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Pure right ventricular hypertrophy in the electrocardiogram : a review of the literature : with particular reference to the electrocardiogram in pure pulmonary stenosis

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PURE RIGHT VENTRICULAR HYPERTROPHY IN THE ELECTROCARDIOGRAM--

A review of the literature with particular reference to the electrocardiogram in pure pulmonary stenosis.

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INTRODUCTION

PURPOSE OF THIS PAPER: The purpose of this paper is to review the literature on the influence of isolated right ventricular hypertrophy on the standard clinical electrocardiogram. An attempt has been made to glean from the literature a discussion of all the electrocardiographic changes that have been ascribed to right ventricular hypertrophy, in general, and to the electrocardiographic changes that have been ascribed to patients with pulmonary stenosis, in particular. It is hoped that with the aid of this paper's organization of various investigator's objective criteria and subjective comments with regard to a particular change in the electrocardiogram in right ventricular hypertrophy, the reader will gain added insight into the value of the electrocardiogram in the diagnosis of isolated right ventricular hypertrophy.

PULMONARY STENOSIS AS A SOURCE FOR THE STUDY OF RIGHT VENTRICULAR HYPERTROPHY: Though right ventricular hypertrophy is associated with a myriad of heart diseases, both acquired and congenital, pulmonary stenosis was chosen, as it represents an ideal prototype in the etiology of isolated right ventri-

cular hypertrophy. This idea was suggested by Braunwald and associates (1955) who stated, "The accurate determination of ventricular hypertrophy and preponderance is the foremost task of the electrocardiogram in congenital heart disease. Certain congenital cardiac lesions produce essentially unilateral ventricular hypertrophy and the electrocardiograms of patients with such malformations affords the opportunity for studying electrocardiographic criteria of ventricular hypertrophy." (15) Kossmann (1962) noted, "In the presence of severe outlet obstruction of either ventricle with the ventricular septum intact the electrocardiogram will accurately reflect hypertrophy of the obstructed ventricle." (75) Pruitt (1962) commented, "Pure hypertrophy should ensue when a ventricular chamber maintains a normal stroke volume against an exceptionally high resistance to flow, and accomplishes this work without sacrifice of myocardial nutrition or efficiency. These requirements can be met by the ventricle supplying either the pulmonary or systemic circulation, and the corresponding clinical state is encountered in young patients having

isolated pulmonic or aortic stenosis." (75) Finally in a recent study on a correlation between the hemodynamic and electrocardiographic changes associated with pulmonary stenosis, Hugenholtz and Gamboa (1964) said, "It appeared useful to study only those cardiac lesions in which increased pressure would be the dominent persistent hemodynamic change and thus should be the chief factor related to adaptive processes. Isolated outflow obstruction caused by congenital pulmonic valvular stenosis of varying severity was selected as the optimal model in which to conduct this study." (63) It is with this rationale that this paper undertakes the study of pulmonary stenosis and right ventricular hypertrophy.

SCOPE OF THIS PAPER: This paper has not been intended to be a critical review of the literature. Rather than to analyze the statistical significance or scientific strengths or weaknesses of the references included, it is only the conclusions and general comments leading up to these conclusions that this paper will attempt to review.

GENERAL COMMENTS ON ELECTROCARDIOGRAPHIC ANALYSIS

THE SIGNIFICANCE OF THE ELECTROCARDIOGRAPHIC DETERMINATION OF CARDIAC HYPERTROPHY: In general, the literature has considered the significance of electrocardiography in the diagnosis of ventricular hypertrophy with high regard. Wood (1950) stated, "The electrocardiogram is the most reliable clinical means of deciding which ventricle is predominent, and the point is of utmost importance in differential diagnosis." (129) Kossmann (1958) commented, "It is maintained by many that hypertrophy is an anatomic modification of the heart which is more evident from electrocardiographic deviations it causes than from any other clinical or laboratory examination." (74) Similarly, according to Burchell (1962), "The electrocardiogram is the best single method available to us clinically as an indicator of predominent hypertrophy of either ventricular chamber." (75) Dotter (1957) studied the value of electrocardiography relative to radiology in the diagnosis of cardiac hypertrophy and concluded, "In the diagnosis of congenital heart disease, when competent radiographic and electrocardiographic interpretations concerning

ventricular enlargement are not in agreement the electrocardiogram is apt to be correct." (42)

Despite this high regard, the electrocardiogram is not without its weaknesses, and Jensen (1960) clearly pointed this out by stating, "The electrocardiogram simply measures the electrical potentials which are generated by the myocardial cells as they polarize and depolarize during the cardiac cycle and are transmitted through the adjacent heterogenous tissues to the electrodes at the body surface. The electrocardiogram cannot measure directly the thickness of the ventricular wall or the diameter of the cardiac valve orifaces, and attempts to quantitate these parameters from the electrical phenomena represented on the clinical electrocardiogram are at best hazardous, frequently inaccurate and often completely erroneous." (65) In a somewhat similar vein, Kossmann (1953) stated, "It is unsound practice to make final anatomic cardiac diagnoses from the form of the electrocardiogram without recourse to other clinical and laboratory data." (73)

THE CORRELATION OF ELECTROCARDIOGRAPHIC AND ANATOMIC EVIDENCE OF HYPERTROPHY: After a considerable amount of autopsy correlation studies,

Kossmann (1953) concluded, "Probably less—and our own observations indicate considerably less—than 70% of patients with right or left ventricular hypertrophy will show the electrocardiographic configuration "characteristic" for the chamber involved. Correlations in general will be better the greater the hypertrophy." (73) In a later work Kossmann (1958) noted, in reference to another worker's autopsy controlled series, that the disease in most of these patients was so far advanced that death resulted. He commented, "Even so, using the best criteria available, both in extremity and precordial leads, the correctness of the diagnosis could be established definitely by the electrocardiographic method only in approximately 70 %. Clearly in less advanced stages of hypertrophy the incidence of positive electrocardiographic findings will be lower." (74) Similarly Scott (1962) stated, "It should be emphasized that because of the high incidence of abnormally heavy hearts occuring in an unselected autopsy population this may result, in autopsy controlled studies, in what appears to be a greater accuracy in the electrocardiographic diagnosis of

ventricular hypertrophy then may actually be the case." (75) Scott (1960) and Walker and associates (1955) concluded that though the accuracy of right ventricular hypertrophy has varied extremely (from as low as 23%), in most instances when the pattern of right ventricular hypertrophy is encountered in the electrocardiogram, the diagnosis will be substantiated at autopsy. (103, 120).

Myers (1948) found that in his group of autopsy controlled cases of right ventricular hypertrophy, there did not appear to be any direct correlation between cardiac weight, ventricular ratio or thickness of right ventricular wall and the electrocardiographic pattern. (71) Similarly Fraser and Turner (1955) studied cases of both acquired and congenital heart disease and found a striking lack of correlation between the electrocardiographic and anatomic evidence of right ventricular hypertrophy. They noted particularly poor correlation between the better developed electrocardiographic signs of right ventricular hypertrophy and the more pronounced degrees of anatomic right ventricular hypertrophy. (47) According to Human (1964), "The poor correlation

between electrocardiographic and anatomic hypertrophy is a well known fact." (64)

THE RIGHT VENTRICULAR PRESSURE AND ITS RELATION TO RIGHT VENTRICULAR HYPERTROPHY AND THE ELECTROCARDIOGRAM: Jewett (1960) rationalized, "The right ventricular pressure is probably at least as good a guide as measurements taken at autopsy, since terminal dilitation is thought to be very common and this event has a marked effect on the observed thickness of the ventricular wall." (66) Orme (1952) stated that it seemed reasonable to assume that there would be a correlation between the degree of hypertrophy in the muscle wall and the ventricular pressure, however, he was quick to add, "Reasonableness alone without any experimental basis, should never be the foundation of any medical diagnostic procedure." (95)

Most of the series reporting electrocardiographic evidence of right ventricular hypertrophy have been restricted to diseases associated with increased pulmonary artery or right ventricular pressure. Johnson and co-workers (1950) found

that all their patients except one with electrocardiographic evidence of right ventricular hypertrophy had pulmonary artery pressure greater than 30 mm Hg. (67) Cosby and his colleagues (1953) have demonstrated similar findings. (35) Walker, Scott and Helm (1955) suggested that factors such as increased pressure in the right side of the heart and in the pulmonary circuit, in addition to increased muscle mass, may contribute to the electrocardiographic pattern of right ventricular hypertrophy. (119) Joos (1954) found that right ventricular systolic hypertension and the gradient between right ventricular systolic pressure and pulmonary systolic pressure were grossly related to the occurrence of right ventricular hypertrophy by the electrocardiogram.

According to Sodi-Pallares (1956) the electrocardiogram in pure pulmonary stenosis may vary from normal or slightly altered tracings to those representing maximal right ventricular hypertrophy of the systolic overloading type. He attributed these variations in the electrocardiogram to the systolic hypertension of the right ventricle. He considered

three principle types of electrocardiogram according to right ventricular pressure: slight, with right ventricular pressure less then 90 mm Hg.; moderate, with right ventricular pressure from 90 to 130 mm Hg. and marked, with right ventricular pressure greater than 130 mm Hg. (110) In a later work (1959) he stated that in pure pulmonary stenosis the electrocardiogram closely correlates with the hemodynamic alterations. (113)

Engle and colleagues (1960) made an extensive study of the electrocardiographic evaluation of pulmonary stenosis. They found that composite evaluation (correlative evaluation of numerous electrocardiographic criteria) permitted distinguishing among patients with mild (right ventricular pressure below 80 mm Hg.), moderate (pressure 80 to 140) and severe stenosis (pressure over 140 mm Hg.). They considered the level of systolic pressure in the right ventricle as an indicator of the severity of stenosis. When the electrocardiogram was normal, they observed that the right ventricular pressure was consistantly less than 50 mm Hg. (45)

Others have found the relationship of the right

ventricular pressure and the electrocardiographic changes to be considerably less specific. Bentiviglio (1960) stated, "The severity of the stenosis and height of the systolic pressure in the right ventricle cannot be gauged in the individual patient by the character of the tracing. Although some mean measurements change in value with increasing degrees of stenosis and right ventricular hypertension, in an individual they cannot be expected to reflect a certain degree of right ventricular hypertension." (8) Similarly Kjellberg (1955) concluded that, "the individual electrocardiographic variations are so large that it is not possible in a particular case to draw any definite conclusions regarding the degree of severity." (71) Hellerstein (1963) felt that despite the good correlation for right sided systolic overloading, the electrocardiogram can be used only in terms of a predicted value for a group, and not for an individual patient. (26)

Braunwald (1955) found no significant difference in the severity of the hypertrophy of the right ventricle, or the level of the right ventricular pressure in those patients with normal electrocar-

diograms showing right ventricular hypertrophy, and he found that he could establish no correlation between the amplitude of any single deflection or combination of deflections and the level of the right ventricular pressure. (15) Gordon and Goldberg (1951), Blount (1954), Donoso (1955) and Kahn (1959) came to similar conclusions. (54, 11, 41, 69)

THE PROBLEM OF AGE IN THE DETERMINATION OF RIGHT VENTRICULAR HYPERTROPHY FROM THE ELECTROCARDIOGRAM: It is well known that there are many differences in the normal electrocardiogram of a child and that of an adult, and it is generally agreed that due to the differences in the electrocardiogram inherent in different age groups that the diagnosis of right ventricular hypertrophy is more difficult in infants and children than in adults. (54, 103) Friedman (1963) elaborated on the problem: "The electrocardiogram of normal infants and children is characterized by right ventricular preponderance. Right ventricular hypertrophy in children increases the tendency to right axis deviation and produces a further increase in right ventricular potentials. However, the differentiation between physiologic and pathologic right

ventricular preponderance in young children is often difficult or impossible because of the similarities and overlap in the electrocardiographic patterns of both. The normal adult electrocardiogram is essentially a levocardiogram because the ventricular mass is composed largely of left ventricle. To offset this ventricular predominence so that the specific diagnostic alterations of the right ventricular hypertrophy become apparent in the electrocardiogram, the right ventricle must enlarge considerably. Even then the diagnostic signs may fail to appear. This accounts for the relative frequency with which the electrocardiogram is normal in the presence of right ventricular hypertrophy." (48)

With this difference in mind, an attempt has been made throughout the subsequent discussions in this paper to separate those comments and criteria used for adults and those which refer directly to infants or children.

