Anatomy of the Coronary Arteries in Health and Disease

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ANATOMY is perhaps the simplest of medical sciences, requiring little more than some curiosity plus careful observation of things as they are. A common mistake in thinking of the value of anatomy in medicine is to consider it as an isolated, purely descriptive exercise. It is no accident that in medical school curricula anatomy is usually one of the first subjects taught. From this foundation the student should then be introduced to physiology as living anatomy and to pathology as the anatomy of the disease.

This presentation is a review of some personal anatomic studies of the coronary arteries. With the accumulation of experience it is now possible to correlate certain findings that did not at first seem related. This perspective serves to increase the pragmatic value of the separate findings by making their interrelationship more clear. In such a presentation one must arbitrarily draw certain limits on what will be covered. For introductory purposes the normal anatomy of the human coronary arteries will be briefly described, followed by a discussion of certain selected aspects of coronary disease.

Anatomy of the Coronary Arteries

Courses of the Left Anterior Descending, Left Circumflex, and Right Coronary Arteries

The left coronary ostium, which is 3 to 5 mm. in diameter, originates from the left coronary sinus of Valsalva. The main left coronary artery lies in epicardial fat between the body of the left atrium and the pulmonary artery, and curves anteriorly about the latter vessel toward the anterior interventricular sulcus (fig. 1). After a usual length of 1 to 2 cm. it divides into two or more branches. The branch that enters the left atrioventricular sulcus originates at a right or greater angle and becomes the left circumflex artery. The branch that enters the anterior interventricular sulcus, virtually as a continuation of the main left coronary artery, becomes the left anterior descending artery (fig. 2). Branches originating between these two (most often one to three in number) are known as diagonal left ventricular arteries, coursing in the free wall of the left ventricle between the circumflex and anterior descending arteries, and are commonly as large in caliber and as long as either of the other two more familiar branches. The diagonal branches are sometimes difficult to differentiate from the left circumflex and anterior descending arteries on angiograms. The left anterior descending artery always reaches the apex cordis and curves about it to enter the posterior interventricular sulcus, in which it ascends a distance of 2 to 5 cm. The apex of the left ventricle is thus supplied almost entirely by the anterior descending artery. The left circumflex artery usually terminates at or just beyond the obtuse margin of the left ventricle, supplying most of the lateral wall and a variable portion (usually about half) of the diaphragmatic surface of the left ventricle.
The right coronary ostium is normally 2 to 3 mm. in diameter and lies near the middle of the right coronary sinus of Valsalva. The noncoronary sinus of Valsalva is between the right and left sinuses and protrudes posteriorLY toward the interatrial septum, with its inferior margin lying just above the membranous portion of the interventricular septum. In addition to the main right coronary ostium in about half of human hearts there is a second ostium located about 1 mm. away and giving rise to the conus artery. This second ostium is usually less than 1 mm. in diameter, but its potential importance as a source of collateral circulation is obvious. From its aortic origin the main right coronary artery passes between the body of the right atrium and the main pulmonary artery into the right atrio-
ventricular sulcus, in which it continues about the margo acutus to the diaphragmatic surface (fig. 3). In about 90 per cent of human hearts it crosses the posterior interventricular sulcus to supply nearly half of the diaphragmatic surface of the left ventricle, in addition to most of the free wall of the right ventricle. One constant major branch descends along the margo acutus toward the apex of the heart. The right coronary artery terminates as several parallel trunks descending in or near the posterior interventricular sulcus toward the terminal branches of the left anterior de-

Figure 3

The normal blood supply of the anterior wall of the right ventricle is shown in this cast. Again the right atrium (RA) and right ventricle (RV) are cast white, as well as the anterior cardiac veins. The anterior interventricular sulcus and portions of the left anterior descending artery are seen along the right margin of the photograph. The right coronary artery courses deep in the fat of the right atrioventricular sulcus, sending looping branches out to the surface of the right ventricle. The longest and stoutest of the ventricular branches passes along the acute margin of the right ventricle. The main trunk of the right coronary artery is crossed by two anterior cardiac veins.

