Secondary R Waves in Right Precordial Leads in Normal Persons and in Patients with Cardiac Disease

By Fernando A. Tapia, M.D., and William L. Proudfoot, M.D.

Secondary R waves in leads V1 or V3R are definitely abnormal if the primary R wave is high for the age group even though the R' is small; if the secondary R wave is more than 6 mm.; or if the R'/S ratio exceeds 1. Other patterns may be found both in patients with heart disease and in normal subjects. In the abnormal cases, the QRS changes persist in leads taken below but in line with V1 and V3R; but in normal cases, the secondary R waves usually disappear in the lower leads.

Considerable disagreement exists concerning the clinical significance of secondary R waves in right precordial leads in instances in which the duration of the QRS complex is normal or slightly prolonged. Their presence is frequently interpreted as indicative of incomplete right bundle-branch block and often is considered a manifestation of cardiac abnormality. Several investigators1-5 have reported the high incidence of secondary R waves in 1 or more of these leads, including additional higher or more lateral chest leads in normal persons.

No uniform criteria are available to differentiate the electrocardiograms of patients who appear to have cardiac disease from those of normal persons having secondary R waves in right precordial leads. In an attempt to find reliable differential criteria, the electrocardiograms of patients with proved cardiac disease and those of normal persons showing secondary R waves in any lead from the right side of the precordium were compared. Additional lower and higher chest leads were taken to observe any differential variation in both groups.

A distinct pattern was found in the majority of patients with cardiac disease, especially in those with congenital cardiac disease. The findings in right lower chest leads seemed to be of sufficient clinical value to clarify the significance of controversial patterns.

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Material and Methods

The routine 12-lead electrocardiogram and additional right chest leads were studied in 63 persons having secondary R waves in 1 or all of the right precordial leads, with "incomplete right bundle-branch block" pattern. Twenty-five of the patients had congenital heart disease producing right ventricular overload; 13 had acquired heart disease; and 25 were normal from the cardiovascular standpoint.

The diagnosis in the 25 patients with congenital heart disease was confirmed by cardiac catheterization and selective cineangiocardiographic studies.° The patients (12 female and 13 male) ranged in age from 6 months to 36 years. Ten had interstitial septal defect, 4 had interventricular septal defect, 4 had pulmonic stenosis, 2 had anomalous pulmonary venous return, 2 had a combination of interstitial septal defect and anomalous pulmonary venous return, and 1 each had tetralogy of Fallot, aortic-pulmonary window, and Eisenmenger's complex. The diagnosis was confirmed by cardiac surgery in 20 of these 25 patients.

The 13 patients (5 female and 8 male) having acquired heart disease ranged in age from 19 months to 61 years. The disease was caused by rheumatic valvular disease (3 patients), arteriosclerotic heart disease (4 patients), pulmonary heart disease (2 patients), lupus erythematosus and collagen diseases (2 patients), myocarditis (1 patient), and carcinoid syndrome (1 patient). Fewer patients were selected for this intermediate control group than for each of the other groups because of difficulty in determining the significance of the organic lesion.

The 25 persons with normal hearts ranged in age from 2½ to 60 years and included 9 females and 16 males. In each there was no evidence of heart disease by history, physical examination, or radiologic cardiac evaluation, and there were no

*Studies carried out by F. Mason Sones, Jr., M.D.
other electrocardiographic abnormalities. The secondary R waves in the right preordial leads were detected during routine electrocardiographic evaluation for periodic electrocardiographic evaluation, preoperative clearance, or diagnostic studies. This group included 3 patients with initial questionable evidence of congenital heart disease, because of the presence of an R' in right preordial leads and a pulmonic systolic murmur, but without symptoms or cardiac enlargement by radiologic or fluoroscopic examination. In all 3 (1 had pectus excavatum) cardiac catheterization and selective cineangiographic studies revealed normal intracardiac hemodynamics.