TERMINOLOGY AND THE ELECTROCARDIOGRAPHIC DIAGNOSIS OF RIGHT VENTRICULAR HYPERTROPHY: Such terms as predominence, hypertrophy, preponderence, and enlargement have been used throughout the electro-

cardiographic literature. Pardee (1920) explained, "...it was not the actual hypertrophy of one of the other ventricle which caused the changes in the electrocardiogram, but the relation of the state of hypertrophy of each ventricle to the other. For this reason it has become necessary to speak of the records as showing right or left ventricular predominence instead of hypertrophy." (96) More recently Friedman (1963) commented, "Although it is usually not possible in the electrocardiogram to distinguish between ventricular hypertrophy and/or dilitation, the term hypertrophy has gained acceptance by common usage. Actually the term enlargement is preferable since it includes both hypertrophy and dilitation." (18)

This terminology has been retained in its original form in the references reviewed in this paper.

VECTOR VERSUS UNIPOLAR ANALYSIS OF THE ELECTROCARDIOGRAM: Opinions in the literature have differed considerably on the relative values of vector versus unipolar analysis. Two examples of relatively contratictory opinions from two highly respected workers in the field are given as representative.

Grant (1957) stated, "It is not meant by this that vector methods should supplant the more familiar "pattern" methods of interpretation, but rather that they should supplement them. From the clinical point of view, when a tracing has the classic pattern of acute myocardial infarction, it is no more necessary to convert it into vectors than it is necessary to get an accurate measurement of body temperature when the patient has an obvious raging fever. However, when the tracing is perplexing or borderline, or when there is a slight difference in a follow-up tracing which is difficult to evaluate, then the vector method is the most accurate, objective, and rational method for interpretation that is so far available." (57) On the other hand, Pipberger (1964) stated, "It soon became obvious that vectorelectrocardiography cannot be used for quantitative studies. The method is crude and allows only an estimation of spatial relationships that does not lead to quantitative statements." (99)

QRS VECTOR FACTORS IN RIGHT VENTRICULAR HYPERTROPHY AND PULMONARY STENOSIS

Many authors have made general positive correlations of right axis deviation and right ventricular hypertrophy. (7, 25, 50, 51, 56, 78, 117, 126) Allanby and Campbell (1949) actually equated right ventricular preponderance and right axis deviation. (3) Abrams and Wood (1951) noted that it is important to distinguish right ventricular hypertrophy—an anatomic change—from right axis deviation, "the electrical position of the heart". (2) Brumlik (1958) stated, "From force of habit, one is inclined to equate right and left deviation of the electrical axis of QRS with right and left ventricular hypertrophy, and vice versa. Though a homolateral hypertrophy and axis deviation may co-exist, such concordance is far from being the rule." (79)

Probably the strongest critic of the importance of axis deviation was Braunwald (1955). In his study of congenital heart disease he concluded, "Little diagnostic significance should be attached to the mean electrical axis obtained from the standard leads." He found that less than two-thirds of

his series with right ventricular hypertrophy had right axis deviation. Of his twenty patients with pulmonary stenosis, he reported that eleven had right axis deviation, five had no axis deviation and four had left axis deviation. (15) Wilson (1944) felt that left axis deviation is "...theoretically possible, but apparantly rare" in cases with right ventricular hypertrophy. (126) Donoso (1955) mentioned that left axis deviation may occur in the presence of anatomic, electrocardiographic and vectorcardiographic evidence of right ventricular hypertrophy. (127)

In conjunction with their discussions about the mean QRS axis to the right, Grant (1950, 1957) and Cabrera (1959) have also described a shift of the mean axis anteriorly. (56, 59, 25) Phillips (1958) elaborated on this point in an enlightening fashion. His work was not based on cases of pulmonary stenosis, but his postulations were adapted for right ventricular hypertrophy in general. He described a pattern of evolution of the electrocardiogram in three stages. In the first stage, the electromotive forces are directed leftward and

posteriorly (normal electrocardiogram). In the second stage, there is a rightward but still posterior direction of the electromotive force. In the third stage there is anterior as well as rightward direction of the mean spatial vector (tall R waves over the right chest as well as right axis deviation). Patients in this last category of Phillips' series had the heaviest hearts with the thickest right ventricular walls. He concluded that, "...those electrocardiographic criteria for right ventricular hypertrophy that depend primarily on anteriorly directed forces tended to detect only the more advanced forms of right ventricular hypertrophy, whereas those criteria that are based on the rightward (although still posterior) direction of the mean spatial vector tended to detect lesser degrees of right ventricular hypertrophy." (98) The reader is referred to the section of this paper on "The Right Precordial QRS" in which the significance of the deep S wave in the right precordials is discussed. This bears an important relationship, from the "unipolar" approach concerning this concept.

Taussig (1960) stated that, "Right axis deviation and evidence of right ventricular hypertrophy are characteristic of pure pulmonary stenosis." She observed that when pulmonary stenosis is extremely mild, the electrocardiogram may show only right axis deviation and the unipolar leads will be normal. (118) Abrahams (1951) and Marquis (1951) also noted the persistent correlation of pulmonary stenosis and right axis deviation. (2, 86)

Campbell (1952) commented on right axis deviation in pulmonary stenosis and other causes of right ventricular hypertrophy. He said, "It has long been clear to me, and no doubt to many others that extreme right axis deviation (large SI and RIII with insignificant RI and SIII) is common in many forms of pulmonary stenosis and other congenital heart diseases, much rarer in mitral stenosis, and quite unusual in chronic pulmonary disease. The more detailed knowledge of right ventricular preponderance obtained from chest leads has not greatly changed this impression." (29)

Some workers have considered right axis deviation in relation to right ventricular pressure in

patients with pulmonary stenosis. Depasquale (1960) found that the degree of orientation of the mean frontal QRS to the right varied directly with the right ventricular systolic pressure. (38) Gotzsche (1950) commented, "Our patients showed a certain correlation between pronounced axis deviation and high ventricular pressure, but it must be emphasized that even pronounced pulmonary stenosis need not be associated with right axis deviation." (55) Engle and co-workers (1960) found right axis deviation to be relatively insensitive in distinguishing mild, moderate and severe cases of right ventricular hypertrophy since it was almost always present in those with systolic pressures over 50 mm Hg. (45) Jewett (1960) concluded that, "There is some relationship of QRS axis and right ventricular pressure." He found that with right ventricular pressure greater than 100 mm Hg. right axis was consistently present. (66) Sodi-Pallares (1956) stated that slight pulmonary stenosis (right ventricular pressure less than 90 mm Hg.) usually has a mean frontal QRS axis approaching 90 degrees and that with moderate pulmonary stenosis (right ventricular pressure 90 to

130 mm Hg.) the QRS axis moves more to the right (about 100 degrees). He found that the axis increases to the right with increasing severity of pulmonary stenosis, but it does not usually reach the superior sextants. (110) In a later work, Sodi-Pallares (1958) compiled his cases of pulmonary stenosis without regard to age or severity and found the following distribution: 0 to 60 degrees - 3 %, 60 to 120 degrees - 59%, 120 to 180 degrees - 34%, 180 to minus 60 degrees - 0%. (111)

Scott (1960) pointed out, "The value of axis deviation has undergone cycles of waxing and waning popularity. When the standard leads constituted the mainstay of electrocardiographic leads, right axis deviation was given considerable importance in the diagnosis of right ventricular hypertrophy. With the introduction of the unipolar extremity and chest leads, the importance attached to the axis deviation diminished. However, with the considerable interest at present in vectorcardiography, the evaluation of frontal plane projection of the mean QRS axis in the scalar electrocardiogram has again gained favor." (103)

A few have established actual numerical criteria of axis deviation on which they base an electrocardiographic diagnosis or suspicion of right ventricular hypertrophy. Scott (1960, 1962) stated that right axis deviation of plus 110 degrees or greater in the adult and plus 120 degrees or more in the child under three years of age were included in his criteria for right ventricular hypertrophy. (103) Hollman (1958) considered an electrical axis over 120 degrees a criterion for right ventricular hypertrophy after one month of age. (62) Nadas (1957) in his textbook of pediatric cardiology described right axis deviation (deep S in I and tall R in III) of 120 degrees or more as "suggestive of right ventricular hypertrophy." (94) Schleris (1963) and Sokolow and Edgar (1949) said that a right axis deviation greater than 110 degrees was one of their criteria for right ventricular hypertrophy. (102, 116) Sodi-Pallares (1956) stated that an axis beyond 90 degrees in an adult is strongly suggestive of right ventricular hypertrophy. (110) Grant (1950) felt that beyond the age of 40 a vertical axis or frank right axis deviation usually means right ventricular

hypertrophy except in thin individuals. (56)

Friedman (1963) made a distinction between right axis deviation and abnormal right axis deviation. He described right axis deviation as indicating a mean electrical axis between plus 91 and minus 90 degrees. He commented, "The values for no axis deviation, right axis deviation etc. are purely descriptive and do not imply normality or abnormality. The range of normal values is variable. Age and body build are significant factors in determining the normality or abnormality of the degree of axis deviation in any individuals." He described abnormal right axis deviation, in adults, as indicating a mean electrical axis between plus 110 and minus 90 degrees. He noted that that a right axis deviation between plus 110 and minus 90 degrees may be abnormal, but that it is frequently a normal varient, particularly in young adults or asthenic individuals. He described abnormal right axis deviation, in children, as indicating a mean QRS to the right of plus 120 degrees—in those from three months to sixteen years of age. He stated that in infants under one month of age, marked

right axis deviation may occur normally, and it is difficult to define standards of abnormality. In infants between one and three months of age, he felt abnormal right axis deviation is probably present if the mean QRS lies to the right of 140 degrees. (48)

The question of the effect of position of the heart on the axis deviation has plagued those who have studied the electrocardiogram since the time of Eintoven in 1913. (44) In more recent studies, Kossmann (1948) described change in heart position in the thorax, distinct from right ventricular hypertrophy, which may cause right axis deviation. In considering the presence of right axis deviation in right ventricular hypertrophy, he concluded, "It is doubtful that the hypertrophied right ventricle, except in rare instances, can cause right axis deviation by itself. It appears to have a dominent effect on the electrocardiogram by changing the position of the heart in the thorax." (72) In a later study, Kossmann (1953) reported on a case of proven right ventricular hypertrophy with left axis deviation and a case of proven left ventricular

hypertrophy with right axis deviation. He noted, "Hypertrophy had little to do with the mean direction of ventricular depolarization except insofar as it determined the position of the heart in two patients with widely different thoracic configurations. The combinations of short, round thorax with right ventricular hypertrophy and long, flat thorax with left ventricular hypertrophy were of such a nature that in each instance position of the chambers with respect to the extremities used for leading in the frontal plane far outweighed the effect of any preponderance caused by hypertrophy of one or the other ventricles." (73)

Burchell (1949) stated, "As axis deviation in the standard leads depends on the position of the heart as well as the relative hypertrophy of one ventricle, the electrocardiographic diagnosis of ventricular hypertrophy will depend mainly on changes in the precordial leads." (23) Blacket (1951) found that increased right axis deviation suggested increased severity of right ventricular hypertrophy in his series of cases of isolated pulmonary stenosis. He pointed out, however,

that, "As this measurement depends so much on the position of the heart, it cannot be expected to supply precise information about the right ventricle." (9) Lepeschkin (1951) stated that dilitation of the right ventricle (not with particular reference to etiology) may cause clockwise rotation of the heart about its long axis and thereby produce right axis deviation. (80)