Figure 4

A posterior (diaphragmatic surface) view of a normal human heart demonstrating the usual blood supply of this region. Right atrium (RA), right ventricle (RV) and cardiac veins are cast white; the left chambers are uncast. The right coronary artery passes about the margo acutus and bifurcates, sending a diagonal branch across the posterior wall of the right ventricle to the midpoint of the posterior interventricular sulcus and an atrioventricular branch, which follows the conventional course to the crux of the heart. This latter branch makes a penetrating U-turn and then emerges over the diaphragmatic surface of the left ventricle, a good portion of which it also supplies. The remaining left ventricle here is supplied by terminal branches of the left circumflex artery. The apex of both ventricles is supplied by terminal branches of the left anterior descending artery. The arrow indicates the approximate location of the base of the posterior papillary muscle of the left ventricle (see also figure 1).

scending artery at the apex (fig. 4). In approximately 10 per cent of human hearts the right coronary artery bifurcates near its origin and sends a large branch coursing diagonally across the anterior free wall of the right ventricle, and the branch in the atrioventricular sulcus may also bifurcate again near the margo
acutus sending a diagonal branch across the posterior free wall of the right ventricle. In 1 or 2 per cent of human hearts the right coronary divides into one conventional trunk and a second unusual one, which curves behind the aorta to emerge in the left atroventricular sulcus and become a "left" circumflex artery; in such cases the main left coronary artery provides only the anterior descending branch.

Figure 5

The blood supply of the normal human interventricular septum is seen in this vinylite cast viewed through the uncast left ventricle, with all the arterial and venous branches of the left ventricle cut away to expose the septal region. The left anterior descending artery (white arrow) courses down the left margin of the photograph to curve about the apex of the heart. Note the length of the septal branches and the fact that virtually the entire supply of the apex is from the anterior descending artery. A few darker branches of the posterior descending artery penetrate into the septum from the right margin of the photograph, where the posterior interventricular vein (white) is the most conspicuous vessel.

Figure 6

A dissection of a normal human heart demonstrating the sinus node artery (black arrow) originating from the right coronary artery. The sinus node artery is easily dissectible from the epicardium until it enters the dense collagen frame of the sinus node; the open arrow indicates the midpoint of the sinus node, which lies beneath the sulcus terminalis at the junction of superior vena cava and right atrium (RA). LA is left atrium and Ao is the root of the aorta.

Arteries to the Papillary Muscles

There is one constant papillary muscle in the right ventricle near the junction of the free wall with the anterior margin of the interventricular septum, and this is supplied predominantly by penetrating branches from the left anterior descending coronary artery. There are two constant papillary muscles in the left ventricle, and their blood supply has multiple origins. The anterior papillary muscle is supplied primarily by one or more branches of the left anterior descending artery or by diagonal left ventricular arteries, but additional branches enter that area from the marginal termination of the left circumflex artery (fig. 1). The posterior papillary muscle is supplied by those arteries that terminate on the diaphragmatic surface of the left ventricle, and most commonly by a junction of terminal branches of the left circumflex artery and of the right coronary artery (fig. 4); when the left circumflex artery supplies nearly all the diaphragmatic surface of the left ventricle (10 per cent of human hearts), its branches provide the entire supply for the posterior papillary muscle. When the right coronary artery extends as far as the margo
obtusus (about 20 per cent of human hearts), then it supplies all the arteries to the posterior papillary muscle.