In all 63 patients, routine 12-lead electrocardiograms were taken. Nine chest leads were recorded with a paper speed of 75 mm. per second and a normal standardization: V₃R, V₁, and V₂ at the usual levels and leads V₃R, V₁, and V₂ at lower levels, 1 and 2 interspaces below the usual levels. In 8 patients, additional higher right chest leads were recorded. All records were made with a Sanborn Twin-Beam Cardiometry Model 62. In each of the leads the following observations were recorded: incomplete right bundle-branch block pattern, QRS duration, time of inscription of R and R', amplitude of R and R', depth of S waves, direction of T waves, presence of slurring or notching, or both, of the QRS complexes, and the R'/S ratio in V₁ and V₃R. Concomitant presence or absence of a relatively broad S in lead I, V₅, V₆, and of a secondary R wave in lead aVR of the routine electrocardiogram were also noted and the R'/S or R/Q ratio in aVR was recorded. Patients with complete right bundle-branch block (QRS more than 0.12 second) were not included in this study.

Findings
From the study of the routine electrocardiogram in the 3 different groups, several findings appeared to be helpful in differentiating "pathologic" from "physiologic" secondary R waves in the right preordial leads. However, no specific criterion was consistently found. Definite identification of abnormality was possible in 20 (80 per cent) of the 25 patients with congenital heart disease and in 6 (46 per cent) of the 13 patients with acquired heart disease.

The criterion for diagnosis of incomplete right bundle-branch block (the presence of a late secondary R in right preordial leads with a QRS duration less than 0.12 second) was present in all the normal subjects. Stricter criteria for such a diagnosis (QRS interval 0.08 or 0.10 to less than 0.12 second8-11) were satisfied in a large percentage of the normal subjects and in a frequency equal to that of patients with definite involvement of the right heart. Secondary R waves considered to be without diagnostic significance for right ventricular hypertrophy9, 10, 12-14 were found in many patients with heart disease and definite right ventricular hypertrophy.

The time of the inscription of the initial R in the right preordial leads was less than 0.03 second in all patients, and the R' (0.03 to 0.035 second being considered as maximum normal values15-18) was delayed in all patients. Slurring or notching of the QRS complex and the presence of a relatively broad S in leads I, V₅, and V₆ were found to be similar in incidence in the normal and abnormal groups. Most of the patients in both groups had inverted T waves in leads V₁ and V₃R.

Presence of R' in aVR and R'/S or R/Q Ratio in aVR
A secondary R wave in a Vᵣ, considered to be "a highly reliable indication of so-called incomplete right bundle-branch block"15 was present in only 12 of 25 patients with congenital heart disease, in 9 of the 13 patients with acquired heart disease, and in 14 of the 25 normal subjects showing secondary R waves in right chest leads.

An "abnormal" R/Q or R'/S ratio in aVR (more than 1.0)12, 19 was present in only 3 patients of the congenital group, in only 2 patients with acquired heart disease, and in 1 normal subject.

Amplitude of R and R' and Comparative Values
Most of the patients in the 3 groups had small initial R waves exceeded by the secondary R waves (fig. 1). The highest primary R in normal subjects was 8 mm. in V₁ in a 2½-year-old child. Primary R waves larger than 8 mm. in V₃R or V₁ were present only in patients with heart disease. The highest R' recorded was 25 mm. in a patient having congenital heart disease. The amplitude of R' in the patients having no heart disease did not exceed 6 mm. except in 1 case in which the abnormal pattern persisted in right lower
Fig. 1
A (normal) and B (congenital heart disease) showing similar patterns. C, Small R' in congenital heart disease. R'/S ratio greater than 1.0.

Heart failure, related to the condition, was found and the findings were similar to those presented for the congenital group.

Depth of S and the R'/S Ratio in V1 and V3R
A relatively small S with R'/S ratio more than 1.0 in V1 or V3R was found in most (19 of 25) of the patients with congenital heart disease, less frequently (6 of 13) in patients with acquired heart disease, and in only a few (4 of 25) of the patients with clinically normal hearts. In the latter group, of the 4 patients with R'/S ratio more than 1.0 in V1 or V3R, 3 had persistent R' and 1 showed a notched tall R on the right lower chest leads.

The R' in Right Lower Chest Leads
When additional right lower chest leads were taken (table 1), rather striking and consistent differences in the secondary R waves were found between the patients with definite heart disease and those with clinically normal hearts.