Jensen (1960) described a case of pectus excavatum displacing the heart leftward and causing deviation to the right of the mean frontal QRS vector. (65) A similar case was described by Scott (1962). (75)

Lasser and Grishman (1951) were critical of those who claimed unusual axis deviation was due to rotation or change of position of the heart. In describing cases of right ventricular hypertrophy with axis deviation between 180 and minus 90 degrees, they stated, "It does not appear likely that anatomical rotation of the heart alone would be capable of causing such marked alterations in position and form of the vectorcardiogram. Careful examination and angiocardiograms failed to disclose

any anatomical rotation or displacement of the heart of sufficient degree to account for the very marked unusual vector pathways reported here." (78) Grant (1953) showed in a careful anatomic-electrocardiographic study that there is rarely more than a 20 degree variation in the anatomic long axis of the heart in either the normal subject or the subject with marked right or left ventricular hypertrophy regardless of body build. (57)

Other factors have also been considered in the development of right axis. Friedman (1963) found that, though right ventricular enlargement is the most frequent cause for right axis deviation, other causes include: pulmonary emphysema, superior myocardial infarction, inferior myocardial infarction, acute cor pulmonale, anterolater myocardial infarction and normal variation in young adults and asthenic individuals. (48) Scott (1962) also mentioned normal hearts and anterolateral myocardial infarction with right axis deviation. (75) Wilson (1944) and Scott (1962) described rare instances where right axis deviation occurs in patients with left ventricular hypertrophy. (126, 75)

Finally, a few workers considered the importance of the QRS vector in the horizontal plane. They described the occurrence of a horizontal loop which, when viewed from above, rotates clockwise. This was described as consistently occurring in right ventricular hypertrophy and in no other conditions except left bundle branch block. (15, 41, 74)

THE RIGHT PRECORDIAL QRS IN RIGHT VENTRICULAR HYPERTROPHY AND PULMONARY STENOSIS

THE R WAVE, S WAVE AND R/S RATIO: Scott (1960) observed that the current electrocardiographic diagnosis of right ventricular hypertrophy has been based largely on criteria which depend primarily on changes in the right precordial leads. He included among these an increase in the height of the R wave, a decrease in the depth of the S wave and an increase in the R/S ratio. (He also mentioned delay in the onset of the intrinsicoid deflection and ST and T wave changes which will be discussed in later parts of this paper.) (103)

Wilson (1944) described the changes as a "reversal of normal precordial lead relationships" in the presence of right ventricular hypertrophy. In the leads from the right side of the precordium he found that the R waves constituted the chief component of the QRS and that the S wave was either absent or relatively small in comparison with the R. (126, 127) Friedman (1963) described a tall R wave in the right precordial leads as one of two characteristic patterns that are seen in right

ventricular hypertrophy (the other being the R' complex—the latter will be discussed in another section of this paper). He, as well as Grant (1957), ascribed the tall R wave patterns to rightward and anterior rotation of the cardiac vector. (48, 59)

Abrahams and Wood (1951) studying cases of pulmonary stenosis with normal aortic root divided right ventricular hypertrophy into four grades according to unipolar chest lead appearances: (1) slight - R and S waves about equal in amplitude in V1, (2) moderate - R wave dominent in V1 but S wave still present, (3) severe - R tall in V1, S small or absent (and T usually inverted) and (4) similar to 3 but extending across the chest to V3, V4 or even V5. (2)

Bentiviglio (1960) in a study of cases of pulmonary stenosis with intact septa found that the majority showed evidence of right ventricular hypertrophy which he equated with the presence of R/S greater than 1, or a pure, notched or slurred R in V4R, V3R and V1—the frequency increasing with the severity of the stenosis. He also noted the amplitude of the S wave in V1 decreased in

association with increasing severity of stenosis. (8) Camerini (1956) concluded that in an adult the pattern of R greater than S in V4R was almost invariably diagnostic of right ventricular hypertrophy and was encountered most frequently in subjects with considerable rather then slight right ventricular hypertrophy. (27) Cosby (1952) stated that tall R waves over the right ventricle, usually tallest in V1 and an R/S ratio over the right ventricle of greater than 1 were included in his criteria for the diagnosis of right ventricular hypertrophy. (34)

Kjellberg (1955) described the tall R wave in V1 as the most characteristic electrocardiographic change in pulmonary stenosis. (71) Human (1964) believed that the large R wave in the leads overlying the hypertrophied right ventricle is probably produced by a similar mechanism under all circumstances, irrespective of whether it is a part of a qR, rsR' or pure R pattern. He added that current criteria depend primarily on (abnormal axis deviation and) the change in the right precordial leads, and the R/S ratio in these leads has proved to be of great value in the recognition of right

ventricular hypertrophy. (64) Numerous others in the literature have presented similar conclusions involving an increased amplitude of the R wave, decreased amplitude of the S wave and increased value of the R/S ratio in cases of pulmonary stenosis (3, 12, 86) and in general for right ventricular hypertrophy (74, 75, 83, 91, 110).

Lantman (1954) found a decreased amplitude in the R wave as a consistent post-operative change after repair of pulmonary stenosis. (77)

The presence of a deep S wave in lead V1 or V2 usually is not associated with right ventricular hypertrophy although it has been mentioned as occurring in right ventricular hypertrophy in congenital heart disease. (41, 79) Shubin (1958) presented and reviewed 10 cases of isolated right ventricular hypertrophy in which deep S waves were present in either V1, V2 or V3. He concluded that it is quite possible that the R/S ratio in these leads may be less than 1.0 and right ventricular hypertrophy may still be present. He noted that as the vector loop is displaced anteriorly, as in early right ventricular hypertrophy, there will be decreased positive

voltage in V1 and V2, however S waves will occur if the vector loop moves more posteriorly as occurs in more advanced right ventricular hypertrophy. (105)

THE FALSE POSITIVE RIGHT PRECORDIAL R WAVE: Cabrera and Monroy (1952) found that an increase in voltage of the R wave in V1 was characteristic of the "systolic overloading" type of ventricular hypertrophy of the right ventricle, but they added that a tall R could also be an expression of "diastolic overloading" of the left ventricle. (24)

Rosen (1964) pointed out that a tall R in the right precordium could be falsely interpreted as a sign of right ventricular hypertrophy and actually be due to a technical error (recording of aVF—a fairly common occurrence), right bundle branch block, or a posterior wall infarction. (100) Scott (1962) explained that a strictly posterior myocardial infarct will, in association with its tall R waves in the right precordial leads, produce sequential ST segment depression and tall T waves which will help distinguish these cases from those with right ventricular hypertrophy. (75)

Angle (1964) has considered esophageal hiatus

hernia as a possible cause for isolated tall R waves in the right precordium. (4b)

RELATIONSHIPS WITH THE RIGHT VENTRICULAR PRESSURE: In considering the relationship of electrocardiographic changes in the right precordial leads and right ventricular pressure the most commonly referred to parameter has been the R wave. Engle (1960) found that the amplitude of the R wave in V1 gave the highest correlation, of all the criteria he used for right ventricular hypertrophy, with right ventricular systolic pressure in their series of cases with isolated pulmonary stenosis. (45) Sodi-Pallares (1958) described with slight pulmonary stenosis (right ventricular pressure less than 90 mm Hg.) normal or slightly increased amplitude of R V1, with moderate pulmonary stenosis (right ventricular pressure 90 to 130 mm Hg.) an R V1 definitely elevated "with an essentially positive QRS", and with marked pulmonary stenosis (right ventricular pressure greater than 130 mm Hg.) the changes in V1 seen in a greater number of chest leads both to the right and left of V1. (112)

Silverman and co-workers (1956) found all

twenty patients (with pulmonary stenosis and intact ventricular septa) who had a right ventricular pressure of 100 mm Hg. or more showed R waves in V4R or V1 of 20 mm or over. (107) Schleris and colleagues (1963) concluded from the data on his series of cases of pulmonary stenosis, "Although exceptions exist, the amplitude of the R wave in V1 tends to increase as the right ventricular systolic pressure increases." (102) Luna and Crow (1961) in a study of pulmonary hypertension and systolic overloading of the right ventricle concluded, "The ratio of right ventricular to systemic pressure correlated well with the configuration of the initial component of the QRS component in lead V1." (84) DePasquale and Burch (1960) found in their cases of congenital isolated pulmonary stenosis, "The ratio of the amplitude of the R wave to that of the S wave in V1 was directly related to the pressure recorded in the right ventricle." (38) Ohme (1952) stated, "There appeared to be a correlation between right ventricular pressure and the height of the R wave in V1." (95)

Caylor and co-workers made some somewhat confusing comments in their study of the relation of

systolic pressure in the right ventricle to the electrocardiogram. They stated, "The ratio of pressure (right ventricular) to voltage (of R in V1) is quite variable and pressure therefore cannot be predicted from a pressure to voltage ratio. The ratio is less variable when the pressure is greater than 100 mm Hg., but it does not correlate well enough to allow accurate calculation." They found that patients with a right ventricular pressure less than 75 mm Hg. (that is those with milder degrees of pulmonary stenosis) have little or no increase in right precordial voltage. They did find, however, that there is a "correlation coefficient for the regression equation" of the pressure to voltage relation. Using pressure equals three times the voltage in mm plus 47, they claimed that a fairly accurate prediction of the pressure could be made from the height of the R wave in V1. They stated, "Patients with an R wave in V1 of 20 mm or greater almost always had systolic pressures of at least 100 mm Hg. and conversely patients with R V1 less than 20 mm rarely had pressures above 100 mm Hg." (30)

Blount and co-workers (1954) observed that all their patients with isolated pulmonary stenosis who demonstrated a tall R wave in V1 pre-operatively, had a significant decrease in the R wave post-operatively—after repair of the pulmonary stenosis. They stated that this reflected a decrease in right ventricular pressure, but that they could find no quantitative relationship between the fall in the right ventricular pressure and the decrease in R V1 amplitude. (12)

The following workers have been impressed with the lack of correlation of these factors. Cosby et. al. (1952) calculated correlation coefficients relating mean right ventricular pressure with right ventricular work and R/S ratio in V1. They concluded that none of these factors appear to be mathimatically related. (34) Jewett (1960) concluded that there is no apparent linear relationship between the amplitude of V1 R and the right ventricular pressure. He stated that the R/S ratio in V1 gives poor correlation with the right ventricular pressure. (66) Kjellberg (1955) found the correlation between the pressure in the right ventricle and the amplitude

of the R wave in V1 "is not particularly strong." (71) Human (1965) studied eighty patients with right ventricular hypertension and found no correlation between the height of the R wave or the R/S ratio in V1 and the height of the right ventricular pressure. (64)

THE RIGHT PRECORDIAL QRS IN CHILDREN: A number of authors have noted that it is common for the R wave to be dominent (R/S ratio greater than 1) in the majority of infants and children during the first one to three years of life. (60, 80, 94, 110, 111, 131) Gordon (1951) stated that the R/S ratio at birth is normally infinity in V4R and V1; it gradually approaches one at about three years of age; and it is normally less than one thereafter. He described an R/S ratio over one in V4R or V1 in those over three years of age as indicative of "right heart strain". (54)

Scott (1960) noted that after age three the majority of children will show an R/S ratio less than one in V1, although occasionally this may not occur until age five or even older. He described a monophasic R or a qR pattern in the right

precordials as being one of the more important electrocardiographic criteria for the diagnosis of right ventricular hypertrophy in infants and children. (103) In Nadas' text on pediatric cardiology (1957) tall, unslurred R V1 and V2 with or without a q wave and with small if any s wave is described as unquestionably indicative of right ventricular hypertrophy. (94) Further comments on the right precordial QRS factors in children are included in a latter section of this paper on quantitative criteria of the right precordial QRS.