**Arteries to the Interventricular Septum**

Most of the blood supply to the human interventricular septum is provided by the left anterior descending coronary artery (fig. 5). Branches into the septum from posterior descending arteries rarely penetrate more than 15 mm. from the epicardium (slightly more than the normal thickness of the free wall of the left ventricle), so that for practical purposes one may consider the entire blood supply of the interventricular septum to be derived from four to six nearly equal size branches of the left anterior descending artery. Branches from the posterior wall serve as important sources of collateral circulation, however. The septal branches of the anterior descending artery sweep posteriorly along the right ventricular side of the septum, sending in smaller divisions to supply local areas. In this respect the septal arteries resemble the other large left ventricular arteries, which also remain away from the high pressure surface (the left ventricular endocardium) for most of their course. Since the septal branches of the anterior descending artery originate below the level of the membranous interventricular septum, the area immediately posterior to the membranous septum is supplied by the only long septal branches of the

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**Figure 7**

A. A drawing of the sinus node artery as it originates from the left circumflex coronary artery (about 45 per cent of normal human hearts). Other arteries are as labeled. The particular importance of Kugel's artery as a source of collateral circulation is discussed in the text. A, aorta; P.A., pulmonary artery; S.V.C., superior vena cava. B. A vinalite cast of a left sinus node artery (black arrow) in a human heart, with its termination being just beyond the sinus node (open arrow). RA identifies the cast of the right atrium; the left atrium is uncast. Orientation is similar to the drawing in A.
ANATOMY OF CORONARY ARTERIES

posterior descending artery. This area includes the region of the atrioventricular (AV) node and bundle of His.

**The Sinus Node Artery**

Although there are commonly several small atrial arteries near the sinus node, its primary supply is virtually always by a single stout artery about 1 mm. in diameter. In about 55 per cent of human hearts the sinus node artery originates from the second or third centimeter of the right coronary artery (fig. 6), and in about 45 per cent from the first few millimeters of the left circumflex artery (fig. 7). It almost never originates from the aorta in man, nor from the main left coronary artery or its anterior descending branch. From either right or left site of origin it courses along the body of the adjacent atrium to the base of the superior vena cava, from the left side coursing through the anterior interatrial myocardial band (Bachmann's bundle) to reach that point. At the base of the superior vena cava it circles either clockwise or counterclockwise to penetrate the sinus node near the auriculocaval junction. It is a point of some interest and importance that the sinus node artery does not terminate in the node, but passes directly through it and sends only small nutrient branches laterally into the sub-

**Figure 8**

Photomicrograph of a normal human sinus node. The left arrow marks the cellular end of the node; the right arrow lies in the middle of the broader atrial end of the node. The epicardium is above. The mass of myocardium below is a cross section of the crista terminalis (CT). The large central artery of the sinus node is well shown, with a small nutrient branch to the node visible adjacent to it. Goldner trichrome stain; × 7.

**Figure 9**

details of the internal structure of the normal human sinus node are shown in this photomicrograph (Goldner trichrome stain; × 325). Note the intimate relationship of the nodal substance to the central artery, seen at the left. The darker fibers are collagen, which forms the normal framework of the node, while the lighter ones cut both longitudinally and in cross section are sinus node fibers.

stance of the node (figs. 8 and 9). In addition to supplying the sinus node, this is the largest and most constant atrial artery, sending branches to the myocardium of both atria.

**The AV Node Artery**

The AV node is supplied by the artery which crosses the crux of the heart, that point at which the atrioventricular sulci cross the junction of the posterior margins of the interatrial and interventricular sulci. In about 90 per cent of human hearts this is the right coronary artery, and in the remainder it is the left circumflex (fig. 10). The artery crossing the crux makes a unique deep U-turn at this point, and the artery to the AV node arises near the apex of this penetrating turn (fig. 11). This U-turn is easily visualized on coronary angiograms and makes a useful anatomic landmark. The U-turn marks the location of the crux of the heart and is therefore only a few millimeters from the AV node and the ostium of the coronary sinus; it lies in the junction of the interatrial and interventricular septa; finally, a line drawn from the apex of the turn to the noncoronary sinus of the aorta divides the interatrial septum (above) from the interventricular septum.
These three drawings illustrate the principal variations in the blood supply of the diaphragmatic surface of the heart. The artery supplying the AV node originates from the apex of the U-turn at the crux of the heart and penetrates the region of the AV node (white arrow in A) from its posterior margin. It is commonest for terminal branches of both the left circumflex and right coronary arteries to supply almost equal portions of the diaphragmatic surface of the left ventricle as shown in A, and in only about 20 per cent of hearts does the right coronary extend to the margo obtusus as shown in B. It is rarer still (10 per cent of human hearts) for the left circumflex artery to cross the crux of the heart and supply nearly all the diaphragmatic surface of the left ventricle as shown in C.

