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Bundle branch block

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BUNDLE BRANCH BLOCK

SENIOR THESIS

by

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Introduction

The bundle of His and its branches have been the subject of considerable investigation and confusion, both in regard to their anatomy and function and the diagnosis of lesions affecting them. A consensus of what seem to be the most original and authoritative sources dealing with the subject will be presented.

Bundle branch block is not to be thought of as a disease entity but merely as a manifestation of myocardial disease. Therefore the diagnosis bundle branch block should always be qualified by an evaluation of the myocardial condition.

Anatomy and Physiology of the Ventricular Conduction System

The development of our knowledge of the auriculoventricular bundle may be said to begin in 1883 when Gaskell proved that the auricular impulse spread to the ventricles by passing over the muscular connection which exists between these two parts of the heart. His work was done on the turtle heart but in 1893 Kent showed that it was applicable to the mammalian heart.

In the same year as Kent applied Gaskell's conclusions to the mammalian heart His was studying the action of the embryonic heart. He found that the auricular impulses passed to the ventricle before nerves had reached or been developed within the heart. He demonstrated a muscular connection between auricle and ventricle at only one point; the junction of the auricular and ventricular septa, the point at which Kent had observed a connection.

The work of His and Kent was verified by a number of workers, however, Tawara (1906) made the next advance. He discovered that the bundle described by His descended on the interventricular

septum and was continued by ramifications to all parts of the ventricular walls; the ramifications of this system he found to be made up of Purkinje fibers. (Purkinje 1845) The following anatomical description is taken from Yater (1938).

The ventricular conduction system begins at the auriculoventricular node which lies close to the right side of the posterior portion of the central fibrous body, that is, in the right auricle a short distance anterior to the orifice of the coronary sinus and just above the attachment of the medial or septal cusp of the tricuspid valve. A relatively large artery with branches usually runs through it. The node is a bundle of fibers which are thinner and smaller than myocardial fibers. They are interlaced in a groundwork of connective tissue and arranged in whorls. At the beginning of the node, that is, in its most posterior portion, its fibers are looser at the periphery and merge with the auricular myocardium. In older persons there is often considerable fatty connective tissue adjacent to the node on its superficial surfaces.

After a short distance, roughly 0.5cm., the node invades the central fibrous body and becomes the

auriculoventricular bundle without any definite line of demarkation. The bundle runs obliquely downward in the lower part of the central fibrous body to the lower edge of the membranous portion of the interventricular septum, being often separated by some fibrous tissue in its anterior part from the myocardium of the septum. It has no true sheath but is contained largely within the central fibrous body, which protects it. The fibers of the bundle are larger than those of the auriculoventricular node and run in parallel bundles. They resemble more the ventricular fibers but do not contain so much myoplasm. The connective tissue framework is delicate, and there is no main artery but a number of arterioles and venules. Sometimes in older persons there is a fair amount of adipose connective tissue in the bundle.

The auriculoventricular bundle runs for about 1 to 1.2 cm. and divides into right and left branches. The right branch appears to be more a continuation of the bundle and passes downward beneath the juncture of the medial and anterior leaflets of the tricuspid valve as a whitish threadlike process, at first being beneath the endocardium and not always distinctly

separated from the surrounding cardiac muscle. A short distance down, about 0.5 cm., the branch comes to lie more deeply in the myocardium, where it runs in a cleft of connective tissue. After a variable distance, roughly 1 cm., it gradually works its way out along a cleft in the myocardium to the subendocardium again. Here it continues for about 1 cm. spreading out to become a thin sheath of Purkinje fibers near the base of the anterior papillary muscle of the right ventricle, beyond which point it can rarely be recognized microscopically in the human heart. In its course it passes downward and naterioriy in the middle of the trabeculum of the ventricle, which corresponds to the moderator band of the beef heart. The fibers of the right branch are about the same size or a little larger than those of the myocardium but they are usually paler. They are parallel and close together, and they often closely resemble the myocardial fibers. On cross-section the branch is variable in shape, being fusiform, oval, round or triangular in different portions.