In all patients with congenital heart disease, the secondary R wave persisted or changed to an abnormal QRS shape. In the majority, R' persisted unchanged; and in a few, the RSR' complex changed to a tall notched R with incomplete fusion of R and R'. In all patients of this group, the S wave of the 'M'-shaped complex decreased progressively in depth with maintenance of a true R' in the right lower chest leads (fig. 2). In 3 patients, however, the S wave became a central noting of the R wave above the baseline with disappearance of the true R' but with persistent 'M'-shaped contour. One of these patients had an interatrial septal defect without pulmonary hypertension and the other 2 had pulmonic stenosis with a QRS pattern in lower leads resembling more closely that of the 'systolic overload' frequently seen with the latter defect.

Similarly, most of the patients with acquired heart disease showed persistent R' in right lower chest leads. In 1 patient with systemic lupus erythematosus the R' changed to a notched R. In 2 patients with asymptomatic rheumatic heart disease and mild mitral insufficiency, the R' disappeared in the lower leads; neither of these patients had clinical or radiologic evidence of cardiac enlargement or right ventricular hypertrophy.

In contrast, the R' disappeared in most of the subjects with clinically normal hearts when additional leads were taken 1 and 2 intercostal spaces below the routine levels. The

<table>
<thead>
<tr>
<th>R' in lower chest leads</th>
<th>Congenital heart disease</th>
<th>No heart disease</th>
<th>Acquired heart disease</th>
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<tr>
<td>Persistent</td>
<td>22 (88%)</td>
<td>4* (16%)</td>
<td>10 (77%)</td>
</tr>
<tr>
<td>Change to notched R</td>
<td>3 (12%)</td>
<td>1* (4%)</td>
<td>1 (8%)</td>
</tr>
<tr>
<td>Disappear in all leads</td>
<td>20 (80%)</td>
<td>2† (15%)</td>
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</tbody>
</table>

*R'/S ratio in V1 > 1.0 except in one of normal cases with persistent R'.
†No right ventricular hypertrophy.

Table 1
R' in Right Lower Chest Leads in Sixty-three Patients

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R' disappeared by gradual reduction in its amplitude and without significant changes of the S wave (figs. 3 and 4). In about half of this group, the R' disappeared in all 3 leads when additional leads were taken 1 interspace below usual levels; and in the other half, the R' disappeared when additional lower chest leads were taken 2 interspaces below routine levels. However, 4 patients showed persistent R' in lower leads, even 2 interspaces below usual levels, 3 of them having small S waves and abnormal R'/S ratios in the routine V1 or V3R lead. Two of these 4 patients, aged 45 and 49 years, respectively, were seen for routine annual physical examination and were apparently in good health. The third patient, 49 years of age, had obesity and functional hypoglycemia, and the fourth aged 50 years was suffering from hyperthyroidism but without clinical evidence of cardiac involvement. A 27-year-old man with a diagnosis of left lower lobe bronchiectasis showed a tall notched R in right lower chest leads and he also had an R'/S ratio over 1.0 in the usual V1 and V3R leads. In all the 3 patients with initial questionable evidence of congenital heart disease in whom the diagnosis was disproved by subsequent studies, the secondary R wave disappeared in lower leads.

Correlation between RSR Patterns, Heart Disease, and Persistent R' in Lower Chest Leads

The most common pattern found in normal persons was a small R, a relatively deep S wave with R'/S ratio less than 1.0, and a small

**Figure 2**

Persistent R' in lower leads (interatrial septal defect). R' is 8 mm. and the R'/S ratio greater than 1.0 in V1.

**Figure 3**

Disappearance of R' in lower lead and R' absent in V3R (normal).

R' (6 mm. or less)—rSR' pattern—and in all, with one exception, the R' disappeared in lower chest leads (tables 2 and 3). Patients with heart disease having a similar pattern showed persistent R' in right lower chest leads.

The majority of patients with heart disease, especially those of the congenital group, had a small S wave with an R'/S ratio greater than 1.0, or an R' larger than 6 mm., or a primary R larger than 8 mm. In all these cases the abnormal pattern persisted in lower leads as it was observed in the few patients with normal hearts having similar patterns.

**R' in Upper Leads**

In all patients in whom additional right upper chest leads were taken, the R' showed an increase in amplitude, regardless of the presence or absence of heart disease. In many normal subjects, secondary R waves appeared in higher leads, not present in the correlative lead at routine level.