(below), and effectively separates the right and left atrioventricular valves.

Anastomoses

In the normal human heart there are anastomoses between nearly all branches of the coronary arteries, particularly in the ventricular epicardium, across the atria and in the interventricular septum. These are normally straight or gently curving and commonly are 100 to 300 micra in diameter. Three strategically located small arteries that play an important role in collateral coronary circulation are the conus artery, Kugel’s artery, and the sinus node artery. The conus artery curves from its aortic or right coronary origin across the pulmonary conus, where it is met by a similar branch from the left anterior descending artery. Its potential importance as a connection between the proximal portions of the right and left coronary arteries is obvious. Kugel described an artery originating from the proximal right or left coronary artery, coursing posteriorly in the base of the interatrial septum to the crux of the heart (fig. 7A). This vessel linking the anterior and posterior ventricular coronary tree likewise has obvious value in collateral circulation to the ventricles, but it also frequently connects with branches of the AV node artery and can provide important accessory circulation to that structure. The sinus node artery, coursing in atrial epicardium and regularly anastomosing with atrial branches from both the right and left, offers a bridge of collateral

Figure 10

Vinylite cast of a normal human heart with left atrium (LA) and left ventricle cast and the right chambers not. The right coronary artery curves about the right upper portion of the photograph to reach the crux of the heart, where it makes a characteristic U-turn. The AV node artery (black arrow) originates from the apex of this turn.

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circulation between the two sides of the heart across the atria. Connection between terminal branches of arteries in the ventricular septum and between neighboring branches over the entire ventricular epicardial surface are numerous and require no special description. When anastomoses function for major flow in collateral circulation, they commonly enlarge to 1 mm. or more in diameter and become tortuous and elongated, an appearance which is in contrast to their shape in normal hearts.

**General Scheme of the Coronary Circulation**

At this point it may be appropriate to review the route which blood flowing through the coronary tree may follow. Blood entering a coronary artery may go through an arteriole and capillary to perfuse myocardial fibers, but it may also pass through an interarterial anastomosis into a second artery (fig. 12). On reaching an arteriole the blood may not enter the capillary bed but instead shunt directly into a venule, bypassing the myocardial fibers. Blood reaching a venule and then veins may finally drain into the right atrium through either the coronary sinus or the anterior cardiac veins, and the anastomoses between these two independent systems are so large and numerous that flow may at any given instant go in either direction. The significance of these points relative to interpretation of data from physiologic experiments may be indicated as follows. Studies demonstrating decreased resistance in the peripheral coronary tree, or increased rate of flow through a given segment of coronary artery, do not tell us whether the blood is actually perfusing myocardial fibers or is being shunted into venules proximal to the capillary bed. Similarly, changes in the arteriovenous oxygen difference based on sampling of blood from the coronary sinus do not tell us whether or how much precapillary shunting took place. Since anastomoses between the two major systems of coronary veins are so extensive, it is difficult to know how much shift occurs from one system to the other under a variety of conditions, including the presence of a cannula offering some resistance in the coronary sinus. The difficulty in assessing myocardial metabolism from changes in coronary sinus blood is compounded in the presence of focal disease of the coronary arteries, since it is impossible to know how much of the sampled blood came from ischemic areas of myocardium. The question of Thebesian venous drainage, arteriocameral channels, and extracardiac coronary anastomoses is beyond the scope of this brief review, but the errors that flow in these channels introduced in physiologic studies may not be negligible. Experimental methods currently employed to study the physiology and pharmacology of the coronary circulation are in some respects becoming stereotyped. New approaches are desirable, but needed even more is a fuller appreciation of the inherent anatomic limitations of most present methods.