QUANTITATIVE CONSIDERATION OF THE RIGHT PRECORDIAL QRS: Though Nadas (1957) states, "The diagnosis of ventricular hypertrophy should seldom, if ever, be based on voltage alone.", Nadas, as well as a few others, have established definite numerical (based on voltage) criteria on which they diagnose right ventricular hypertrophy. (94)

Nadas stated that R in V4R or V1 greater or equal to 20 mm without an S wave could be considered indirect evidence of right ventricular hypertrophy. (94) Sokolow (1949) considered R V1 greater or

equal to 7 mm a criteria for right ventricular hypertrophy. (115) Engle (1960) observed that when the right ventricular systolic pressure was over 140 mm Hg., the R wave in V1, exceeded 20 mm in amplitude. When the R wave in V1 was more then 10mm in amplitude, the pressure was over 80 mm Hg. (45) Orme (1952) stated that the R in V1 greater than 7 mm in those over 5 years old and greater than 11 mm in those under 5 years indicated right ventricular hypertrophy. (95) Friedman (1963) used R V1 greater or equal to 7 mm in adults, greater or equal to 16 mm from 1 year to 16 years and greater or equal to 20 mm in those under 1 year as criteria for right ventricular hypertrophy. (48) Braunwald (1955) considered R V1 greater than 15.5 mm in those over 20 years, greater than 16.7 mm in those from 10 to 20 years, greater than 20 mm in those from 1 to 10 years, and greater than 29 mm in those under 1 year of age as criteria for right ventricular hypertrophy. (15)

Friedman (1963) and Hollman (1958) established criteria for right ventricular hypertrophy by maximum R/S ratio in V1. These criteria are tabulated

below for comparison:

Friedman's criteria (48)	Hollman's criteria (62)
R/S V1 greater than:	R/S V1 greater than or equal to:
6.5 from 0-3 months	7.0 from 1-3 mo.
4.0 3-6	4.5 4-11
2.4 .... 6 mo.-3 years	2.5 .... 1-2 yr.
1.6 .... 3-5 yr.	2.0 .... 3-5 yr.
1.0 .... 6-15 & adults	1.5 .... 6-15 yr.

Orme (1952) considered R/S V1 greater than 1 in those over 5 years of age and greater than 4 in those under 5 years of age to indicate right ventricular hypertrophy. (95)

THE RIGHT PRECORDIAL R ' (PRIME) COMPLEX: The occurrence of the secondary R wave in the right precordials has been correlated with cases of pulmonary stenosis by a number of investigators (9, 10, 12, 45, 102, 107), and it has been correlated with right ventricular hypertrophy, in general, by considerably more. (6, 13, 14, 15, 19, 24, 26, 40, 43, 48, 59, 64, 75, 88, 94, 103, 106, 110, 111) Most of these latter studies have been concerned with the significance of right bundle block in either its incomplete or complete form and its relation to right ventricular hypertrophy.

Booth, Chou and Scott (1958) summarized their

criteria for right bundle branch block. It is recorded here in its entirety to make the subsequent discussion more meaningful.

Booth et. al. criteria for RBBB:

1) S wave in lead I.
2) Primary and secondary R waves in leads from the right precordium with the R' exceeding the initial R wave in height, i.e. rsR', rSR'.
3) Delay in the onset of intrinsicoid deflection in the right precordial leads greater than 0.05 second.
4) An S wave in V5 or V6.
5) No initial Q waves over the right precordium.

The block was deemed incomplete if the QRS interval measured 0.08 to 0.11 seconds inclusive, and complete if the QRS interval measured 0.12 second or greater. (13)

Blacket (1951) found the "right ventricular hypertrophy pattern" and the "incomplete right bundle branch block pattern" with equal frequency in cases of pulmonary stenosis, and he observed that the occurrence of one or the other pattern did not seem to depend on the severity of the lesion. (9) Silverman (1956) concluded that there seemed to be no correlation between the degree of right ventricular hypertrophy in his series of cases of pulmonary stenosis and the presence of incomplete right bundle branch block. (107)

Although complete bundle branch block has been reported in cases of pulmonary stenosis (10), widening of the QRS interval, according to Scherlis (1963) is rare in isolated pulmonary stenosis. (102)

Blount (1954) described a transition of the QRS in V1 from a typical right ventricular hypertrophy pattern to one suggestive of incomplete right bundle branch block in cases of pulmonary stenosis after operative repair. He theorized that this change may reflect a stage in regression of the right ventricular hypertrophy pattern or may reflect the dilitation of the right ventricle with an alteration in the type of ventricular stress. (12) Cabrera (1952) also considered incomplete right bundle branch block as a reflection of a change in ventricular stress; he described incomplete and complete right bundle branch block as characteristic for "diastolic overloading of the right ventricle." The term diastolic overloading, according to Cabrera, indicates that the ventricle is contracting against an increased residual blood volume in diastole, resulting from either increased flow or valvular insufficiency. The greater the initial

length of the fibers the more powerful the cardiac contraction will be. The basic compensation is thus ventricular dilitation and increased stroke volume. In contrast they considered pulmonary stenosis as resulting in "systolic overloading" which leads to the development of concentric hypertrophy and a different electrocardiographic picture. (24)

Scott (1960) stated that the rSR' pattern in V1, when it occurs in right ventricular hypertrophy, is ordinarily associated with right ventricular dilitation. This dilitation is thought to cause stretching and in some other way interfere with the right ventricular conducting network, and as a result cause slowing of the right ventricular conduction. (103)

Human (1965) studied 80 cases of systolic right ventricular hypertension and found no typical pattern for "systolic overloading" in the right precordial lead (V1). He found that R and rsR' patterns occurred in roughly equal proportions. (64)

In the general consideration of bundle branch

block and its relationship to hypertrophy, Master (1940) stated, "We have been impressed with the frequent occurrence of cardiac enlargement usually of marked degree, when bundle branch block is present. Furthermore we have found that left bundle branch block is usually associated with enlargement of the left ventricle and right bundle branch block with that of the right ventricle. We believe that these relationships are causal, not fortuitous. Our observations make it evident that ventricular enlargement and myocardial disease are almost constantly present when the electrocardiogram shows bundle branch block." (98) Dow (1950) stated that persistent incomplete right bundle branch block is very good evidence of right ventricular hypertrophy. (43) Grant (1957) stated that if the terminal frontal QRS vector is directed rightward and inferiorly with no prolongation of the QRS interval with an R' at V1, "...right ventricular hypertrophy is nearly always present." (59) Schleris (1963) described the rsR' in V3R or V1 with an intrinsic deflection of 0.05 - 0.75 as a criterion for right ventricular

hypertrophy. (102) Camerini (1956) considered the rSr' pattern not to be diagnostic, while the rSR' was thought to be suggestive of right ventricular hypertrophy. (27)

In contrast to those who have been impressed with the correlation of bundle branch block and hypertrophy, Booth and colleagues (1958) concluded, "...in cases unselected as to postmortem etiologic diagnosis, the presence of right bundle branch block, either complete or incomplete, does not necessarily indicate right heart hypertrophy or even heart disease at all. Right bundle branch block is merely suggestive, but certainly not pathognomonic of right ventricular hypertrophy. " (14) Similarly Sodi-Pallares (1956) stated, "M complexes in the right precordials are expressions of incomplete right bundle branch block which in about 75% of cases represent right ventricular hypertrophy, but in other cases there may be no right ventricular hypertrophy." (110) With reference specifically to infants and children, Sodi-Pallares (1958) stated, "There are instances in which the ventricular complex shows a double positivity suggesting

some degree of right bundle branch block. From the clinical point of view most of these are normal." (111)

Bryant (1958) stated that right ventricular hypertrophy in the presence of a major degree of incomplete right bundle branch block can be diagnosed from the electrocardiogram with little accuracy. (19) Braunwald (1955) concluded, "The criteria of right ventricular hypertrophy derived in the presence of normal conduction must not be applied to an electrocardiogram with an RSR' pattern from the right precordium, since this pattern may represent a conduction disturbance." (15) Scott (1960) concluded that the diagnosis of right ventricular hypertrophy in the presence of right bundle branch block is frought with considerable difficulty. He added that right bundle branch block is associated with enlargement of the left ventricle either alone or in conjunction with right ventricular hypertrophy in too many cases to render it a significant sign of an isolated right ventricular lesion. (103)

The rSR' pattern has been interpreted by some

to be a manifestation of localized right ventricular hypertrophy. Kossmann (1962) interpreted the rSR' pattern to represent hypertrophy of the right ventricular outflow tract unless "...the R' is broad and accompanied by broad S waves in leads I, V5 and V6." The latter description he felt indicates terminal slowing of the inscription of the QRS which he thought, "...is more likely to be due to incomplete right bundle branch block." (75)

Myers (1956) and Silver (1959) distinguish two types of rsR' patterns according to the duration of the initial r. They attribute rsR' patterns with initial r waves of less than 0.025 seconds to hypertrophy of the crista-supraventricularis, whereas those with an initial r wave greater than 0.03 seconds represent incomplete right bundle branch block. (93, 106) Blount (1957) stated, "...The rSR' pattern with a total QRS time of 0.08 to 0.10 seconds observed in certain congenital and acquired lesions...is due to hypertrophy of the right ventricular outflow tract rather than to incomplete right bundle branch block." (13)

Many investigators have attempted to correlate

the height of the secondary R wave with the severity of right ventricular hypertrophy or right ventricular hypertension. Scott (1960) stated that with an R' greater than 10 mm right ventricular hypertrophy was present. (103) Barker (1949) agreed with this value of a 10 mm R' wave (6), but he revised this figure to 15 mm in 1952 because he felt the lower figure resulted in a significant number of false positive diagnoses of right ventricular hypertrophy. (5) Kossmann (1962) felt that in the presence of incomplete right bundle branch block tall secondary R waves (greater than 10 mm in height) may, but do not necessarily, indicate concomitant right ventricular hypertrophy. (75) Friedman (1963) considered a secondary R wave of greater than 6 mm as an indication of probable right ventricular hypertrophy. (48) Nadas (1957), commenting on the electrocardiogram in infants and children, stated that the incomplete right bundle branch block pattern in the right precordials consisting of rsR' probably indicates right ventricular hypertrophy if the R' is more than 10 mm.