**Comparative Amplitude of R' in V3R and V2**

When a secondary R wave was present in all 3 usual V3R, V1, and V2 leads, it tended to be higher in V3R in the patients with heart disease (11 of 14 patients) than in those without heart disease and higher in V2 in those without (7 out of 10) than in those with heart disease. The secondary R wave was present in V3R in all but 1 of the patients with heart disease; the exception was a patient with mild
mitral insufficiency having no evidence of right ventricular enlargement and in whom the R' disappeared in the right lower chest leads. In 3 normal subjects, the R' was absent in V_{3R} but present in V_1 and V_2; and in all, the R' disappeared when right lower chest leads were taken (fig. 3).

The R' was present in V_{3R} alone in 3 patients with congenital heart disease, in 2 with acquired heart disease, and in 1 normal subject. In all these cases, it persisted when right lower chest leads were taken.

Discussion

The presence of a secondary R wave in right precordial leads per se does not mean electrocardiographic or clinical abnormality. It may be present in patients with definite heart disease and in those with clinically normal hearts. In this study, certain associated electrocardiographic changes were found in most of the patients of the abnormal group (especially in those with congenital heart disease) but in few of the patients in the normal group. When routine chest leads were used, a definite diagnosis of abnormal secondary R wave in right precordial leads was established in 20 (80 per cent) of the 25 patients with congenital heart disease and in 6 (46 per cent) of the 13 patients with acquired heart disease. However, a significant number of the patients with heart disease showed secondary R waves in routine right chest leads that could not be differentiated from those found in the normal subjects, and accepted diagnostic criteria of "incompletely right bundle-branch block" were present in the majority of subjects with clinically normal hearts.

From the analysis of our findings, the most reliable diagnostic signs of abnormal or pathologic RSR pattern in right precordial leads were (1) a primary R higher than 8 mm. in V_{3R} and V_1, regardless of the amplitude of the secondary R; (2) a secondary R wave higher than 6 mm., regardless of the amplitude of the primary R; and (3) a small S with an R'/S ratio more than 1.0 in V_1 or V_{3R} or both (tables 2 and 3). When R' was higher than R, it was not significant for abnormality unless the R' exceeded 6 mm. in height or the R'/S ratio exceeded 1.0. It seems that a large R' in right precordial leads (apparently greater than 6 mm.) whether it is larger or smaller than the primary R, is definitely abnormal. On the other hand, an R' less than 6 mm., whether it is larger or smaller than R, is not necessarily abnormal. However, when large primary R waves exceed normal-range values for the involved age group,15, 16, 18, 20 and are consistent with right ventricular hypertrophy,8, 21-25 the presence of an R' (even if less than 6 mm.), is confirmatory of right ventricular hypertrophy or abnormal electrocardiogram. Small secondary R waves (less than 6 mm.) but larger than S (R'/S ratio more than 1.0), were also considered abnormal (fig. 1C).

In the 3 groups, all cases with an R'/S ratio in V_1 or V_{3R} of more than 1.0 had a persistent R' in lower leads (table 3). The fact that an R'/S ratio less than 1.0 in these leads was present in the electrocardiograms of all normal subjects in which the R' disappeared in lower chest leads (with one exception) and the observation that a ratio more than 1.0 was present in 19 (76 per cent) of the 25 patients with congenital heart disease, make its presence a reliable criterion of abnormality but its absence does not exclude organic changes.

The presence of a secondary R wave in right chest leads in the absence of the 3 previously mentioned criteria may still represent an

Figure 4
Disappearance of R' (normal).
electrocardiographic abnormality and differentiation from those patterns seen in persons with normal hearts is more difficult. It was evident that a relatively deep S wave (R'/S ratio less than 1.0) and a small R' (6 mm. or less) were present in practically all the normal subjects in whom R' disappeared in lower chest leads (fig. 1A), but some patients with congenital and acquired heart disease had similar patterns (fig. 1B). Therefore, the differentiation of physiologic and abnormal secondary R waves cannot be established on the basis of this pattern alone (table 2). However, there were several associated findings suggesting that the R' represented a normal electrocardiographic variation and not a manifestation of heart disease. The above pattern in V1 or V2, or both, but not in V3R, was seen only in subjects with normal hearts (fig. 3) and in one patient with rheumatic heart disease without right ventricular enlargement in whom the R' disappeared in lower leads. The same pattern with R' in V2 larger by more than 1 mm. than the R' in V3R, was also present in several normal subjects but in none of the patients with heart disease.

