out above. Other thin-walled veins have been observed as they crossed the fibrous ring independently, and then anastomosed freely with sinusoids accompanying the conduction tissue.

 Superior to the fibrous ring the sinusoids may terminate in either of three ways: directly injection drawings and wax model reconstructions from the serial sections of two human fetuses. Drawings of these wax models are shown in figures 5 and 6.

 Human hearts of this age frequently fail to demonstrate a well differentiated conduction system, but one can determine the common bundle in serial sections by its course, related structures, and lighter staining reactions.

 It will be noted in figure 5 that three veins
were selected crossing the fibrous ring and included in the model, while only two were incorporated in the model shown in figure 6. In both instances many additional arterial and venous vessels were present in the serial sections but were omitted to facilitate the mechanics of model construction and preserve simplicity.

It will be noted in figure 5 that the venous sinusoids (VS) form a wide anastomotic channel as they course through the main bundle of conduction tissue to terminate superiorly in the right atrial cavity and the coronary sinus.

A similar but more extensive sinusoidal network (VS) is shown within the common bundle in figure 6. This model, made from the heart of a 6 month old fetus and comprising 90 serial sections, demonstrates an elaborate anastomotic pattern between sinusoids and large communicating veins which ultimately terminate in the coronary sinus. Although not included in these two models we have also observed anastomoses between the sinusoids, capillaries, and terminal arterioles.

The authors were impressed by the number of autonomic ganglion cells frequently observed in close association with these sinusoids, often embedded in the substance of the atrioventricular node itself. Such neurons were observed as individual cells scattered along the atrioventricular junction, or more commonly as small discrete ganglions. These relations are particularly well demonstrated in figure 7 which is a drawing of the conduction tissue at the juncture

*Fig. 5. Drawing of a wax model reconstruction showing the course and relationships of three veins which cross the fibrous ring, traverse the substance of the common bundle as sinusoids (VS) and terminate in the coronary sinus and lumen of the right atrium. † indicates junction of fibrous ring and endocardium of right atrium. Human fetus of 4.5 months. C. R. 160 mm.*
ure 7 it is not difficult to conceive of toxic substances having ready access to either of these two essential elements, both of which are capable of producing marked changes in the electrocardiogram.

**DISCUSSION**

Anatomically the common bundle of conduction tissue has a closely related network of irrespective proof of this statement must await the outcome of further investigation which is now in progress.

Further proof that such venous channels are involved in scarlet fever has been presented by Brody and Smith.\(^20\) It is not surprising that these authors demonstrated their sharpest lesion (see figure 2 in their paper) surrounding an endothelial sinusoid at the atrioventricular junction. Whether discrete lesions in this area are consistent we cannot say. In similar manner we do not know the pathologic nature of the lesion, for to the best of the authors' knowledge, this area of the heart has not been studied in an extensive series following toxic diseases. It is hoped that future clinical-pathologic studies and experiments now being undertaken will cast some light upon the nature of the lesion.

The natural question that comes to mind is,

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**Fig. 6.** Drawing of a wax model reconstruction showing course and relationships of two myocardial veins which cross the fibrous ring, form a sinusoidal network (VS) within the substance of the common bundle, and after anastomoses with numerous communicating veins terminate in the coronary sinus. \(\dagger\) indicates junction of fibrous ring and endocardium of right atrium. Additional sinusoids within the bundle (not shown in the model) anastomosed with the communicating veins at the point indicated by an asterisk (*). Human fetus of 6 months. C. R. 210 mm.
can such a venous sinusoidal system play an active role in normal myocardial circulation? Affirmative experimental evidence that such is the case has been presented in the excellent studies of Batson and Bellet. These authors performed several interesting perfusion experiments in the dog utilizing minute glass beads, carborundum powder, and commercial flake graphite. Their results demonstrated that venous pressures within the range of pathologic heart conditions could cause a reversal of flow in the heart veins. In a similar way they found that a falling arterial pressure and an increased intra-atrial pressure result in a reversal of flow on the venous side with recovery of the graphite suspension in the coronary sinus, coronary and cardiac veins. As a result of their experiments simulating double coronary artery occlusion they further conclude, "With a low pressure in the capillary bed, flow in the veins during atrial systole is probably toward the capillary bed, the capillary bed being emptied during the ensuing ventricular systole. In arterial occlusion this would give a flow and ebb circulation for the myocardial capillaries by way of the veins."