The left bundle branch spreads out from the auriculoventricular bundle on the left side of the interventricular septum, at first as a thin layer of fibers. It is subendocardial throughout most of its course and becomes much thicker as it descends in the septum.

It also gradually spreads out like a fan beneath the endocardium, and in the lower half of the septum it often divides into anterior and posterior divisions, one passing to the region of the base of the anterior papillary muscle and the other to the base of the posterior papillary muscle. In this region the Purkinje fibers rapidly spread out subendocardially among the usually numerous trabeculae and are no longer definitely recognizable. The endocardium is usually thicker in the upper part of the septum and contains descrete groups of smooth muscle fibers, which should not be mistaken for Purkinje fibers. Normally there is only a small amount of loose areolar connective tissue separating the bundle branch from the endocardium superficially and the myocardium deeply. The appearance of the fibers of the left branch in horizontal sections depends on the size of the heart and the degree of dilation, as well as on the portion of the septum being examined. These fibers are larger and much paler than myocardial fibers, containing a much less dense myoplasm. The myofibrils are more peripheral and more prominent; the cross-striations are usually readily observed but not as distinctly as in the myocardium. The Purkinje fibers become larger as the branch descends. Offshoots from the

branch into the myocardium are practically never recognized in the human heart.

Contrary to the previous statement Mahaim in 1932 described a branch off the posterior portion of the left bundle branch. It arises high up near the bifurcation of the bundle of His and passes directly into the septal myocardium. In a more recent report (1943) he states that the two branches of the bundle of His are not isolated in their course along the interventricular septum. Connecting branches as high as the top level of the musculature occur in hearts of men and animals. In most the common trunk is connected to the ventricular myocardium.

The blood supply of the auriculoventricular node and bundle and of the posterior division of the left bundle branch comes mainly from the perforating septal arteries of the posterior descending, or interventricular, artery (in 90 per cent of the cases a branch of the right coronary artery), whereas the blood supply of the right bundle branch and of the anterior division of the left branch is almost entirely derived from the perforating septal branches of the anterior descending, or interventricular, artery.

Tawara's work was verified and the embryological development of the auriculoventricular node and bundle was given by Retzer (1906 and 1908) and Keith and Flack (1906 and 1907). Much work has been done on the anatomy of the system the most important of which are DeWitt in 1909, Mahaim in 1931, Todd in 1932, Blair and Davies in 1935, Abramson and Margolin in 1936, Yater in 1938, Nonidez in 1934, and Walls in 1945.

In recent years Glomset and Glomset 1940, Glomset Glomset and Birge 1944, and Glomset and Birge 1945 have presented evidence contrary to the above works. To quote, "The evidence presented to prove the existance of a special conducting system is irrelevant and immaterial...The ridge fasciculus (the His bundle) is a slender muscle bundle in the upper part of the interventricular septum... It does not bifurcate, and therefore, has no left branch...It is structurally identical with the other muscle fasciculi of the heart. We have not found any muscle connection between the fibers of the ridge fasciculus and those of the right atrium. We have found no anatomic evidence to support the myogenic theory of cardiac conduction."

The mode of transmission of the cardiac impulse has presented many problems. Two theories are foremost, the myogenic and the neurogenic. The first and second Stannius ligatures (Best and Taylor 1945) showed that the auricles and ventricles possesed inherent power of rhythmical contraction when isolated and that their rates of contraction were different. Gaskell (1881) showed that by means of a specially devised clamp(Gaskell's clamp), compression of the auricle anywhere between the sinus and the ventricle, in an area devoid of nervous elements, caused standstill of the heart below. Also, when muscle tissue below was pricked with a needle rhythmical beats followed. In the heart of the turtle it is possible to separate the sinus and the ventricle, except for a narrow bridge of muscular tissue and a single nerve (the coronary nerve). The only nervous communications between the two chambers is by means of this nerve. If in a heart so prepared the coronary nerve be cut no change in the rhythms of sinus and ventricle occurs. On the other hand, if the nerve trunk be left intact but the muscular connection severed, the two chambers then beat quite independently of one another.