He added that the narrower the QRS complex and the taller the R', the more right ventricular hypertrophy is suggested. (94)

Dodge and Grant (1956) observed that in cases of intermittent right bundle branch block that the R' wave in V1 ranged in height from 4 to 12 mm in 90% of their 80 cases and was 20 and 23 mm in two cases. In none of these was there evidence of right ventricular hypertrophy prior to the development of the block. They thus demonstrated the range of deformity that right bundle branch block may produce in the absence of electrical evidence of right ventricular hypertrophy prior to the development of the block. They concluded that the magnitude of the deflection of the last 0.04 seconds must exceed these values (20-23 mm) considerably before the diagnosis of right ventricular hypertrophy plus right bundle branch block can be made. (40) Barker and Valencia (1949) and Wilson (1947) showed similar illustrations of intermittent right bundle branch block in which the secondary R wave in V1 was 15 mm, yet during normal intraventricular conduction there was no

electrocardiographic evidence of right ventricular hypertrophy. (6, 128) Levine (1958) also made a comparable observation. (81)

Human (1964) found no correlation between the height of the R' wave and the height of the ventricular pressure in patients with right ventricular hypertension. (64) Booth and co-workers (1958) concluded that "If one attempts to relate the height of the R' wave over the right precordium to the ratio right ventricle/left ventricle thickness, there can be noted no correlation between the voltage produced over the right precordium and the relative preponderance of the left or right ventricle." (14)

Other factors have been considered to aid in the diagnosis of right ventricular hypertrophy in association with right bundle branch block. Friedman (1963) felt that a primary R greater than 8mm or an R'/S ratio greater than 1.0 with a normal QRS interval is an indication of probable right ventricular enlargement. (48) Hellerstein (1963) believed that with right bundle branch block an R/S ratio greater than 1.0 in V1 suggests right

ventricular hypertrophy. (26) Human (1964) stated that he could find no correlation between the R'/S ratio in V1 and the height of ventricular pressure in patients with right ventricular hypertension. (64)

THE RIGHT PRECORDIAL VENTRICULAR ACTIVATION TIME: The terms ventricular activation time, pre-intrinsicoid deflection, and intrinsicoid deflection have been used considerably in the recent electrocardiographic literature. Apparently these expressions have been considered synonymous, but few have actually defined their usage. Friedman (1963) defined "intrinsic deflection" as the deflection that signifies activation of muscle directly beneath the exploring electrode of a unipolar lead. (48) This is a theoretic deflection which has been considered comparable with the intrinsicoid deflection as actually seen on the clinical electrocardiogram. Friedman (1963) stated that the time of onset of the intrinsicoid deflection is measured from the beginning of the QRS complex to the peak of the R wave and that it is measured only in the precordial leads. He commented, "While the intrinsicoid deflection has

been considered as representing depolarization of that portion of the ventricular myocardium beneath the exploring electrode of a unipolar precordial lead, present opinion regards the intrinsicoid deflection as representing the turning point of a cardiac vector along the axis of derivation of the lead." (48)

Without particular regard for its theoretic implications, many workers have, in general, considered a delayed or prolonged ventricular activation time or pre-intrinsicoid deflection or intrinsicoid deflection as suggestive or characteristic of right ventricular hypertrophy. (15, 23, 43, 74, 91, 117, 126,) Scott (1960) stated, "Delay in the onset of the intrinsicoid deflection in the right precordial leads in right ventricular hypertrophy has constituted an integral part of the conventional criteria of ventricular hypertrophy. Although the exact significance and importance of the intrinsicoid deflection is at the present time still uncertain, it is an empiric observation that the onset is delayed in at least some cases of ventricular hypertrophy. " (103) Carter (1964)

noted that there was an "unexpected high correlation of the ventricular activation time with right ventricular wall thickness" in his autopsy controlled series. (31) Friedman (1963) stated that the time of onset of the intrinsicoid deflection is delayed in the right precordial leads in right ventricular hypertrophy because the vectors representing activation of the right ventricle usually occur later in the QRS interval then they do normally and are of increased magnitude. (48)

The following workers were more specific, in terms of actual time, in their consideration of ventricular activation time prolongation in right ventricular hypertrophy. As a point of reference, in the right sided precordial leads, such as V1 or V2, the time of onset of the intrinsicoid deflection has been stated to normally be 0.03 seconds or less. (48) Goodwin (1952), Scott (1960) and Orme (1952) considered a ventricular activation time greater than 0.03 seconds in V1 to be due to right ventricular hypertrophy. (52, 103, 95) Nadas (1957) described an intrinsicoid deflection of 0.03 seconds or more in V1 or V2, if

not associated with bundle branch block, as good evidence of right ventricular hypertrophy. (94) Myers (1948) included in the precordial pattern which he considered diagnostic of right ventricular hypertrophy an abnormally long ventricular activation time which he stated was between 0.03 and 0.05 seconds. (91) Sodi-Pallares (1956) stated that the intrinsicoid deflection was usually delayed greater than 0.035 seconds in right ventricular hypertrophy. (110) Schleris (1963) considered the intrinsicoid deflection in V1 from 0.035 to 0.05 as diagnostic of right ventricular hypertrophy. (102) Grant (1957) described the R wave in V1 as prolonged to 0.04 seconds or longer with right ventricular hypertrophy. (59) (It should be noted here that the R wave duration is not actually the same as the ventricular activation time.) Sokolow and Lyon (1949) used a ventricular activation from 0.04 to 0.07 in V1 and/or V2 as one of their criteria for right ventricular hypertrophy. Hollman (1958) considered the onset of the intrinsicoid deflection in V1 of 0.04 seconds or greater in the absence of right bundle

branch block in those from one month to 15 years of age to be diagnostic of right ventricular hypertrophy. (62)

One can only speculate on why an upper limit of prolongation of the ventricular activation was chosen in some of the above references, but it seems reasonable to assume that this is to rule out cases with right bundle branch block. According to Friedman (1963) in right bundle branch block the time of onset of the intrinsicoid deflection in V1 or V2 is delayed to 0.07 to 0.08 seconds. (48) Further discussion of the diagnosis (of the diagnosis) of right ventricular hypertrophy in association with right bundle branch block has been included in an earlier part of this paper.)

In the study of isolated pulmonary stenosis, Sundell (1957) substantiated the delay in intrinsicoid deflection as a criteria for right ventricular hypertrophy in his series. (117) Blount (1954) found the intrinsicoid deflection greater than 0.03 seconds in V1 in 9 of 10 patients with isolated valvular pulmonary stenosis without regard to the severity of the disease. (122)

Bentiviglio (1960) described an increasing prolongation of the ventricular activation time in the right precordials associated with increasing severity of stenosis. (8) Lantman's (1954) study on the electrocardiogram of patients before and after surgical repair of their pulmonary stenosis revealed that the intrinsicoid deflection in V1 decreased to normal in post-operative electrocardiograms. (77)

Consideration of the relationship of right ventricular pressure and intrinsicoid deflection led Blacker (1951) to state, "Some relationship should exist between the intrinsicoid deflection and the right ventricular pressure, for the right ventricular pressure must be a measure of the stenosis, while the intrinsicoid deflection is related to the muscle mass of the ventricle. However the intrinsicoid deflection is somewhat age related and patients may show a large variation in right ventricular pressure on account of anxiety. In spite of these deficiencies, measurement of the intrinsicoid deflection in lead V1 does give a fairly good indication of the height of the right

ventricular pressure and the severity of the disease." (9) Engle (1960) found a delayed intrinsicoid deflection in V1 in almost all cases with a right ventricular systolic pressure greater than 50 mm Hg. and therefore considered it relatively insensitive in distinguishing mild, moderate and severe cases of pulmonary stenosis. (45) Taussig (1960) described a slight delay in the onset of the intrinsicoid deflection appearing in right ventricular "strain" as the pressure in the right ventricle increased to levels over 120 mm Hg. (118) Friedman (1963) stated that the measurement of the time of onset of the intrinsicoid deflection has limited value in the diagnosis of right ventricular enlargement because, when it is delayed, the electrocardiogram usually shows other abnormalities which permit the diagnosis to be made. (48)

Cosby (1952) studied cases of congenital heart disease and elevated right ventricular pressure and could find no mathematical relationship of the pre-intrinsicoid deflection and the right ventricular pressure. (34) Similarly Jewett (1960)

concluded that progressive prolongation of the intrinsicoid deflection could not be correlated with right ventricular elevation. (66)

Finally, Milnor (1957) stated that he does not make measurements of the intrinsicoid deflection because he believes that it is both "misleading in theory and of no value in practice." (40)

THE RIGHT PRECORDIAL Q WAVE: Although consideration of an initial downward deflection in the right precordial QRS complexes has not taken a major place among the classic signs of right ventricular hypertrophy, it has been considered by a number of workers in its association with right ventricular hypertrophy, as well as pulmonary stenosis and/or elevated right ventricular pressure.

Schlerlis (1963) and McGregor (1950) considered the Q wave in V1 and/or V3R as diagnostic of right ventricular hypertrophy. (102, 89) Wilson (1944), Myers (1948, 1950) and Brumlik (1958) considered the right precordial Q as characteristic of right ventricular hypertrophy. (127, 91, 92, 74) Myers (1950), however, added that such

a change can occur and thus be confused with an anterior myocardial infarction. (92) Nadas (1957) described the Q wave in V4R or V1 as indirect evidence of right ventricular hypertrophy. (94) Hollman (1958) used the presence of a Q in V1 as a criterion for right ventricular hypertrophy in patients from one month to fifteen years of age. (62)

Cabrera (1963) described a very tall single R wave with a notched tip with a tiny initial and/or terminal negativity (Q and/or S wave) in V1 as commonly associated with severe right ventricular systolic overloading. (26)

Friedman (1963) considered the qR complex in the right precordials to be a variant of the "R pattern", with the difference being that the initial septal vector, instead of being directed rightward and anteriorly, is oriented slightly leftward. He stated, "Why this should occur has never been adequately explained." Nevertheless he felt that in adults and children (age limit was not specified) a qR or qRs pattern in leads V1 and/or V3R is diagnostic of right ventricular

enlargement regardless of the amplitude of the deflections, provided pre-excitation (Wolff-Parkinson-White syndrome), myocardial infarction or the combination of myocardial infarct and right bundle branch block are ruled out. (48)

Two reports were found in which the right precordial Q wave was associated with right ventricular pressure. Bentiviglio (1960) studied cases of pure pulmonary stenosis and found that the incidence of Q waves in V3R and V1 increased with increasing severity of stenosis and increasing right ventricular pressure. (8) Luna (1961) studied patients with pulmonary hypertension and reported that when the right ventricular pressure was more than 15 mm Hg. greater than the systemic pressure, a Q wave in V1 was present in 78% (of 28 cases) whereas when the right ventricular pressure was no more than 15 mm Hg. different from the systemic blood pressure, the Q wave was present in only 5% (of 114 cases), and when the right ventricular pressure was more than 15 mm Hg. less than systemic pressure, the Q wave was not present in any of 85 cases. (84)

Consideration of the etiology of this pattern led Human and colleagues (1964) to comment, "It seems to us the rsR' and qR patterns in V1 are essentially similar patterns and probably represent hypertrophy of the outflow portions of the crista regions of the right ventricle." They observed that with simultaneous recording of a right precordial lead with V6 that the q wave in the former lead followed the q wave in V6 by 0.01 seconds. They reasoned that the initial r of the rsR' pattern may be lost in the preceeding isoelectric line during transmission to the precordium. (64) Myers (1956) and Wasserberger (1958) also considered the q wave as an atypical expression of an incomplete right bundle branch block. (93, 121)

According to Wilson and associates (1947), the initial q wave in V1 could be due to a "decreased density of the junction between Purkinje and ordinary muscle in certain areas as a result of dilitation of the chamber. " (128) Lepeschkin (1951) stated that the q wave in V1 corresponds to electrical forces due to activation of the

septum from the right to the left, "...which are no longer opposed by those due to radial activation of the lateral wall of the right ventricle, as these appear later and are perhaps smaller due to decreased density of transitions between the conducting system and myocardium as a consequence of dilitation." (80)

According to Sodi-Pallares, Bisteni and Herrmann (1952) the presence of qR complexes in the right precordial leads may present in congenital and other cardiopathies having an enlarged right atrium. They have postulated that if the right atrium is enlarged enough the electrodes located in the right precordium face the epicardium of the right atrium instead of the right ventricle and register the potentials normally encountered on the surface or inside of the right atrium. But they added, "That this configuration is considered evidence of right ventricular hypertrophy rests simply on the clinical and pathologic fact that enlargement of the right atrium almost invariably is accompanied by hypertrophy of the corresponding ventricle." (108)

THE LEFT PRECORDIAL QRS IN RIGHT VENTRICULAR HYPERTROPHY AND PULMONARY STENOSIS

As reversal of the normal right precordial pattern (from a predominantly negative QRS complex to one which is more positive) has been described as characteristic of right ventricular hypertrophy, similarly there has been general agreement in the literature describing reversal of the normal left precordial pattern. Thus descriptions of the left precordial leads in patients with right ventricular hypertrophy have included, in general, decrease in the amplitude of the R wave, increase in the depth of the S wave and a resultant decrease in the R/S ratio. (34, 38, 83, 74, 75, 89, 81, 49, 127)

Bentiviglio and co-workers (1960) concluded from their studies on cases of isolated pulmonary stenosis that the duration and amplitude of the S wave increased and the amplitude of the R wave decreased in association with increasing severity of stenosis. (8) With regard to the significance of the increased duration of the left precordial S wave, Wasserburger (1962) noted, "Although the

rR' or rsR' pattern in right precordial QRS complexes are identified with both right ventricular preponderance and incomplete right bundle branch block, the broad left ventricular S wave of incomplete right bundle branch block aids in differentiating them on a conventional electrocardiogram." (121)

Actual criteria for the maximum depth of the normal left precordial S wave were established by Sokolow and Lyon (1949) and Orme and Adams (1952) at 7 mm in V5 or V6. (115, 95) Braunwald (1955) established the maximum normal S wave in lead V5 by age as follows: less than one year-30mm, one to ten years-13mm, ten to twenty years-11.3mm, and greater than twenty years- 14.3mm.