The presence of a secondary R wave with an R'/S ratio more than 1.0 in V3R alone seems abnormal. In all cases in which R' was present in V3R alone, it persisted in lower leads, suggesting definite abnormality.

In doubtful cases, the use of additional right lower chest leads may be helpful in differential diagnosis; it permitted the establishment of a differential criterion in those cases of indeterminate pattern, common to patients with and without heart disease. There was a consistent correlation between abnormal RSR patterns, heart disease, and persistent secondary R waves in right lower chest leads (table 3). The R' persisted in lower leads in all patients with RSR patterns interpreted as abnormal (figs. 1C and 2) and in all patients with heart disease showing indeterminate or not definitely abnormal RSR patterns. The possibility of an organic conduction defect without clinically detectable heart disease cannot be excluded in those few patients whose R'/S ratios were abnormal in V1 and V3R and in whom the R' persisted in lower leads. The secondary R wave disappeared in all but one of the normal subjects showing indeterminate RSR pattern in conventional right precordial leads (figs. 2 and 3).

A persistent R' in right lower chest leads probably indicates an organic change, either a disturbance in the conduction system or right ventricular hypertrophy. Whatever the position of the exploratory electrode, the abnormal conduction pathway or delayed activation of hypertrophied muscle of the basal zones should be recorded. On the other hand, secondary R waves in right precordial leads that disappear when right lower chest leads are taken, as found in all young healthy subjects in our study and in most of the adult group with clinically normal hearts, may be due to a variable distribution of the areas on

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**Table 2**

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<thead>
<tr>
<th>RSR Patterns in Sixty-three Patients</th>
<th>Number of Patients Having</th>
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<tr>
<td></td>
<td>Congenital heart disease</td>
</tr>
<tr>
<td>rSr'</td>
<td>5 (20%)</td>
</tr>
<tr>
<td>(Relatively deep S with R'/S &lt; 1.0 + R' &lt; 6 mm.)</td>
<td></td>
</tr>
<tr>
<td>rsR - RsR' - RsR'</td>
<td>15 (60%)</td>
</tr>
<tr>
<td>(Small s + R'/S &gt; 1.0 + R' &gt; 6 mm. or R &gt; 8 mm.)</td>
<td></td>
</tr>
<tr>
<td>rsr</td>
<td>5 (20%)</td>
</tr>
<tr>
<td>(Small s + R'/S &gt; 1.0 + R' &lt; 6 mm.)</td>
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*Persistent abnormal pattern in lower leads.
the surface of the chest to which the normal potential variations of the ventricular surfaces are transmitted. Sequence of mural and septal activation are detected, therefore, in different ways according to the changes in the position of the electrode and to variations in the position of the heart rather than to changes in intraventricular conduction due to organic defect.

The fact that secondary R waves were progressively larger or first appeared when higher chest leads were taken in our patients—a frequent finding by Overy and Johnston and by Said and Bryant in young healthy adults—corroborates the above impression. This observation supports the view that secondary R waves in normal subjects may be due to physiologic late activation of muscle near the outflow tract of the right ventricle, more specifically of the crista supraventricularis or of the basal zones represented by the direction of the terminal portion of the QRS vector and may be best detected with high right chest leads and are not recorded when the electrode is displaced to right lower chest positions.

The use of additional right lower chest leads—specifically V₃₉ or V₁ recorded 2 interspaces below usual levels—in doubtful cases of so-called incomplete right bundle-branch block, seems more reliable than the use of lateral right chest leads to differentiate the pathologic from the physiologic presence of secondary R waves in the usual right precordial leads. Overy and Johnston have suggested that in subjects without heart disease, secondary R waves may occur in high right chest leads but are absent in lateral right chest leads; however, Said and Bryant found secondary R waves in right lateral chest leads at the level of the fourth and fifth intercostal spaces in 73 per cent of 100 young healthy adults.

Our findings do not entirely agree with those of Camerini and Davies, who found a higher incidence of R' in leads obtained from one intercostal space below V₃₉, V₄₉, and V₅₉ than we found; but they reported a total incidence of R' in any additional lead and no correlative changes 1 and 2 interspaces below usual levels.