**The Coronary Arteries in Disease**

In limiting the perspective of this discussion, histologic details of coronary sclerosis and thrombosis are omitted. There are numerous excellent reviews of that subject, with the recent one by Osborne being particularly commendable both for its factual information.

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*Figure 12*

A schematic diagram indicating the possible routes by which blood entering a coronary artery may reach the right atrium via the coronary veins.

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and its hypotheses concerning the genesis of coronary sclerosis. What will be considered here are three subjects: (1) the special clinical consequences of occlusion of certain major coronary arteries; (2) the vascular basis for arrhythmias and conduction disturbances; (3) a histologic lesion of small coronary arteries which is common to several heritable diseases.

**Topography of Coronary Occlusions Relative to Certain Clinical Problems**

**Sudden Death**

Early in the development of clinical knowledge concerning coronary occlusion and myocardial infarction there was some inclination to consider the left anterior descending coronary artery as "the artery of sudden death." Subsequent observations indicated no sound basis on which this intriguing designation could be substantiated. There is now some basis to suspect that the designation may apply to the right coronary artery. Since the right coronary artery supplies the AV node in 90 per cent of human hearts, many instances of heart block with or without escape ventricular rhythms are due to its occlusion (fig. 13). Such an occlusion may be an abrupt recent event, but if the right coronary occlusion was old the same consequences develop when the source of collateral circulation is interrupted. For this reason occlusions of the left anterior descending or circumflex arteries may lead to heart block or sudden death, but in such cases the right coronary nearly always has previously been occluded or markedly narrowed. If the right coronary occlusion was sufficiently remote, there may be no residual electrocardiographic evidence of posterior myocardial infarction, and if it was sufficiently gradual, there may be no history of prolonged chest pain. The important point is that sudden development of heart block in the patient with coronary disease virtually always indicates the right coronary artery is occluded or markedly narrowed, whatever other coronary lesion may be additionally associated. Besides supplying the AV node in 90 per cent of human hearts, the right coronary artery also supplies the sinus node in over half, so an occlusion proximal to both these nodal arteries deprives the heart of circulation to the two most efficient pacemakers. Finally, there is recent physiologic and anatomic evidence suggesting that there are vagal neuroreceptors in the region of the AV node. These may be stimulated by local acidosis during tissue asphyxia and lead to marked vagal suppression of both the sinus node and AV node, in addition to the direct effects of ischemia in these critical structures. Acute posterior myocardial infarction is for these reasons a more dangerous event (relatively) than infarction elsewhere in heart, and the intense bradycardia, heart block, and

**Figure 13**

Photomicrograph of an infarction of the AV node in a heart from a patient dying with complete heart block beginning during an acute posterior myocardial infarction. The crest of the interventricular (IVS) is also infarcted. The cavity of the right atrium is indicated by RA. The AV node artery is seen in the upper left corner of the photograph, with a band of collagen extending diagonally from the left upper to the right lower corners and separating the AV node from the septum. Goldner trichrome stain; × 18.
arrhythmias that are frequently associated with it are ominous events. The precise incidence of sudden death in acute posterior myocardial infarction and the rhythm and conduction disturbances associated with it are difficult to determine, since so many of these abrupt fatalities occur at home or on the street.

Rupture of the Heart

Because of their multiple sources of blood supply the ventricular septum, the free wall of the left ventricle, and the papillary muscles rarely rupture as the consequence of a single coronary occlusion. In the septum, for example, the combination of left anterior descending and right coronary occlusions is virtually a necessity in order to produce sufficient myocardial necrosis to lead to rupture. It should be kept in mind, however, that these two occlusions need not occur simultaneously de novo, but that one may be a remote event which may even have partially recanalized. An exception to this set of circumstances is the 10 per cent variation in which the left coronary artery supplies both the anterior and posterior descending branches and therefore all the blood supply to the interventricular septum. But for a left coronary occlusion in such a variation to interrupt flow in both branches it would have to be so proximally located that the immediate consequences of the occlusion may be fatal prior to development of sufficient septal necrosis for rupture. Since dual coronary occlusions which may lead to septal rupture necessarily deprive the AV node of most of its normal arterial supply, heart block is a frequent precursor or accompaniment of ventricular septal rupture. Similar consideration to that dealing with the septum may be made concerning rupture of the free wall or a papillary muscle. Ventricular aneurysms, which are in a sense incomplete ruptures of the heart, are also nearly always the consequence of more than one coronary occlusion.