In addition to the evidence just given the

following facts in favor of the myogenic theory of the origin of the beat may be cited. The heart of the human fetus begins to beat after three weeks of gestation; nervous elements do not appear until two weeks later. The muscle tissue of the apex is said to be free from ganglion cells yet a strip excised from this region beats rhythmically. If zig-zag cuts be made in the ventricle so as to interrupt any nervous path of transmission that might exist, the sequence of the beat is unaffected. The muscular elements of the conduction system have been shown to be capable of rhythmical contraction. Also if the continuity of the tissue be interrupted the gap is subsequently bridged with scar tissue. Regeneration does not occur and conduction is never restored; this is contrary to what one would expect if the pathway of conduction were composed of nervous tissue. (Erlanger and Blackman 1907 and 1910)

Lewis, (1915) by direct leads from the surface of a dogs heart and Barker, Maclead and Alexander (1930) by direct leads from the human heart showed that the impulse first reached the ventricle over the auriculo-ventricular node and was transmitted through the conduction system at a rate of about

5000 mm. per second. The rate of transmission through ordinary ventricular myocardium is about 500 to 1000 mm. per second.

The neurogenic theory has been supported by various owrkers. Wilson in 1909 noted an important nerve pathway incorporated in the muscle bundle and suggested a nervous mechanism. Wollard (1925) showed that the nerve trunks break up into pericardial and endocardial nerve plexuses, both in the atria and in the ventricles. Glomset and Birge (1945) believe that the cardiac musculature is under a nervous control similar to that of other muscle tissue, and consider it physiologically unsound to draw conclusions concerning cardiac conduction without taking cognizance of the rich, intrinsic nerve septem of the heart. Ninidez (1943) recognized the nerve elements in the main bundle and the proximal parts of the branches but said they were parasympathetic and of vagus origin. He suggested that they only influence the conducting system in this region as they do at the sinoauricular node.

To sum up the more widely accepted theory of the origin and spread of the excitation wave we may say: The beat is initiated in the tissue of the sinoauricular node. From here it is "broadcast" through the auricular

musculature with practically equal velocity in all directions. The auriculoventricular node acting, so to speak, as a relay station, receives the impulse and transmitts it to the ventricle via the auriculoventricular bundle. At the upper part of the interventricular system the pathway forks and the wave of excitation reaches each ventricle simultaneously. Its further course is through the branches of the bundle and its terminal arborizations.

Etiology

Bundle branch block is usually due to disease of the coronary arteries, either rheumatic or degenerative, or to hypertension resulting in strain of the left ventricle and impairment of the nutrition of the endocardium and bundle branch. (Yater 1938) With prolonged coronary insufficiency degeneration of cardiac muscle occurs, with replacement fibrosis. This is most marked in the subendocardial region of the left ventricle, where the metabolic strain is greatest. The fibrosis may involve the conduction system, which ramifies in the subendocardium, either directly or by impairing the blood supply. The right bundle branch may be involved by a similar process in long standing right ventricular enlargement. (Gubner and Ungerleider 1943).

From the facts seen in the description of the vascular supply to the conductive system it may be deduced that since obliterating vascular disease affects most often and most severely the anterior descending artery, especially high in its course, the right bundle branch and the anterior division of the left branch are simultaneously affected, and more often than other parts of the conduction system, by the resulting reduction of blood supply. If the

posterior descending artery is mainly affected by obliterating vascular disease, which is rare, only the posterior division of the left bundle branch will be seriously undernourished.

Wearn (1928) showed that the number of capillaries per square millimeter in the conduction is approximately only half of their number in the ventricular myocardium. This gives the system much less reserve blood supply and it will suffer earlier.

In cases studied histologically there seems to be a preponderant involvement of the conduction tissue over the myocardium. This suggests that the conduction tissue, like nerve tissue, is more susceptible to reduction of blood supply and anoxemia. The fact that there is a common vascular supply for the conduction system and the myocardium speaks for this conclusion. Perry (1934) reported a case in which he observed the onset of bundle branch block which was followed by frank coronary thrombosis 45 hours later. This may indicate that the conduction system is relatively more susceptable to decreased blood supply than the myocardium.

