SECONDARY R WAVES IN RIGHT CHEST LEADS

BY

FULVIO CAMERINI AND L. G. DAVIES

From the Postgraduate Medical School of London

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The use of the electrocardiogram as an aid in the diagnosis of right ventricular hypertrophy is a comparatively recent advance. It has been suggested that an electrode placed in a similar position to V4 but to the right of the sternum (V4R) would be of additional value in that it would record right ventricular patterns more accurately than the present chest leads. We have been recording this lead (V4R) routinely for the last year and have frequently had difficulty in assessing the significance of the patterns found, mainly because complexes with secondary R waves often occurred in both normal and abnormal tracings. There is no agreed terminology for describing these patterns but the different terms used and the theories underlying them have been comprehensively reviewed by Katz *et al.* (1950). The complexes have been termed RSR¹ or denoted as showing the presence of an "embryonic r wave" or "incomplete right bundle branch block." A more recent suggestion is that the pattern occurs as the result of "defective intraventricular conduction" which may be "focal" or more widespread (Segers, 1949). The confusion in nomenclature is well illustrated by the term "physiological incomplete right bundle branch block" which aptly reflects the uncertainty as to the significance of this type of pattern.

It was decided to record V4R in a series of normal subjects in order to determine the variations that might be found. In addition, it was felt that a study of records taken from other positions over the right chest might help determine the significance of complexes with secondary R waves.

Material and Methods. Fifty subjects (30 men and 20 women) were studied, their ages ranged from 10 to 75 years. None had symptoms of cardiovascular or pulmonary disease, all had blood pressures within the normal range and their hearts were normal on clinical examination.

All the records were taken on an Elmquist three-channel recorder.

The leads taken from the right of the chest were initially V5R, V4R, V3R, and V1. V5R, V4R, and V3R were recorded with electrodes placed in similar positions to V5, V4, and V3 but to the right of the sternum. At first these leads were recorded in different phases of respiration, but then it was realized that complexes with secondary R waves were more often found when further leads were taken from the intercostal spaces above and below V5R, V4R, V3R, and V1 with the patient breathing normally. The standard and unipolar limb leads and V3, V5, and V7 were also recorded in each patient.

RESULTS

In V4R, the S wave was always greater than either R deflection; in over half the subjects (52%) the pattern was RSR¹ or RSR¹S¹ and in the remainder was rS. We would stress the necessity of placing the electrode in the correct position, because occasionally we found that small changes in electrode position caused important changes in the configuration of the QRS complex.

The incidence of patterns with secondary R waves in the additional leads was also high (Table I) and in 42 of our 50 subjects (84%) RSR¹ or RSR¹S¹ complexes were found in one or more of the leads taken from the right chest. The distribution is shown as a percentage of the total number of recordings in each position as we recorded fewer leads at the beginning of the series. It will be seen that complexes with secondary R waves are most commonly obtained from the intercostal spaces below V5R, V4R and V3R.

TABLE I
THE INCIDENCE OF PATTERNS WITH SECONDARY R WAVES IN THE DIFFERENT ELECTRODE POSITIONS

Intercostal space above 36%	38%	28%	35%	—
V5R 45%	V4R 52%	V3R 34%	V1 26%	V2 13%
Intercostal space below 55%	61%	61%	35%	—

FIG. 1.—Different types of RSR⁸ and RSR¹S¹ patterns in normal subjects.

Fig. 1 illustrates the different types of RSR¹ or RSR¹S¹ patterns recorded, and in particular shows the wide variation in the position and size of the R¹ wave which was encountered. That these R¹ waves have a common origin was evident from a study of leads recorded simultaneously (Fig. 2) and the variation in pattern is seen to depend almost entirely on the position of the electrode. The “embryonic r wave” in V4R (Fig. 2) is clearly identical with the large secondary R wave recorded from the space below V4R—a complex that might be described as “incomplete right bundle branch block.”

All transitions were found between subjects who showed RSR¹ patterns in only one or two leads (Fig. 3) and those in whom it was present in nearly all the right chest leads (Fig. 2).

The duration of QRS in these 42 subjects varied between 0.05 and 0.09 sec. In 22 the duration was 0.08 sec., in 11 it was 0.07 sec., in 5 it was 0.09 sec., in 3 it was 0.06 sec., and in one it was 0.05 sec.

The time of the intrinsicoid deflection in complexes with RSR¹ patterns varied between 0.02 and 0.07 sec. In 14 it was 0.05 sec., in 13 it was 0.06 sec.; in 7 it was 0.04 sec., in 5 it was 0.07 sec., in 2 it was 0.02 sec., and in one it was 0.03 sec.

Large S waves in standard lead I have been considered a feature of tracings showing incomplete or complete right bundle branch block. Prominent S waves were found in standard lead I in 3 of our subjects; in these 3, RSR¹ complexes were found widely in the right chest leads. In 4 others, however, who showed a similar distribution of RSR¹ complexes across the right chest the S waves in lead I were not remarkable.