An apparently contradictory disappearance of secondary R waves in right lower leads was seen in 2 patients with rheumatic heart disease. Each had asymptomatic mild mitral insufficiency without clinical or radiologic evidence of right ventricular enlargement, and an R'/S ratio in lead V₁ of less than 1.0; one showed an R' only in V₁ and V₂ and none in V₃₉. The R' was probably a physiologic variation and not the result of right bundle-branch block or right ventricular hypertrophy, this interpretation being more in accordance with the entire clinical picture. These findings suggest the possible value of additional lower chest leads in interpreting the presence of intermediate secondary R waves in right precordial leads in patients with rheumatic heart disease and mitral involvement, when there are apparently contradictory findings in the electrocardiogram and the roentgenogram regarding right ventricular enlargement.

**Summary**

The routine 12-lead electrocardiogram and additional right chest leads were studied in 63 persons having secondary R waves in right precordial leads and QRS duration less than 0.12 second. This group included 25 patients with congenital heart disease, 13 with acquired heart disease, and 25 with normal hearts.
SECONDARY R WAVES IN ELECTROCARDIOGRAM

A distinct pattern of abnormality was found in the majority of patients with heart disease, especially in those with congenital cardiac lesions. The accepted criteria for the diagnosis of incomplete right bundle-branch block were found in the majority of the normal subjects, and secondary R waves considered without apparent pathologic significance were found in many patients with heart disease and definite right ventricular hypertrophy.

Criteria based upon the following findings were of no value in differentiating secondary R waves in right precordial leads associated with heart disease from those not associated with heart disease: the QRS duration; time of inscription of the intrinsicoid deflection; greater amplitude of R’ compared with R without regard to the absolute height of R’; presence of slurring or notching of the QRS complexes; presence of a relatively broad S in leads I, V5 and V6; presence of secondary R wave or “abnormal” R’/S or R/Q ratio in aVR; and the T-wave direction.

Reliable signs of an abnormal RSR pattern in right precordial leads were (1) A primary R wave higher than accepted normal range values for the involved age group, regardless of the amplitude of the secondary R. In our series a primary R higher than 8 mm. in V3R and V1 was always abnormal. (2) A secondary R wave higher than 6 mm., regardless of the amplitude of the primary R. (3) An R’/S ratio more than 1.0 in any lead from the right side of the precordium.

On the basis of these criteria, a definite diagnosis of abnormality was possible in 20 (80 per cent) of the 25 patients with congenital heart disease and in 6 (46 per cent) of the 13 with acquired heart disease.

A pattern of small R, relatively deep S (R’/S ratio less than 1.0), and a small R’ (6 mm. or less) (rSr’ pattern) was present in practically all the normal subjects but also in a significant number of patients with heart disease, 5 (20 per cent) of the 25 with congenital heart disease and 7 (54 per cent) of the 13 with acquired heart disease, and a definite electrocardiographic diagnosis of abnormality was not possible in these patients with the usual precordial leads.

The secondary R wave disappeared when additional right lower chest leads were taken in all the normal subjects with an rSr’ pattern (relatively deep S with R’ less than 6 mm.) in right precordial leads; it persisted unchanged or occasionally changed to an abnormally large notched R in all the patients with heart disease. In the latter group these changes were independent of the amplitude of the R’ or of the RSR pattern.

The presence of an rSr’ pattern in V1 or V2, or both, but its absence in V3R, or an R’ in V2 larger by more than 1.0 mm. than the R’ in V3R, suggested that the R’ represented a normal electrocardiogram variation and was not a manifestation of heart disease.

The use of additional right lower chest leads—specifically V3R or V1 2 interspaces below the usual levels—may help in the differential diagnosis of doubtful cases of so-called incomplete right bundle-branch block. A persistent R’ in lower leads probably indicates abnormal right ventricular conduction or right ventricular hypertrophy. Disappearance of secondary R waves in lower right chest leads and their increase in higher leads, seen in normal subjects, support the impression that these secondary waves may be due to physiologic late activation of muscle near the outflow tract of the right ventricle or more specifically of the crista supraventricularis. The depolarization of these basal zones is represented by the direction of the terminal portion of the QRS vector.

Sumario in Interlingua

Le electrocardiogramma routinari a 12 derivaciones e in plus derivaciones dextero-thoracic esseva studiate in 63 personas qui haberba secundari undas R in der-ivaciones dextero-precordial e un duration QRS de minus que 0,12 secondas. Le grupo consisteva de 25 patientes con congenite morbo cardiae, 13 con ac-quirite morbo cardiae, e 25 con cordes normal.