Goodwin (1958) noted that while the commonest cause of a deep S wave in V5 is isolated or dominent right ventricular hypertrophy, it may also occur in anterior infarction with or without right ventricular hypertrophy. Thus any electrocardiogram showing an rS pattern in V5 or V6 should be interpreted with caution, and the possibility of a concealed infarct should be considered. (53)

Criteria for the minimum normal R wave in the left precordial leads were established by Sokolow and Lyon (1949) at 5 mm and by Braunwald and co-workers (1955) according to age as follows: less than one year-0 mm, from one to ten years-5 mm, from ten to twenty years-3.5mm, and greater than twenty years-2 mm. (15)

Consideration of the R/S ratio in V5 or V6 in adults has led a number of investigators to regard ratios less than one as indicative of right ventricular hypertrophy. (75, 95, 115) Hollman (1958), considering the R/S ratio with regard to age, stated that the R/S ratio in V5 in infants under three months of age normally is always 0.6 or greater, while in right ventricular it is 0.5 or less—this being of particular value in the diagnosis of right ventricular hypertrophy in the very young. His criteria for right ventricular hypertrophy included an R/S ratio equal or less than the following: from one to three months-0.5, from four to eleven months-0.7, from one to two years-0.8, and from three years through adulthood-0.9. (62)

Engle (1960) found in his series of cases of pulmonary stenosis that when there was reversal of the normal R and S wave relationship across the precordium so that the S wave was the predominent deflection in V5 or V6, the right ventricular pressure was 100 mm or more. (45)

The left precordial ventricular activation time or pre-intrinsicoid deflection does not appear to have attained the prominence in the literature as that of its right precordial counterpart. Three articles were found, however, which did describe it as being relatively short or relatively early in right ventricular hypertrophy. (34, 91, 127)

THE PRECORDIAL "rS" PATTERN

The "rS" pattern cannot rightly be included in the discussion of either the right or left precordials, as its presence can be evaluated only in terms of all the precordial leads. Thus it will be considered separately.

This pattern, as suggested by its name, consists of an initial small positive deflection (r wave) followed by a relatively large negative deflection (S wave) seen in all the precordial leads. Though this pattern in the left precordials corresponds more or less with what many consider characteristic of right ventricular hypertrophy, its right precordial pattern does not conform to the characteristics usually ascribed to right ventricular hypertrophy. Nadas (1957) stated that he considered the least certain indication of right ventricular hypertrophy in the chest leads to be marked rotation of the heart resulting in rS or RS deflections from V1 to V6. (94) Kossmann (1962) described small r and deep S waves extending across the precordium as possibly indicating dilitation

or hypertrophy of the trabecular region or inflow tract of the right ventricle. (75) Scott (1962) noted that anterolateral myocardial infarction may produce an rS pattern in the left precordial leads and falsely suggest right ventricular hypertrophy. (75) According to Friedman (1963) the rS pattern in all the precordial leads is suggestive but not diagnostic of right ventricular enlargement. He described it as also being found in emphysema. (48)

ST AND T WAVE CHANGES IN RIGHT VENTRICULAR HYPERTROPHY AND PULMONARY STENOSIS

GENERAL ASSOCIATION OF RIGHT VENTRICULAR HYPERTROPHY WITH T WAVE CHANGES: Wilson (1944) stated that in preponderant hypertrophy involving either ventricle, the T waves of the leads in which the largest R waves occur are very commonly inverted. More specifically he described the T wave in right ventricular hypertrophy to be characteristically inverted in the right precordium. (127) Similarly Littmann (1949) described inverted T waves in V1, V2, and V3 as characteristic of right ventricular hypertrophy, whereas Myers (1948) found only a "...tendency to have inversion of the T in V1." in right ventricular hypertrophy. (83, 91)

GENERAL ASSOCIATION OF PULMONARY STENOSIS WITH ST AND T WAVE CHANGES: Marquis (1951) described ST depression and inverted T waves extending across the chest towards the left as the usual features of pulmonary stenosis. (86) Sodi-Pallares (1956) stated that T wave changes occur only in pulmonary stenosis of marked severity. In such

cases the T in V1 and other right precordial leads is flattened or negative and in extreme cases is "ischemic", that is, deep and peaked—the latter being highly suggestive of pure pulmonary stenosis. He added that with increasing severity of pulmonary stenosis the frontal axis of the T wave deviates to the left in opposition to the frontal mean axis of the QRS which deviates to the right. (110) In 1959 he qualified his previous comments on severity of pulmonary stenosis by stating that, "With severe stenosis the T waves in the right precordial leads are usually positive when the right ventricular systolic pressure is lower than the systemic pressure." (113)

Taussig (1960) stated that in cases of pulmonary stenosis as the pressure in the right ventricle increases to 120 mm Hg. the pattern of right ventricular "strain" develops in which the T wave becomes inverted across the left precordium. (118) Engle and colleagues (1960) found that when right ventricular systolic pressure in cases of pulmonary stenosis was over 140 mm Hg. there was deep inversion of the T waves in the right pre-

cordial leads, aVF and often in leads II and III. They also noted that the changes in the T waves were frequently associated with ST segment depression in the same leads. (45)

In Lantman's study (1954) on post-operative changes after repair of pulmonary stenosis, he found that the ST segment depression and T wave inversion in leads II, III, aVF, and V1 disappeared. (77)

Goodwin (1952) discounted the importance of T wave changes. He concluded, "Inversion of the T wave in precordial leads V1 to V3 was not found to be a valuable sign of right ventricular hypertrophy, since it was often present in normals. Conversely, the T was often upright in the same leads in right ventricular hypertrophy." (52)

T WAVE CHANGES IN ASSOCIATION WITH DIFFERENT DEGREES OF SEVERITY OF RIGHT VENTRICULAR HYPERTROPHY: In 1933, before the present standard precordial lead system was well established, Wilson described the T wave changes in right ventricular hypertrophy only in the standard leads. He noted that right ventricular hypertrophy with inversion of the T

waves in leads II and III is associated with the largest hearts and with the greatest increase of pressure in the pulmonary circuit. According to Wilson, next in severity is the right ventricular preponderance with only T inversion in lead III and least in severity is the patient with right ventricular preponderance without any inversion of the T wave. (87) Only two other authors were found whose statements about T wave changes in right ventricular hypertrophy could be implicated in the standard and augmented leads—both making statements using general vectorial analysis. Grant (1957) stated that there is little change in the direction of the ST and T vectors in right ventricular hypertrophy. However, he noted that when hypertrophy is marked the ST and T vectors may point leftward and posteriorly, away from the lie of the right ventricle and opposite to the direction of the mean QRS vector. Friedman (1963) described similar changes which he called the "strain pattern". (59, 48)

All of the remaining literature reviewed, with regard to T wave changes, confined its

comments to T changes in the precordial leads. Abrahams (1951) and Blacket (1951) both described T inversion in V1 and the extension of this inversion across the chest to V3, V4, or even V5 as suggesting increasing severity of right ventricular hypertrophy. (2, 9) In conjunction with his comments on the precordial T wave changes, Blacket commented, "The precordial leads have furnished the most direct and convincing evidence of right ventricular hypertrophy." (9) Brumlik (1958), Nadas (1957), Cabrera (1959) and Silverman (1956) made similar observations correlating the inversion of the T wave and depression of the ST segment across the precordium toward the left with increasing severity of right ventricular hypertrophy. (74, 94, 25, 107) Cabrera (1963), in describing an "almost stereotyped electrocardiogram for cases with systemic pressure values in the right ventricle", listed a negative T wave in V1 and a minus-plus T wave in lead V2 as being usually present and accompanied by a depressed ST segment. He concluded, "The diagnostic value of these is not infallible, but clear separation of these patterns may suggest the

diagnosis." (26)

Caylor (1958) found that patients with right ventricular pressures of 150 mm Hg. or higher almost always had ST and T wave changes. (30) Silverman (1956) observed that none of his patients with right ventricular pressure less than 100 mm Hg. showed abnormal ST or T wave changes. (107) Campbell and co-workers (1954) studied cases of "simple pulmonary stenosis with closed ventricular septum" (including some cases with atrial septal defects) and noted that, "Right ventricular preponderance was found in all the cyanotic group, generally with deep T inversion across the chest leads to V4 and sometimes to V5 and V6." But in the acyanotic group the T inversion across the chest leads was "less constant, but still common." In addition they found that in all the patients with T inversion to V4, whether cyanotic or acyanotic the right ventricular systolic pressure was over 100 mm Hg. and often greatly over this. There were however some where this pattern was absent although the systolic pressure was greater than 100 mm Hg. (29b) In conjunction with T wave

changes and their relationship with cyanosis, Kjellberg (1955) stated that a negative T wave in the precordial leads in an adult is not a reliable indication of the degree of severity of pulmonary stenosis. He noted that these changes have occurred chiefly in patients with severe cyanosis. (71)

T WAVE CHANGES IN CHILDHOOD AND THEIR RELATION TO RIGHT VENTRICULAR HYPERTROPHY: All of the previous comments on T wave changes either implicitly or explicitly referred to the electrocardiogram in the adult, with the possible exception of Nadas (1957) from which the reference source was his textbook Pediatric Cardiology. (94) After a review of the T wave in the normal infant and child and notation of its marked difference from the normal adult T wave pattern, the following comments referring to both the normal and abnormal infant and child T waves seem noteworthy.