Vascular Basis for Arrhythmias and Conduction Disturbances

In the patient with a vascular accident in the brain a physician can with considerable accuracy determine which artery was occluded by analyzing the neurologic consequences. It is possible to employ the same approach in the heart, where the same sort of vascular accidents occur. Acute ischemia of the sinus node or AV node has characteristic effects that are clinically manifest. "Apoplexy of the heart" has not received the attention it deserves.

Most coronary thromboses that lead to arrhythmias or conduction disturbances occur in the main coronary artery proximal to the origin of the sinus node artery or AV node artery. The pathogenesis of sudden complete heart block and its relationship to right coronary occlusion was discussed in the section on sudden death. The vascular basis of bundle-branch block is more difficult to define, especially on the left side. Septal branches of the anterior descending artery supply most of the septal course of the left bundle-branch system, although the left bundle branches nearest the AV bundle are supplied (like the bundle itself) by terminal distribution of the AV node artery. The right bundle branch, originating as a single slender structure, is more vulnerable to interruption by a small focus of ischemic injury, particularly as a con-
sequence of occlusions proximal to the origin of the AV node artery.

Atrial arrhythmias appearing during acute myocardial infarction are usually due to a coronary occlusion proximal to the origin of the sinus node artery and are associated with infarction of the node (fig. 14). Most such occlusions also are proximal to the origin of the AV node artery as well, or there is a second occlusion (old or new) at such a location, suggesting that efficient regular pacemaking fails only if both the sinus node and AV node are injured during myocardial ischemia. Ventricular arrhythmias as a consequence of coronary occlusion are generally considered to arise in an ischemic ventricular focus, and in this respect one might anticipate no special artery to be involved more frequently. As long as a supraventricular pacemaker is functioning to drive the ventricles, however, it is less likely that a ventricular focus will escape than if the supraventricular pacemaker fails or is blocked in the AV connections. For this reason one may logically anticipate that ventricular arrhythmias, like heart block, are more often the consequence of right coronary occlusion.

Just as in the brain, vascular accidents in critical centers of the heart may be due to coronary thrombosis, embolism (fig. 15), or hemorrhage. The incidence of coronary embolism or hemorrhage in the nodes is uncertain, since the small arteries of the sinus node and AV node are not routinely examined in most necropsies. When unexpected sudden death occurs, or when heart block or an atrial arrhythmia occurs shortly before death in a patient with a disease generally considered noncardiac, vascular lesions in the sinus node or AV node are commonly present. These may be direct involvement of the nodal artery, such as in lupus erythematosus or polyarteritis nodosa, or there may be extensive hemorrhage due to a clotting deficiency from a primary hematologic disorder such as acute fibrinolytic syndrome or leukemia. How often sudden death in “noncardiac” systemic diseases may be due to a vascular accident in

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**Figure 15**

Photomicrograph of an embolus occluding the sinus node artery of a patient with mitral stenosis who died shortly after the onset of atrial fibrillation. Goldner trichrome stain; × 75.

**Figure 16**

Hereditary medial necrosis of small coronary arteries is shown here in sections of the sinus node artery from patients dying with so-called primary pulmonary hypertension (A) and with progressive muscular dystrophy (B). In both there is granular degeneration of the tunica media with associated hemorrhage. Both prepared with the Goldner trichrome stain; × 192.
the heart can only be determined with more complete observations on the cardiac electrical mechanism in the late stage of such diseases, coupled with more careful examination of the heart at necropsy.