Of the limb leads, however, the configuration of VR should be more helpful in reflecting patterns

FIG. 2.—Simultaneous records showing common origin of R' waves.

FIG. 3.— RSR' patterns in a small number of leads only.

from the right chest. Of the 42 subjects whose chest leads showed complexes with secondary R waves, an rSr¹ pattern in VR was found in 17 and in a further 17 a Qr pattern was present.

DISCUSSION

Irrespective of whether the QRS pattern in V4R was rS, RSR¹, or RSR¹S¹, we always found the S wave to be larger than either R deflection. This finding is useful clinically but we would stress again the importance of positioning the electrode correctly. For example, in Fig. 2 there is a considerable difference between the V4R complex and that recorded from the intercostal space below V4R.

A secondary R wave was present in V4R in over half our subjects and this agrees with the observations of Mounsey *et al.* (1952). The presence of RSR¹ complexes in this or other right chest leads in the majority of normal subjects has not been reported previously. The importance of this finding is firstly that RSR¹ patterns have also been reported in subjects who showed post-mortem evidence of right ventricular hypertrophy (Thomas, 1948; Myers *et al.*, 1948): so that when using the electrocardiogram to make this diagnosis in life, it is important to realize the full range of normal. Secondly, the stigma implied when the term "block" is used to describe complexes of this type makes a review of the terminology pertinent.

Studies made when the electrocardiogram was first introduced were in subjects with abnormal hearts or in animals where conduction was blocked by experimental means; recent work too, has largely concerned patients with clinical heart disease. On this basis, many notched complexes have been explained as being the result of a block or partial delay in conduction. Indeed, if we accept the definition of Barker and Valencia (1949) most of our subjects have incomplete right bundle branch block, a term bearing the implication of some pathological defect in conduction. The same criticism may be made of more recent nomenclature. Segers (1949) has suggested that the cause of patterns of this type is a "block" which is peripheral or "parietal" in that it affects the junction between Purkinje fibres and myocardium or the myocardium itself rather than the conducting system proper. When the complexes are localized (as in Fig. 3) the "block" is said to be "focal" and the defect limited to only a portion of the ventricular wall.

Other workers, however (Wilson *et al.*, 1947), have been doubtful whether there could be any conduction defect in the right bundle branch with a QRS duration of 0.08 sec. or less and a small R¹ wave. Our finding of RSR¹ patterns in most of our subjects confirms this suspicion. It is generally agreed that the initial R wave in V4R and VI is due to activation of the septum from left to right with the S wave resulting from activation of the left ventricle. The R¹ wave must be due to some later stage, activation of some other part of the normal myocardium; that is, there must be some physiological explanation not involving the concept of block and its pathological implications.

It is tempting to believe that the R¹ wave results from normal late activation of at least some part of the right ventricle. Certainly in patients with progressive pulmonary heart disease, an increase in the size of this R¹ wave from normal to abnormal heights has been recorded by Mounsey *et al.* (1952). As long ago as 1915, Lewis and Rothschild noted in dogs that the conus of the right ventricle and the postero-basal portion of the left ventricle are the last parts of the myocardium to be activated. These findings were confirmed by Wilson *et al.* (1930) in their observations on the exposed human heart.

Sokolow and Friedlander (1949) in their work on normal unipolar leads mention the possibility that the second R wave represented activation of the conus of the right ventricle. The only right chest lead they recorded, however, was V1, they found R¹ deflections in only 5 per cent of their subjects and so were uncertain of its real significance.

A study of cavity potentials should be helpful; these have now been recorded from the right atrium and ventricle of normal subjects and terminal R¹ waves have been found. Battro and Bidoggia (1947) concluded that they represented activation of the basal region of the left ventricle and the conus of the pulmonary artery. Sodi-Pallares *et al.* (1947) related them to the late activity of some parts of the right ventricular muscle. Another suggestion—made by Kossmann *et al.* (1950)

—was that the *crista supraventricularis* of the right ventricle may be the source of these secondary R waves.

Our leads showing RSR¹ complexes were recorded from the electrical field of the right ventricle and we feel that our results support the view that activation of the conus is responsible for secondary R waves.

It is not easy to see, however, why RSR¹ patterns are found widely in one subject and only locally in another. Kert and Hoobler (1949) have shown that there is a difference between the times of activation of the two ventricles in normal subjects and that this difference is greater in some subjects than in others. As the QRS complex recorded in any position represents the algebraic sum of the activation potentials of the two ventricles, this asynchronism may give prominence to the potential from the conus, or alternatively this potential may be damped out.

We would suggest, therefore, that QRS complexes of less than 0.10 sec. duration be termed simply RSR¹ or RSR¹S¹ when these patterns are found; they are a normal finding and these are logical descriptive terms.

SUMMARY

In 42 of 50 normal subjects, complexes with secondary R waves (RSR¹) were found in one or more of the many right chest leads recorded.

Many variations of this pattern appeared in different leads taken from the same patient.

The secondary R wave (R¹), may be due to activation of the conus of the right ventricle.

In V4R the S wave was always found to be larger than either R deflection.

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