In a large series of cases Willius, Dry, and Reeser found about 70 per cent to be due to coronary sclerosis and/or hypertension. (Willius, Dry and Reeser 1941)

In a series of cases of acute coronary artery occlusion with bundle branch block Master, Dack and Jaffe (1938) found that 51 per cent were left bundle branch block, 28 per cent were right bundle branch block and 21 per cent were interventricular. At antopsy 80 per cent of the infarcts were septal.

In a series of 700 cases of angina pectoris without myocardial infarction 7.7 per cent showed introventricular block, while among 328 cases of myocardial infarction there were 9.5 per cent with intraventricular block. On the other hand, among 181 cases of bundle branch block, 39.1 per cent showed angina pectoris without myocardial infarction. (Salcedo-Salgar, and White 1935)

A less common cause of bundle branch block is rheumatic heart disease. In 1906 Aschoff had noted rheumatic infiltrations along the bundles in the connective tissue sheaths. Butterfield (1912) found the left bundle branch, in a case of bundle branch block, surrounded by rheumatic infiltrations throughout its length. Of the six cases Yater studied by serial sections (Yater 1938), three were right bundle branch block. These three were all thought to be due to rheumatic heart disease and in two rheumatic arteritis was found in or near the bundle. He suggests that since the right branch is surrounded by myocardium

in part of its course that rheumatic myocarditis and arteritis would be more apt to affect it.

White (1944) notes that rheumatic myocarditis generally involves the right bundle branch. Of a series of 52 cases of intraventricular block 5.8 per cent were apparently rheumatic in origin. Willius (1941) found about 10 per cent of his series due to rheumatic heart disease. Bayley (1934) claims that when patients with rheumatic heart disease and mitral stenosis develop bundle branch block, the conduction defect is almost invariably on the right side.

Less common causes of organic intraventricular block are syphititic infection in the heart (gummatous or diffuse) acute diptheria, rarely bacterial endocarditis and very rarely tumors and traums. (White 1944)

Functional or transient bundle branch block is relatively infrequent. During development of sclerosis there may be periods of anoxemia to the bundles which lift and come and go, giving the picture of transient recurrent bundle branch block. (Willius and Anderson 1935) It has also been noted as a toxic result of too much digitalis or quinidine or of other poisoning. It

may occur as the result of fatigue in very rapid heart action, as temporarily in auricular flutter without auriculoventricular block. (White 1944) Herrmann and Ashman (1931) note that the bundle branches may be the seat of funetional disturbances the same as organic.

Males show bundle branch block much more commonly than do females, probably because of a higher incidence of serious coronary disease. The ratio of males to females varies from 4-1 (Willius 1941) to 3-2 (Freund and Sokolov 1939).

80 per cent of cases are diagnosed in patients over 50 years old (White 1944). The average age of diagnosis is about 59 years (Willius 1941) with the highest percentage found in the seventh decade.

Glomset, Glomset and Birge (1944) believe that the same factors which lead to cardiac failure might bring about bundle branch block complexes whenever they produce failure in one ventricle while the other is relatively normal. Therefore bundle branch block complexes are caused by (a) abnormal unilateral strain (b) unilateral ventricular coronary insufficiency and (c) a combination of a and b.

Pathology

Serious damage of the conduction system may result from coronary arterial disease long before complete occlusion, slow or sudden, produces a myocardial scar or infarct. The ultimate effect of progressive arterial disease on the conduction system is fibrous replacement, just as it is the ultimate result on the myocardium. Oppenheimer and Rothschild (1917) noted that in cases of bundle branch block there are widely disseminated areas of patchy sclerosis of the subendocardial region. The sclerosis predominates in the endocardial and subendocardial layer, that is, in the region of the bundle branches, as compared with the other two-thirds of the ventricular musculature.

The coronary impairment may be caused two ways; athero-sclerosis or rheumatic arteritis. In case of acute coronary occlusion in which the bundle is incorporated in the infarct it undergoes degeneration and scarring just as the myocardium does.

Aschoff (1906) and Butterfield(1912) reported rheumatic infiltrations along the course of the bundle branches which might have impaired conduction mechanically.

Syphilitic gummas are rarely found damaging the bundles. Even more rarely congenital conditions such

as patent interventricular septum may cause conduction defects.