Hereditary Medial Necrosis of Small Coronary Arteries

From a continuing study of the nodal histology in patients dying with recent onset of arrhythmias or conduction disturbances an unusual coronary lesion has been demonstrated to be present and similar in several heritable diseases (figs. 16-18). These include progressive muscular dystrophy,9 the Marfan syndrome,9 “primary” pulmonary hypertension,10, 11 Friedreich’s ataxia,12 and the syndrome of congenital deafness associated with long QT intervals, syncope, and sudden death.13 Since all these diseases are associated with cardiac arrhythmias, fainting spells, and unexpected sudden death, the vascular lesions in the sinus node and AV node are a logical explanation of many of these events. However, in the hearts of the patients with these heritable diseases there was additionally extensive identical involvement of many ventricular arteries of the same size as the nodal arteries (1 mm. or less in diameter). From the appearance of various segments of a single artery cut serially, as well as certain similarities in arteries from one heart to another, the primary pathology appears to be medial necrosis, which may be cystic or noncystic. As a consequence of the medial necrosis, several related secondary histologic changes develop. The first of these is intimal hyperplasia or proliferation. When medial degeneration and collapse is focal, hyperplastic endothelium forms as an eccentric layer directly over the lesion, while collapse of the entire circumference of tunica media (seen in cross-section) is more often associated with radial proliferation of the endothelium into the lumen of the artery. Another secondary lesion is the replacement of some areas of medial degeneration by debris or material which is finely granular in appearance and is sometimes (but not usually) Schiff positive; this material has not been amyloid, fibrin, or fat on specific staining for these substances. The third lesion secondary to medial degeneration is a bizarre proliferation of the remaining smooth muscle leading to diffuse thickening of the artery with marked stenosis of its lumen. Since all three types of lesion have been observed in various segments of the same artery, it seems unlikely that they are

Figure 17

A disorderly medial hyperplasia seen following medial degeneration is shown in these photomicrographs of the sinus node artery from patients dying with the Marfan syndrome (A; X 75), so-called primary pulmonary hypertension (B; 146), and the syndrome of congenital deafness, long QT interval, syncope and sudden death (C; X 292). All sections are stained with Verhoeff-Van Gieson elastic stain.
A more frequently observed reparative process following medial degeneration in small coronary arteries is intimal proliferation, shown in these six photomicrographs. A is the AV node artery of the patient with progressive muscular dystrophy; × 192. B is the sinus node artery of a second patient with so-called primary pulmonary hypertension; × 192. C is the sinus node artery of a patient dying with Friedreich's ataxia; × 480. D is a left ventricular artery of the same patient with Friedreich's ataxia; × 315. E is the AV node artery of one patient dying with the Marfan syndrome; × 192. F is the AV node artery of a second patient dying with the same disease; × 192. All sections prepared with Verhoeff-Van Gieson elastic stain except D, which is a Goldner trichrome. The basic similarity in all these sections is a thin or degenerating tunica media with intimal proliferation.
separate processes but more likely local variations in reparative attempts for a single basic defect, which is necrosis in the tunica media. In view of many necropsy reports in these five diseases describing "normal" coronary arteries, it should be stressed that the larger coronary arteries in all these hearts were indeed essentially normal. It is the smaller coronary branches (0.1 to 1.0 mm. in diameter) that are the ones involved, and unless their condition is specifically stated one cannot presume that the entire coronary tree was free from disease. Because this medial necrosis of small coronary arteries has been observed in such a number of heritable diseases, it seems reasonable, at least for the time being, to consider it as also heritable. Finally, the prevalence of this medial necrosis of small coronary arteries in a variety of diseases generally recognized as frequently exhibiting "myocardiopathy" suggests that hereditary medial necrosis may be an etiologic factor in the development of otherwise unexplained cardiac enlargement, especially if there are associated arrhythmias or conduction disturbances.

Summary

This is a brief perspective review of some personal observations on the normal and abnormal anatomy of the human coronary arteries. Since it is possible to cover in detail but a few aspects of such a broad subject, the presentation was oriented to stress topics of special value to the physician dealing with patients having heart disease.

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