Kjellberg (1955) made the following comments regarding the inverted right precordial T wave in children, "A negative T wave in V1 through V4 has

been regarded as highly characteristic of severe pulmonary stenosis and often decisive as an indication for operation. In children the correlation between the negative T wave in different precordial leads and the right ventricular pressure is not so evident, since a negative T may occur normally in V4 up to five years of age and in V3 up to ten." (71) Engle (1960) similarly stated, "The leftward spread of T wave inversion might be expected to be more reliable in adults than in children, since T wave inversion may be normal through V4 in the latter. " (45)

Gros (1951) described normal T wave alterations as follows: in children less than 24 hours old the T is upright or diphasic in V4R, V1 and V2 and inverted in V4, V5 and V6; thereafter, there is a gradual reversal of the T wave direction so that at the fourth day all have inverted T waves in V4R, V1 and V2 and upright T waves in V5 and V6. (60)

Whereas the negative or inverted T wave has been regarded by some as a significant sign associated with right ventricular hypertrophy in the

adult, the positive or upright T has been similarly associated in the child. Cabrera (1959) noted that a positive T in the right precordials in patients less than 10 years old, with minimal or absent alterations of the QRS complex, is valuable for detecting a mild hypertension of the right ventricle (systolic pressure about 40 mm Hg.), and in a later report (1963) he stated that an upright T wave in the right precordial leads after 2 weeks of age usually means "systolic overloading of the right ventricle." (25, 26) Kossmann (1962) and Scott (1960) included in their criteria for right ventricular hypertrophy in infants and children a positive T in V1 after the first 48 hours of life. (75, 103) Nadas (1957) described a positive T in V1 beyond the first 24 hours as suggestive of right ventricular hypertrophy. (94) Kieth (1958) described the positive T wave in V1 between 1 and 12 years of age as suggestive of right ventricular hypertrophy. (70) Friedman (1963) felt that a positive T wave in lead V1 from the third or fourth day of life till the twelth year, particularly when

associated with an R/S ratio greater than 1.0 in this lead, is strongly suggestive of right ventricular enlargement. (48)

Ziegler (1956) found that T waves are normally positive in the right precordial leads during the first 24 hours of post-natal life, that there is progressive inversion of this deflection during the subsequent three to five days, and that only in a small percentage of cases is it again positive after the first decade. He postulated that the most likely explanation for these T wave characteristics is the neonatal pulmonary artery and right ventricular hypertension incident to incomplete expansion of the lungs and later decreasing arteriolar resistance secondary to the assumption of progressively normal function. He observed that within a given QRS pattern there is a direct relationship between the incidence of positive T waves in the leads from the right side of the precordium and the degree of elevation of right ventricular mean pressure. He concluded, "The occurrence of positive T waves in the right precordial leads after the first 24 hours of life is

highly suggestive—if not actually diagnostic—of right ventricular hypertension and therefore pathologic right ventricular hypertrophy of the so-called "systolic-overload" or "pressure-work" type." He added that "...the earliest recognizable and least complicated precordial lead pattern of pressure hypertrophy of the right ventricle, particularly in infants, consists of a single peaked RS complex...followed by a positive T wave in the right precordial leads," and he considered this pattern to represent minimal to moderate right ventricular hypertrophy. However, with greater degrees of right ventricular hypertrophy, as observed in marked pulmonary stenosis with a closed interventricular septum, he found that there is frequently a pattern of right bundle branch block (notched, or double peaked R) and inverted instead of upright T waves in the right precordium. He stated that it has been suggested that in this latter type of precordial lead pattern some estimate of the degree of right ventricular enlargement can be made by the extent of T wave inversion across the precordium.

The final paragraph of Ziegler's paper follows in its entirety as it is particularly appropriate to this discussion.

> "This discussion of the importance of positive T waves in right precordial leads would not be complete without an added word of caution. Throughout the discussion it has been understood, of course, that the T wave patterns described are observed in patients in whom the clinical condition is consistent with the presence of single or combined cardiac chamber enlargement of the type specified. The occurrence of T wave positivity in right precordial leads as an isolated or inconsistent finding cannot necessarily be interpreted in the manner described, since there are other factors which may also produce this patterm. Especially in older children and adults it may be entirely normal, as it so frequently is during the first day of life. It may also occur in such diverse situations as hyperpotassemia or coronary artery occlusion with posterior wall myocardial infarction. The word of caution is, therefore, that one must interpret the electrocardiographic observations discussed, as one should data obtained by practically any laboratory procedure, in the light of the clinical situation and not as isolated information." (130)

P WAVE CHANGES IN RIGHT VENTRICULAR HYPERTROPHY AND PULMONARY STENOSIS

The occurrence of large peaked P waves has been correlated with cases of pulmonary stenosis by numerous investigators. (3, 9, 7, 11, 29, 38, 43, 45, 59, 66, 71, 77, 86, 94, 102, 107, 110, 111, 118, 129) However Jewett's (1960) correlation was decidedly minimized by his conclusion that "P wave abnormalities are minimal in the electrocardiograms of patients with pulmonary stenosis." (66) In general the occurrence of such waves is considered an electrical manifestation of right atrial hypertrophy, (48, 94, 110, 123) but this has also been considered as indirect evidence of right ventricular hypertrophy. (43, 48, 62, 94)

The prominence of this change has been ascribed by most to be most evident in lead II. (3, 9, 29, 62, 86, 94) Hollman (1958) considered a P wave of 3 mm or more in lead II or 2.5 mm or more in any other lead a criteria for right ventricular hypertrophy from one month of life through adulthood. (62) Others also mentioned its

prominence in lead I, III, aVf, and the right precordial leads. (11, 38, 48, 71, 110) Sodi-Pallares (1956) described this change in pulmonary stenosis as "...usually in I and II, but sometimes in II and III," implying a left axis shift of the mean P vector rather than a right axis shift. (110) On the other hand, Friedman (1963) described a right axis shift of the mean electrical axis of the P wave as characteristic of right atrial enlargement. (48)

Sodi-Pallares (1956) noted, "These P changes are related not only to right atrial enlargement, but also to low oxygen saturation in blood i.e. under 70 %." (110) Wood (1950) inferred an association with oxygen saturation when he stated, "P pulmonale is common, therefore not very helpful in all cyanosed cases (of congenital heart disease), but in acyanotic cases it usually means pulmonary stenosis or severe pulmonary hypertension." (129)

Numerous investigators have considered P wave changes in relation to right ventricular pressure. Taussig (1960) explained, "The P waves reflect the amount of work required of the auricle.

As the pressure in the right ventricle rises, the pressure in the right atrium also rises and the right atrium undergoes dilitation and hypertrophy. Under such circumstances the P waves are both broad and tall, consequently they resemble the Appalachian mountains rather than the tall sharp peaks of the Himalayas." (118) Bentiviglio (1960) concluded that the duration and amplitude of the P wave increases with increasing severity of pulmonary stenosis. (8) Blacket (1951) described a "surprisingly good correlation" between the amplitude of the P waves in lead II and the mean right ventricular pressure, the latter which he believed mirrored the severity of the pulmonary stenosis. He concluded that "...while moderate stenosis and normal sized P waves (less than 2 mm) can coexist, abnormally high P waves are a sign of severe stenosis provided they are not due to position of the heart." (9) Kjellberg (1955) concluded, "The hypertrophy and dilitation of the right atrium which occurs in severe pulmonary stenosis are reflected in a tall P wave over the right precordium, however the P wave may be normal even

in severe stenosis, and peaked P waves may be present in moderate stenosis." (71) Schleris (1963) stated that the increased amplitude of P waves in pulmonary stenosis was more frequent in those with the highest right ventricular pressures. (102) Braunwald (1955) described 20 patients with pulmonary stenosis of which only 3 had abnormal P waves. Each of these patients had right ventricular pressures greater than 120 mm Hg. (15) Silverman (1956) concluded that there was a good correlation of P pulmonale and right ventricular hypertrophy when the right ventricular pressure was greater than 100 mm Hg. Conversely none of his 25 patients with right ventricular pressures below 100 mm Hg. showed P pulmonale. (107) Engle (1960) described high peaked P waves as appearing when the right ventricular pressure was over 140 mm Hg. in cases of pulmonary stenosis. (45)

In support of the concept of P wave correlation with pulmonary stenosis, Lantman (1954) described a decrease in the P wave to normal amplitude as a typical post-operative change after pulmonary stenosis repair. (77)

DePasquale (1960) noted that the P wave tended to be prominent, but he concluded that its magnitude showed no significant relationship to the pressure in the right ventricle. (38) Finally in Jensen's (1960) study of the "False-Positive Electrocardiogram" he described increases height of the P wave in association with emotional stresses, exercise, ingestion of food, smoking and hyperventilation. Naturally increased amplitude of the P wave secondary to these factors may suggest right atrial hypertrophy and confuse the interpretation in hearts where this artifact is marked. (65)

MISCELLANEOUS CHANGES OF THE QRS IN RIGHT VENTRICULAR HYPERTROPHY AND PULMONARY STENOSIS

QRS DURATION: Relatively little information was found in the literature relating QRS duration, per se, and pulmonary stenosis and/or right ventricular hypertrophy, suggesting its probable lack of importance in this regard. Bentiviglio (1960) noted that the duration of the QRS increases with increasing severity of pulmonary stenosis.(8) Wilson (1944) stated that in right ventricular hypertrophy, "...the QRS interval is rarely much greater than normal." (128) Barker (1952) concluded that the QRS duration is not ordinarily prolonged, "...because the pathway of excitation, the right free wall is not, except in extreme right ventricular hypertrophy, as long as it is in the left free wall." (5) Bayley (1958) explained, "Activation of the right ventricle is normally completed in about 0.06 seconds or less. Therefore a noraml QRS of 0.09 seconds would not become prolonged until marked right ventricular hypertrophy had developed. In fact, ordinarily in uncomplicated right ventricular hypertrophy the

QRS interval should not be prolonged beyond 0.10 seconds." (7) Sundell (1957) concluded that no correlation could be found between the QRS interval and the increased right ventricular pressure and right ventricular hypertrophy associated with pulmonary stenosis. (117) DePasquale (1960) noted that the QRS interval was prolonged in association with the right ventricular hypertrophy of atrial septal defects, however he observed that it was not prolonged in pulmonary stenosis. (38)

Further consideration of the QRS duration has been made in the earlier part of this paper reviewing incomplete and complete bundle branch block.

QRS CHANGES IN THE STANDARD AND LIMB LEADS: In considering both the standard leads (I, II, and III) and the limb leads (aVR, aVL, and aVF) with regard to unipolar interpretation (c.f. vector interpretation), probably lead aVR has received the most attention in the electrocardiographic literature in reference to changes seen in right ventricular hypertrophy. Myers(1948) noted that lead aVR is often equivical or normal when classical signs of right ventricular hypertrophy are

demonstrable in V1, but on the other hand, it may exhibit an unusually prominent and definitely abnormal R wave when normal findings are present in V1 and V2. He added, however, that the diagnosis of right ventricular hypertrophy cannot be based upon the findings in aVR, in the absence of any supportive findings in the precordial leads. (91)

Marquis (1951) described a tall R in aVR as a usual feature of pulmonary stenosis. (86) Nadas (1957) considered a dominent R or R' in aVR as characteristic of right ventricular hypertrophy. (94) Scott (1960) stated that the dominent R in aVR is a useful criterion for the diagnosis of right ventricular hypertrophy in those under age 3. (103) Wasserburger (1962) said, "A tall predominent late R wave in aVR serves to denote an advanced degree of right ventricular hypertrophy, whether the classic patterns of right ventricular preponderance or an incomplete right bundle branch block associated with right ventricular preponderance presents on the routine electrocardiogram." (122)

Engle (1960), found a predominent R in aVR was

present in almost all cases of pulmonary stenosis with right ventricular pressures greater than 50 mm Hg. and therefore it was considered relatively insensitive in distinguishing mild, moderate and severe cases.