A source of confusion is that both bundle branches are usually involved in the pathologic process. Usually however one or the other can be seen to be more seriously affected. (Yater 1938)

Symptoms and Signs

There are no symptoms of bundle branch block other than those of cardiac disease. There are frequently associated, however, the symptoms of angina pectoris and congestive failure, and palpitation due to various complicating arrhythmias.

King and Mc Eachern (1932) found that in 50 cases of bundle branch block general physical examination revealed visable apex reduplication in 84 per cent, and palpable apex reduplication in 80 per cent. Auscultation revealed reduplication of the first sound at the apex in 56 per cent, asynchronous systolic murmur in 12 per cent and a single first sound with separated systolic murmur in 16 per cent. They stress that a combination of these findings should be present in order to make a diagnosis of bundle branch block.

Adams (1942) says that bundle branch block can not be recognized clinically although the presence of diastolic gallop rhythm or reduplication of the first heart sound is suggestive.

Diagnosis

The original concept of the electrocardiographic manifestations of bundle branch interruption, as developed by Eppinger and Rothberger (1910) and confirmed by Rothberger and Winterberg (1913) Lewis (1916) Smith (1920 and 1921) Wilson and Herrmann (1921) and others, has been attacked to the extent that the term right bundle branch block has been changed to left bundle branch block and vice versa. The main mass of evidence in favor of the original concept was accumulated as a result of experiments on animals, mainly dogs, in which interruption of part or all of one or the other bundle branch, usually by surgical section, was produced in a normal heart. The diagnosis of bundle branch block was then applied to conditions studied in the clinic in which the electrocardiograms resembled those made experimentally.

In 1914 Carter established certain criteria for diagnosis of bundle branch block in man. These were as follows: (1) widening of the QRS complex beyond 0.1 sec., with notching; (2) preponderance of the ventricle with a healthy bundle branch; (3) exaggeration of the amplitude of the ventricular deflections (QRSand T); (4) the T wave directed oppositely to

the main ventricular deflection. If the initial ventricular deflection was upright in lead I and downwardly directed in lead II, right bundle branch block was assumed to exist; if it was down in lead I and upright in lead III, left bundle branch block was predicted.

As early as 1920, however, Fahr, on purely theoretic grounds, expressed doubt as to the validity of the interpretation of bundle branch block, as well as the curves indicating ventricular preponderance, and suggested that the terms right and left should probably be interchanged. The same year Oppenheimer and Pardee stated that they found the interrupting lesion in the branch opposite the one anticipated when they examined two hearts histologically. Mann (1920) also concluded that the original terminology for bundle branch block was incorrect.

In (1930) more serious doubt began to develop regarding the accuracy of interpretation as to which is the levocardiagram and which is the dextrocardiogram in the case of humans. Barker, Macleod and Alexander (1930) published curves exactly opposite to those previously obtained for animals. Wilson, Macleod and Barker in 1932 suggested that the common type of bundle branch block (formerly called right bundle branch block) is really due to obstruction

of the excitation wave along the left bundle branch, whereas the unusual variety is due to a block of the right bundle branch.

Later Marvin and Oughterson (1932) and Vander Veer (1933) made similar observations on exposed human hearts and in general confirmed the results.

In human beings for whom a diagnosis of bundle branch block had been made, Nichol (1932) found that in instances in which the chief initial deflection was up in lead I and down in lead III the subclavian pulse was definitely delayed. He concluded that the curves really signified left bundle branch block.

By means of serial precordial leads Wilson, Johnston, Hill, Macleod and Barker, (1934) for patients who showed electrocardiographic curves which were formerly said to typify left bundle branch block, obtained curves in which the lead from the right side of the precordium showed a late chief upstroke, whereas the lead from the left side of the precordium showed an early chief upstroke approximately synchronous with the peak of R in a simultaneously run lead I.

Since the symptoms and signs of bundle branch block are not definitely diagnostic the electrocardiogram must be used. It is the only method of

diagnosing bundle branch block. There is only one criterion which is necessary to make the diagnosis electrocardiographically and that is QRS complexes with intervals that are greater than 0.12 sec. (New York Heart Association 1945) This does not include the syndrome of short P-R interval, less than 0.1 sec, and prolonged QRS, (Wolff, Parkinson, White 1930) which is almost certainly not bundle branch block (White-Heart Disease 1944) In cases in which the QRS interval is slightly less than 0.12 sec. precordial leads should be taken to determine the presence or absence of bundle branch block.