Un distinete configuration de anormalitades esseva trovate in le majoritate del patientes con morbo cardiae, specialmente in le patientes con congenite le-isiones del corde. Le acceptate criterios pro le diagnose de incomplete bloco de branca dextere esseva trovate in le majoritate del subjectos normal, e secundari
undas R—considerate como disprovistoe de apparente signification pathologie—esueva presente in multe pa-
tientes con morbo cardiae e definite hypertrophia
dextero-ventricular.

Criterios basate super le sequeste lista de datos
esueva sin valor in le differentiation inter secundari
undas R in derivaciones dextero-precordial que es-
seva associate con morbo cardiae e illos que non esueva
associate con morbo cardiae: Le duration de QRS;
le tempore del insersion de deflexion intrinsicoide;
aumento del amplidute de R' in comparation con illo
de R sin referentia al altor absolute de R'; presentia
de dentation o de continuitate indistinente in le com-
plexos QRS; presentia de un relatively large S
in le derivaciones I, V6 e V4; presentia de secundari
unda R o de un proportion "anormal" R'/S o R/Q
in aV2; e le direction del unda T.

Signos indicante fidelemente un anormalitate del
patron RSR in derivaciones dextero-precordial esueva:
(1) Un primari unda R de altor in excesso del area
de valores considerate como normal pro le gruppo de
etate in question, sin reguardo al amplidute del unda
R secundari. In nostre serie un R primari de plus que
8 mm de altor in Vm e V1 esueva semper anormal. (2)
Un secundari unda R plus alte que 6 mm, sin reguardo
al amplidute de R primari. (3) Un proportion R'/S
de plus que 1,0 in non importa qual derivation al
dextera del precordia.

Suple le base de iste criterios, un definite diagno-
se de anormalitate esueva possibile in 20 del 25 pa-
tientes con conogenite morbo cardiae (80 pro cento) e
in 6 del 13 pacientes con acquirite morbo cardiae (46 pro
cento).

Un combination de miere R e relativamente pro-
funde S, i.e. un proportion R'/S de minus que 1,0, e
un miere R' de 6 mm o minus (patrono RSR') esueva
presente in practicamente omne le subjectos normal
sed etiam in un numero significativo de pacientes con
morbo cardiae. Isto valesa pro 5 del 25 patients con
congenite morbo cardiae (20 pro cento) e pro 7 del
13 con acquirite morbo cardiae (54 pro cento), e un
definite diagnose electrocardiographie de l'anormalitate
non esueva possibile in iste patients per medio del
usual derivations precordial.

Le secundari unda R dispareva quando derivationes
additional ab le thorace dextero-inferior esueva ob-
tenite in le casos del patients qui habeva un pa-
trono RSR' (S relativamente profonde con R' de minus
que 6 mm) in le derivations dextero-precordial; illo
persisteva o, a vices, se alterava in un anormamente
grande R a dentation in omne le patients con morbo
cardiae. In iste ultime gruppo, le mentionate altera-
tiones esueva independente del amplidute del R' o
del patrono RSR.

Le presentia de un patrono RSR' in V1 o V6 o in
ambes insimul con su absentia in Vm o le presentia,
in V6 de un R' que exceede le R' de Vm per plus que
1,0 mm de largor sugereve que le R' representava
un normal variation electrocardiographie e non esueva
un manifestation de morbo cardiae.

Le uso de derivationes additional ab le thorace dex-
tero-inferior—specificamente le uso de Vm o V1 a 2
interspation infra le nivellos usual—pote esser de
ajuda in le diagnose differential de casos dubitose
del si-appelate incomplete bloco del branca dextere.
Un persistente R' in derivationes inferior indica
probablemente un anormalitate del conduction
dextero-ventricular o un hypertrophia dextero-ventricular.
Le disparition de secundari undas R in derivationes
ab le thorace dextero-inferior e le augmento de tal
undas in derivationes superior, vidite in subjectos
normal, supporta le impression que iste undas secun-
dari es le effecto de un tardive activation physio-
logie del museulo in le vicinitate del tracto de efflussi
del ventriculo dextere o plus specificemente del
crista supraventricular. Le dispolarization de iste
zonas basal es representate per le direction del portion
terminal del vector QRS.

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Sheps
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