Sokolow and Lyon (1949) and Schleris (1963) both considered an R in aVR greater than or equal to 5 mm in height as one of their criteria for right ventricular hypertrophy. (115, 102) Orme and Adams (1952) described an R in aVR greater than 5 mm in those less than 5 years of age and greater than 3 mm in those over 5 years among their criteria for right ventricular hypertrophy. (95) Braunwald (1955) made the diagnosis of right ventricular hypertrophy when the R in aVR exceeded the following maximum normals: under one year-9 mm, from one to ten years-19.5mm, from ten to twenty years-8 mm, and over twenty years of age-3 mm. (15)

Hollman (1958) considered the R in aVR in relation to the whole QRS complex in this lead and established that an R/S or R/Q ratio greater than 1.0 indicated right ventricular hypertrophy

in those one month or older. (62) Goodwin (1952) turned this ratio around and stated, "A Q/R ratio less than one in aVR, although in itself merely a sign of extreme clockwise rotation or backward tilting of the heart, is a highly suggestive indirect sign of right ventricular hypertrophy, although its absence does not exclude the diagnosis. It is a particularly important sign in cases in which V1 is normal." (52) Lantman (1954) described a decrease to normal in the R/Q ratio in aVR in patients with pulmonary stenosis after surgical repair. (77)

In 1914 Lewis suggested a method of obtaining an index of ventricular predominence by adding the value RI minus RIII to the value SIII minus SI. (82) In 1918 White and Bock considered this same index in the following formula: Index = (UI plus DIII) minus (DI plus UIII), where U indicates the amplitude of the primary upward deflection and D, the amplitude of the primary downward deflection. They found values of plus 20 and minus 15 to be close to the borderline of normality. They considered values of minus 15 to minus 18 usually

indicate right ventricular preponderance, and beyond minus 18 always indicate right ventricular preponderance. (123) In 1924 White and Burwell revised these figures to minus 10 rather than minus 15 as borderline normal with respect to right ventricular preponderance. (124) Pardee (1920) stated, "Of all the formulas which have been considered for determining the ventricular predominence from the electrocardiogram, the simplest to apply and the one giving the smallest apparent error is the formula suggested by Lewis of (RI plus SIII) minus (SI plus RIII)." (96) Sodi-Pallares (1956) stated that the Index of Lewis (which he equated with the net voltage in lead III subtracted from the net voltage in I) has been very useful to resolve the question of whether a given axis deviation is due to ventricular hypertrophy or due to body constitution. He also mentioned use of the Jinich Index in which Index = (UaVL plus DaVF) minus (UaVF plus DaVL) and stated that values under minus 11 were suggestive of right ventricular hypertrophy. (110)

The value of measuring the amplitudes of

deflections in the limb leads was commented on by Engle (1960); "Amplitudes of the R and S deflections in the limb leads were not tabulated separately since they bore a close resemblance to the amplitudes described in the right and left precordial leads. Patients with an abnormally tall R wave in V1 and deep S in V6 showed a tall R in aVF and lead III and a deep S in lead I." (45)

Only a few workers, in addition to the one just mentioned above, have described characteristic changes in the individual limb or standard leads that are characteristic of right ventricular hypertrophy. Nadas (1957) noted that a deep S in lead I and a tall R in III is suggestive of right ventricular hypertrophy. (94) Cabrera (1959) described systolic overloading of the right ventricle as characteristically producing an rS pattern in lead I and a qR pattern in III. (25) Sodi-Pallares (1956) stated that with right ventricular hypertrophy lead aVL shows a small R wave and deep S wave, and aVF shows a prominent R wave preceded by a q wave. (110)

Bentiviglio (1960) stated that he found the

duration of the S wave in lead I increased in association with increased severity of stenosis in his series of cases of pure pulmonary stenosis. (8)

The pattern of small r waves and large S waves in the standard leads I, II, and III has been associated with pulmonary stenosis specifically (111, 114), and right ventricular hypertrophy, in general. (48, 59, 94, 126) Grant (1957) and Friedman (1963) described the $S_1S_2S_3$ syndrome as being characterized by: (1) predominently negative deflections of the S wave type in the three standard leads, (2) a normal QRS interval, and (3) usually a small r' deflection at V1. From a vector standpoint, this was described as having the terminal QRS vector directed rightward, superiorly and usually slightly anteriorly. It was stated that the syndrome is seen under five different circumstances: (1) in young adults with no detectable evidence of heart disease, (2) in congenital heart disease when associated with right ventricular hypertrophy, (3) in severe cor pulmonale, (4) in emphysema with or without cor pulmonale, and (5) in acute myocardial infarction. Grant added that

this syndrome is supportive, but by no means diagnostic of right ventricular hypertrophy, unless the terminal QRS vector is also greatly increased in magnitude. (59, 48)

SUMMARY

INTRODUCTION:

The purpose of this paper has been to glean from the literature a discussion of all the EKG changes that have been ascribed to isolated right ventricular hypertrophy.

Emphasis was placed on isolated pulmonary stenosis, as it represents an ideal prototype as the etiology of isolated right ventricular hypertrophy.

This paper was intended only as a review of the literature's discussions and conclusions. It was not intended to be a critical review.

GENERAL COMMENTS ON EKG ANALYSIS:

The EKG has been considered with very high regard as a clinical tool, with due regard for its weaknesses.

Opinions are highly variable on the correlation of EKG and anatomic evidence of right ventricular hypertrophy. Probably less than 70% with autopsy evidence of right ventricular hypertrophy will show EKG evidence of this. EKG evidence is more likely to be

substantiated at autopsy.

There has been a general association of elevated right ventricular pressure and right ventricular hypertrophy and EKG changes associated with right ventricular hypertrophy. The degree of positive correlation in individual cases is controversial—the preponderance of opinion being that it is quite variable.

There is a progressive change in the EKG from infancy to old age such that what may be normal for one age group is abnormal for another. There is a gradual change from a predominance of right ventricular potentials at earlier ages to one of left ventricular potentials at older ages.

Various terms such as predominance, hypertrophy, preponderance and enlargement are used in the literature more or less synonymously with regard to the EKG.

The value of vector versus unipolar or pattern interpretation is quite controversial. Most investigators use both methods.

QRS VECTOR FACTORS IN RIGHT VENTRICULAR HYPERTROPHY AND PULMONARY STENOSIS:

Opinion has varied on the value of axis deviation from one extreme in which right axis deviation (RAD) is equated with right ventricular hypertrophy, to a more moderate position in which a general correlation of RAD is made with right ventricular hypertrophy with awareness that RAD may sometimes be due to other causes, to the other extreme in which RAD has no correlation with right ventricular hypertrophy. Most have taken the previously mentioned moderate position.

Generally in an adult a mean frontal QRS axis at about 90° first suggests right ventricular hypertrophy and one at 110° or beyond is considered fairly diagnostic.

For children axis deviation to the right of 120 to 140° has been considered characteristic of right ventricular hypertrophy in those older than one month.

Consideration of other causes of abnormal RAD includes: thin body build, abnormal position of the heart in the thorax, pulmonary emphysema,

myocardial infarction, cor pulmonale, left ventricular hypertrophy, and occasionally also normal hearts in younger individuals.

A few workers have considered the horizontal QRS vector loop characteristic of right ventricular hypertrophy if it rotates clockwise as viewed from above and left bundle branch block can be ruled out.

THE RIGHT PRECORDIAL QRS IN RIGHT VENTRICULAR HYPERTROPHY AND PULMONARY STENOSIS:

The EKG diagnosis of right ventricular hypertrophy has been based largely on criteria in the right precordial leads, including a relative increase in the height of the R wave, decrease in the depth of the S wave, and increase in the R/S ratio. The R/S ratio greater than 1.0 in adults in lead V1 is a very common criterion for right ventricular hypertrophy. Some correlated a progressive change in these parameters with progressively increased hypertrophy. The possibility of right ventricular hypertrophy in association with deep S waves was considered in milder cases of right ventricular hypertrophy.

Consideration of causes of the false-positive (tall) right precordial R wave have included: diastolic overloading of the left ventricle, technical error (e.g. recording of aVF), right bundle branch block, myocardial infarction and hiatus hernia.

The parameters of the QRS in the right precordium have been correlated with right ventricular pressure—the height of R in V1 generally being considered most valuable. An equal number have commented on the lack of good correlation here.

It has been noted that the R/S ratio in the majority of infants and children is greater than 1.0 during the first 3 to 5 years of life. Lack of an S wave or minimal S wave in the right precordials was considered very diagnostic of right ventricular hypertrophy.

The maximum normal R wave in the right precordium in the adult has been considered from 7 mm to over 15 mm. Between five and twenty years of age the maximum normal R wave ranged from 7 to 20 mm; from one to five years, 11 to 29 mm; and under one year, from 20 to 29 mm.

The maximum normal R/S ratio in the right precordium ranged from 1.0 to 1.5 in those from five through fifteen years of age; from three to five years, 1.6 to 2.0 was the range of maximal normal; from six months to three years 2.0 to 4.0 was considered; and for those under three months the maximum normal ranged from 4.0 to 7.0.

The occurrence of a secondary R wave in the right precordium has been correlated with pulmonary stenosis and right ventricular hypertrophy, and in most references it was considered in relation to right bundle branch block. Right bundle branch block was described as reflecting possible right ventricular dilitation, diastolic overloading of the right ventricle, right ventricular hypertrophy, right ventricular hypertrophy in association with left ventricular hypertrophy, localized right ventricular hypertrophy, intraventricular conduction defect, or possibly no heart disease at all. The height of the R' wave at which right ventricular hypertrophy has been considered in adults has ranged from 6 to 23 mm and in children greater than 10 mm. Other

criteria included R greater than 8 mm, R'/S greater than 1.0, and R/S greater than 1.0.

Many workers have considered a delayed or prolonged ventricular activation time (also known as pre-intrinsicoid or intrinsicoid deflection) as suggestive or characteristic of right ventricular hypertrophy. Abnormal delay ranged from greater than 0.03 or 0.04 seconds. Some found that the ventricular activation time could be directly correlated with right ventricular pressures; others found no correlation and no value in this parameter.

The right precordial Q wave has generally been considered diagnostic or at least characteristic of right ventricular hypertrophy—provided anterior myocardial infarction, pre-excitation, and right bundle branch block are ruled out. Some have found strong positive correlation with the incidence of right precordial Q waves and elevated right ventricular pressure. Other possible causes of right precordial Q waves have included dilitation of the right ventricular wall, and enlarged right atrium.

THE LEFT PRECORDIAL QRS IN RIGHT VENTRICULAR HYPERTROPHY AND PULMONARY STENOSIS:

There has been general agreement in the literature describing reversal of the normal left precordial pattern with decrease in the amplitude of the R wave, increase in depth of the S wave, and a resultant decrease in the R/S ratio. This pattern was also described following anterior infarction. The minimum normal R wave in the left precordials in adults ranged from 2 to 5 mm. The maximum normal S wave in adults ranged from 7 to over 14 mm. The minimum R/S ratio in adults ranged from 0.9 to 1.0; the minimum R/S ratio in children decreases progressive to a lower limit of 0.5 for those under three months of age.

THE PRECORDIAL "rS" PATTERN:

This pattern consists of a small r wave and relatively large S wave seen in all the precordial leads and has been considered as minimal evidence of right ventricular hypertrophy. It has also been described with anterolateral infarction.

ST AND T WAVE CHANGES IN RIGHT VENTRICULAR HYPERTROPHY AND PULMONARY STENOSIS:

There has been a general correlation of the presence of ST depression and T wave inversion in the right precordial leads in adults with right ventricular hypertrophy or advanced pulmonary stenosis. This has been referred to as the "strain pattern". Others have discounted the correlative value of these changes. Some have correlated these ST-T wave changes with decreased oxygen saturation.

The ST-T wave changes described for adults have generally been found of very little value in analysis of the EKG of the infant or child. The precordial T wave was described as being normally upright in the right precordials and inverted in the left precordials in the first 24 hours of life with a gradual reversal so that at the fourth day the right precordials have inverted T waves and the left precordials have positive T waves. The positive right precordial T wave has been strongly correlated with right ventricular hypertrophy in patients from 24 hours up to 10 years of age.

P WAVE CHANGES IN RIGHT VENTRICULAR HYPERTROPHY AND PULMONARY STENOSIS:

The occurrence of large peaked P waves has been correlated with right atrial hypertrophy secondary to right ventricular hypertrophy. This change has usually been considered most prominent in lead II. The enlarged P wave is usually described as peaked and greater than 2.5 to 3 mm in height. The peaked P wave was correlated by some with oxygen saturation. Others discreted the value of the P wave, and notation of its being abnormal with emotional stress, exercise, ingestion of food, smoking and hyperventilation was mentioned.

MISCELLANEOUS CHANGES OF THE QRS IN RIGHT VENTRICULAR HYPERTROPHY AND PULMONARY STENOSIS:

Relatively little information was found with regard to the QRS duration. Most considered the QRS to be usually normal in duration in right ventricular hypertrophy.

The presence of a tall R in lead aVR was noted to be of value in the diagnosis of right ventricular hypertrophy. The use of the Lewis and Jinich index was considered. Changes in the

standard and limb leads was generally consistent with changes in axis deviation and otherwise not too helpful—except that increased right ventricular potentials was correlated with increased likelyhood of right ventricular hypertrophy.

The $S_1S_2S_3$ syndrome was correlated with right ventricular hypertrophy, but also with normal hearts, cor pulmonale, emphysema, and acute myocardial infarction.

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