When only limb leads are employed, there are only two criteria which are of value in differentiating right branch block from left branch block. The first of these concerns the presence or absence of a conspicuous S deflection in lead I. When there is such a deflection, and the QRS interval measures 0.12 sec. or more, the precordial curves are, with rare exceptions, characteristic of complete right branch block. When no such deflection is present, and the QRS interval measures 0.12 sec. or more, the precordial leads very seldom fail to yield complexes of the kind characteristic of complete left bundle branch block. The second criterion is based upon

the general outline of the QRS complexes. If these are definitely diphasic or triphasic in those leads in which the chief deflection is of at least moderate size, right branch block is probably present; if they are monophasic or essentially monophasic left branch block is probably present. These criteria are not infallible; the first is more reliable than the second. It was formerly thought that right bundle branch block was for less common than left. When the above criteria are used right branch block is nearly or quite as common as left, but the classic curves which have been considered characteristic.of right bundle branch block are very uncommon. (Wilson 1942)

In both right and left branch block, the QRS complexes of the standard limb leads are far more variable in form than those of the precordial leads. The chief reason seems to be that the more common variations in the position of the heart have a much greater effect upon the former than on the latter. In bundle branch block, the human heart is usually in such a position that the potential variations of the left arm resemble those of the left ventricular surface while the potential variations of the left leg are either small or like those of the right ventricular surface. The position of the heart may vary to such an extent

that these relations are reversed, left bundle branch block may then be mistaken for right bundle branch block and vice versa if precordial leads are not taken. (Wilson 1942) (Ackerman and Katz 1933)

Burch and Windsor (1945) stress the fact that localization of the involved bundle is possible only if the block was present when lead I was recorded or during the recording of the precordial leads. When the deflection of greatest duration of the QRS complex is up in lead I left bundle branch block is present, when the deflection of greatest duration is down in lead I right bundle branch block is present. The deflection of greatest duration is not necessarily the deflection of greatest amplitude.

In left bundle branch block two types are recognized. Discordant is the most common and depends on the QRS being principally below the isoelectric level in lead III. Concordant depends on the QRS being principally above the isoelectirc level in lead III. (New York Heart Association 1945)

In right bundle branch block many groupings have been advocated, however they all have similarities and can be included in the following grouping, (a) the largest of the initial ventricular deflections in lead I is the S wave. In lead III the largest deflection

is usually the R wave. (b) Lead I consists of a tall narrow R wave, a broad shallow S wave, and a T wave usually upright. In lead II and III the QRS deflections are variable. (c) An infrequent form which resembles discordant left bundle branch block except for the presence of an S wave of variable dimensions in lead I. (New York Heart Association 1945) In regard to the last group above Wilson et al in 1934 mentions three cases in which in lead I all the ventricular deflections were small and there was a conspicuous S wave. Leads II and III are similar to left bundle branch block. Precordial leads in these cases revealed curves similar to those found in right bundle branch block in the dog and the more typical cases of right bundle branch block in man.

Katz, Landt and Bohning (1935) studying subclavian pulse records concluded that the QRS deflections aren't diagnostic and that too many other factors enter. The position of the heart being one of the most important of these.

In 1939 Braun-Menendez and Solari made a study using simultaneously recorded electrocardiograms and venous and arterial pulse curves from which they question the reliability of the electrocardiogram in

diagnosis of complete bundle branch block and which branch is involved. They studied patients whose electrocardiographic curves had all the characteristics of bundle branch block without there being any evidence of asynchronism and hence without there being complete block.