Thus the sinusoidal system which is implicated in the common bundle block of infectious childhood diseases now appears to assume an active beneficial role in the older decades in which arterial occlusion becomes most prevalent. Although this role of the myocardial sinusoids is beyond the scope of the present communication, we wish to emphasize their relation to the common bundle and to the problems of
intercoronary circulation. For a more complete discussion of the coronary arteries and veins, the reader is referred to the fascinating papers of Wearn and his colleagues,33–36 Kugel,37 Meek, Keenan and Theisen,38 Moore,39 Batson and Bellet,32 Gross and Kugel,40 Gregg and Shipley,41 Stenstrom,42 Vineberg and Niloff,43 Beek and co-workers,44–51 and Helmsworth and associates.42

In figure 8, we have rearranged a schema based on the work of Wearn, Mettier, Klumpp and Zschiesche to demonstrate the relations of the described sinusoids and common bundle (see figures 5 and 6) to the remainder of the cardiac circulation. Solid lines represent the results of the above authors, while the broken lines indicate vascular communications described by the present authors. We are confident the myocardial sinusoids of Wearn and his associates are identical with the sinusoidal system related to the common bundle above the atroventricular junction and we have so indicated in figure 8.

We have drawn the dotted arrows to indicate structural termination of the myocardial sinusoids in the cardiac veins, coronary sinus and right atrium. If, however, one is aware of the tremendous arterial, capillary, sinusoidal and venous anastomosis in the myocardium, it is not improbable that under certain cardiac conditions the blood flow might well be in a reverse direction to that now indicated by the broken lines in figure 8. For example, it is interesting to speculate that such a reversal of flow may account for the low incidence of heart block observed following the gradual occlusion of the coronary arteries. It is equally possible that such sinusoids, functioning as capillaries, account for the favorable results and low incidence of heart block in experimental animals following arterIALIZATION of the coronary sinus.42,51 Heart block is not frequently encountered after the above procedure in dogs even though the major portion of the left coro-

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**Fig. 8.** Schema modified after Wearn, Mettier, Klumpp and Zschiesche.35 See text for discussion. Solid lines and arrows indicate above authors work, dotted lines represent results of this investigation.
and the coronary sinus. In these acute experiments the heart continued to beat for at least two hours, but with a gradual fall in blood pressure. Postmortem examinations uniformly revealed extensive hemorrhage throughout the entire thickness of the right ventricular wall. The left ventricular wall was similarly affected except that its inner third and the ventricular septum appeared normal. The normal appearance of atria, left ventricle and basal septum led these authors to conclude that these portions of the heart may have a separate venous drainage system, possibly involving thebesian channels. Similar areas of anastomoses at the junction of septal and ventricular myocardium were demonstrated in the dog experiments of Batson and Bellet (1962) (see figures 4 and 5 of these authors).

Further experiments are now being undertaken in this laboratory in an attempt to determine the site of selective action of certain toxins, as well as the major communications of the larger venous channels.

SUMMARY

Myocardial veins and sinusoids have been anatomically demonstrated at the atrioventricular junction which are intimately related to the common bundle of conduction tissue and atrioventricular node. The morphologic structure and distribution of such venous channels indicate that they may well play important physiologic roles in certain pathologic conditions.

It is suggested that the thin-walled venous sinusoids may serve as avenues for the dissemination of toxins into the common bundle in man, and thus explain the transient heart blocks observed in certain childhood diseases. The relationships of conduction tissue, autonomic ganglia and vascular structures are demonstrated and discussed.

It is further suggested that these venous structures may play a significant role in intercoronary circulation in the older decades in man. They may also explain the low incidence of common bundle block following gradual and acute occlusion of the coronary arteries.

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