As previously stated precordial leads give much more reliable information as to the true character of the bundle branch block. Not every case with the QRS greater than 0.11 sec. is bundle branch block but may be due to hypertrophy of the myocardium. With a thicker myocardium. the impulse takes a longer time to penetrate thus prolonging the QRS complex. (Goldberger 1945) In 1930 Wilson and Herrmann concluded from a comparison of the QRS interval and the ventricular weight that the length of the QRS interval increases in average value with the cube root of the ventricular weight. Comparison of the QRS interval and the thickness of the left ventricular wall shows a similar increase in the average valve of this interval with an increase in muscle thickness. However, QRS intervals which exceed 0.10 sec. should not in general be ascribed to increased size of the heart or to increases thickness of the left ventricular wall alone, but to retarded intraventricular conduction.

The use of precordial leads in diagnosis of bundle branch block has been most adequately described by Wilson and his co-workers. Wilson (1944) believes that there can be no reasonable doubt that, in man as in the dog, the potential variations of the right side of the precordium (Vland V2) ordinarily resemble the potential variations of the anterior surface of the right ventricle, while the potential variations of the left side of the precordium (V5 and V6) ordinarily resemble potential variation of the anterolateral surface of the left ventricle.

The character of the QRS deflections of the precordium depends mainly on the time at which the muscle under the electrode becomes active in relation to the beginning and end of the QRS interval. If this muscle becomes active very early in this interval, the R deflection is narrow and usually small and the S wave broad and usually deep. If it is activated very late in this interval, R is tall and broad, S small and narrow, and a Q wave is often present. If it is activated at the very end of the QRS interval, S is absent but there may be a conspiquous Q. As the time of activation of the muscle beneath the exploring electrode approaches the beginning of the QRS interval, R becomes smaller, S larger and Q less frequent. As it approaches the end of the QRS interval R becomes larger, S smaller

and Q more frequent.

To be more specific in right bundle branch block leads V1 and V2 will show a small R and small S and a broad and large R'. Leads V5 and V6 will show a small Q a large R and a rather small S or a large R and large, broad, notched and slurred S which is not always deep. In left bundle branch block leads V1 and V2 will show a small R and a broad S. Leads V5 and V6 will show a single broad notched R. (Wilson 1942)

Goldberger (1945) describes the basic precordial patterns as follows: (1) an M shaped QRS in unipolar leads over or facing the affected ventricle and side of septum. (2) W shaped QRS from unipolars over the normal ventricle and side of septum. (3) QRS interval longer than 0.11 sec. (4) a negative T wave with M shaped QRS, and a positive T with W shaped QRS, there are however frequent exceptions to this last point.

The method of interpretation which Pardee (1941) gives is slightly different. In right bundle branch block CF1 and CF2 show the peak of the R wave delayed (0.08 to 0.10 sec.) After the beginning of the QRS. The wave is often notched, slurred or double. Over the left ventricle, CF5, CF6, and CF7, the peak of the R is soon after the beginning of the QRS, less than

0.04 sec. usually, then a broad slurred S wave. In left bundle branch block CF_1 and CF2 show a small or no R wave with a very large S wave. CF5 may show an M shaped QRS. Beyond the apex CF6 and CF7 show an R wave with the peak delayed after the onset of the QRS by 0.08 to 0.12 sec. There is often slurring or notching on the ascending limb.

As formerly mentioned lesions of the ventricular septum play a part in the cause of bundle branch block. An initial downward, or Q deflection in lead I is very uncommon in human left branch block. When this component occurs in an electrocardiogram otherwise characteristic of this conduction defect, a lesion of the ordinary muscle of the ventricular septum should be suspected, and a full set of precordial leads should be taken. (Sodeman Johnson and Wilson 1944)

For many years it has been recognized that bundle branch block is not always a permenent phenomenon. Probably if electrocardiograms were made more frequently, the transient or intermittent nature of the block would be evident more frequently. It is probable that in most of these cases there are partial lesions of one or both bundle branches without complete interruption of function except when toxic or metabolic factors further compromise the conduction tissues or when

release of vagal tone increases the heart rate. The onset may be abrupt, as when due mainly to a sudden insult, such as infarction, with gradual disappearance of the block, during the latter part of which period there may be frecuent transitions; or the onset may be gradual, with or without more or less frequent transitions due to progressive involvment of the branch, or the transitions may occur with great frequency, regularly or irregularly, in cases in which the pathologic processes may be relatively stationary, in which event the heart rate may be of greatest importance in the production of the transition.

Bilateral bundle branch block has been recognized for some years also. The electrocardiogram is that of complete heart block but it is associated with ventricular complexes of varying form. (Wilson and Herrmann 1921) In complete bilateral bundle branch block the impulses are ventricular in origin and arise from different foci, thus causing variation in the ventricular complexes. (Bain 1941) Supraventricular impulses may travel down each branch alternately in some cases. This would be bilaterial partial bundle branch block. Strauss and Langendorf (1943) did an autopsy and microscopic examination of the septum of such a case which revealed fibrosis of both branches.

Prognosis

Many conflicting conclusions have been drawn in regard to the prognosis of bundle branch block. However, Carter, in 1914, very aptly summarized the matter. "An organic lesion confined to one branch of the A-V bundle is hardly one which can be regarded as jeopardizing life. We find that a bundle branch lesion may be present for many years and the heart still show general efficiency. But lesions of the conducting system probably are rarely confined to it but spread beyond it into the general musculature to a lesser or greater extent. Therefore, we must regard a bundle branch lesion as significant of an invasion of the heart muscle, and of considerable gravity in most cases."

This statement has in general been upheld by most men since. Probably the most important prognostic indication is the character of the underlying disease and the condition of the myocardium. (Kaplan and Katz 1939, Bishop and Carden 1939, and many others). That bundle branch block indicates serious cardiac disease is shown by Cameau, Hamilton and White. (1938) Kaplan and Katz go even further and state that neither the configuration of the electrocardiogram or the

duration of the Q R S interval is of prognostic significance. Many series show that nearly 50 per cent of the cases are dead within one year after diagnosis. (Willius 1919, Herrick and Smith 1922, Willius, Dry and Reeser 1941, and many others)

In 1925 Oppenheimer, Rothschild and Mann called attention to the favorable course which patients with an electrographic pattern showing a wide S wave run. This has been confirmed by Von Deesten and Dolganos (1934) and Wood, Jeffers and Wolferth (1935).

Willius, Dry and Reeser (1941) have divided bundle branch block tracings into three types of curves. (1) Curves in which the QRS is greater than 0.10 sec. and the T waves directed opposite to the QRS in leads I and III. (2) Any other type of curve. (3) Curves which show a wide S wave. In group number 1, 43 percent die in the first year after diagnosis and only 10 percent are alive at the end of 10 years. From group 2, 26 per cent are alive at the end of 10 years. In group 3 or the ones with wide S waves, only 20 percent die in the first year and 30 per cent are alive at the end of 10 years. The death rate of the group with wide S wave after the first year or two approximately parallels the normal death rate for that age group, the second group parallels the normal after 5 years but the rate of the first group remains

higher than normal.

The presence of a gallop rhythm is considered a bad prognostic sign. Frank coronary occlusion usually results in serious consequences. Syphilitic heart disease usually means a poor prognosis. Bundle branch block caused by congenital heart disease or thyrotoxicosis carry a relatively good prognosis. Females seem to survive longer than males. (Freund and Sokolov 1939)

Summary

The anatomy and physiology of the conduction system is still being questioned, however the concepts of His, Tawara and Purkinje are probably correct.

Bundle branch block is usually due to disease of the coronary arteries, either degenerative or rheumatic, or to hypertension resulting in strain of the left ventricle and impairment of the nutrition of the endocardium and bundle branch.

Bundle branch block is usually associated with bilateral bundle branch lesions, although one branch is usually more seriously affected than the other and probably usually determines the essential form of the electrocardiographic curve.

Right bundle branch block is probably usually due to rheumatic arteritis or rheumatic myocarditis.

Left bundle branch block is probably usually due to coronary arteriosclerosis or arterial hypertension or both resulting in fibrosis of the bundle.

In both right and left bundle branch block, the QRS complexes of the standard limb leads are far more variable in form than those of the precordial leads.

When there is a conspiquous S deflection in lead I right branch block is probably present, when there is no such deflection left branch block is probably present.

Prognosis depends mainly on the character of the underlying disease and the condition of the myocardium.

Patients whose curves show a broad S wave in lead I probably will run a more favorable course than others.

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