THE SIGNIFICANCE OF DEEP S WAVES IN LEADS II AND III.

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The competence of the electrocardiogram in the diagnosis of cardiac pain is not in dispute, but doubt as to what constitutes a normal tracing still prevails, and the meaning of certain lesser changes has yet to be determined.

Erstwhile the T wave and its surrounds and the initial moiety of the QRS complex have received most attention whenever myocardial disease has been sought. The last portion of the QRS has not been subject to the same scrutiny, although as long ago as 1933 Wilson *et al.* drew attention to the presence of conspicuous S waves in the cardiogram of patients with cardiac infarction.

The purpose of this work has been to re-examine the contribution that changes in this terminal portion of the QRS complex can make to the diagnosis of heart disease.

The Investigation Described. The electrocardiograms of 655 cases were examined critically. These consisted of 200 healthy subjects, 161 with a raised blood pressure, 200 with cardiac pain, 53 with cardiomyopathy, 21 with emphysema, and 20 with obesity.

On finding an S wave in leads II and III, its magnitude in relation to the R wave was noted, as well as any irregularity in the complete tracing. The pattern eventually to be appraised was one showing *an S wave greater than the R wave in leads II and III in the absence of an S wave in lead I.*

Some writers apply a rigid definition to left axis deviation, wherein the mean QRS axis lies farther to the left than -30° and S exceeds R in lead II as well as in lead III. This last interpretation gives to the pattern here discussed the same meaning as left axis deviation, and directs attention to the wave form in lead II as of equal importance to that of the other two limb leads. It is necessary to emphasize at the outset that the electrocardiographic sign under review is not synonymous with changes accepted as denoting left axis deviation when customarily defined as showing dominant R waves in lead I and S waves in lead III (White, 1951; Friedberg, 1956; Wood, 1956).

Recognizing this want of unanimity in the interpretation of left axis deviation we have preferred, for the sake of clarity, to apply the designation S2S3 to the pattern under consideration (Fig. 1 and 2). Moreover, we have not accepted into the series as typifying this specific pattern those cases that showed an S wave in lead I in addition to leads II and III, for such a cardiogram may have a different meaning (Fig. 3).

FINDINGS

Healthy Series. If a pathological significance is to be given to a cardiographic finding, its incidence in healthy subjects must be low. For this reason the selection of cases considered to be healthy was a special care. Thus, they had to show no symptoms suggesting a cardio-arterial fault, nor clinical signs of this, nor disease in any other system that might affect the heart, nor a raised blood pressure. For the purpose of this investigation we rejected from the healthy group any subject whose systolic and diastolic blood pressure exceeded 160 and 95 mm. of Hg respectively. Significant peripheral arterial disease was also excluded and so was retinal arteriolar abnormality. Examination of the urine showed no abnormality. Radiological examination of the chest showed

FIG. 1.—The S wave is deep in leads II, III, and IIIr. From a man aged 53 years without chest pain. No significant changes appeared in an exercise cardiogram.

FIG. 2.—The S wave is deep in leads II, III, and IIIr, and natural Q waves are present in leads I, CR4, and CR7. From a man aged 75 years without chest pain. He was too frail for a recording of an exercise electrocardiogram.

FIG. 3.—The S wave is deep in leads II, III, and IIIr; it is also deep in I as part of the right bundle-branch block pattern. From a patient with atrial septal defect of the *ostium primum* type (necropsy control).

no disease of either the heart or the lungs. Finally, the resting electrocardiogram had to be an unblemished tracing, apart from the change in the S wave that is the concern of this paper.

Two hundred healthy subjects were convened in accordance with these criteria, 110 being over the age of 40, and 90 under 40 years. The S2S3 pattern did not appear in the younger group, and only three times in the older group (Table I). An exercise cardiogram, recorded in 59 of the 200 cases, was positive in only one, a man of 48 years without chest pain but showing the S2S3 pattern. The exercise test was negative in the other two symptomless cases that showed this pattern.

It would seem, therefore, that the S2S3 pattern is a rare finding in young healthy subjects. In older and apparently healthy subjects, its low incidence of 3 among 110 subjects over the age of 40 years suggests for it a significance comparable with some other electrocardiographic signs that are known to occur with increasing frequency in old age (Lepeschkin, 1951).

TABLE I
AGE INCIDENCE OF THE S2S3 ELECTROCARDIOGRAPHIC PATTERN IN 200 HEALTHY SUBJECTS

Age in years	Number of cases	Number of cases showing S2S3 pattern
10 to 20	8	0
21 to 30	45	0
31 to 40	42	0
41 to 50	48	1
51 to 60	38	1
61 to 70	16	0
71 to 80	3	1

Obesity. The cardiographic sign was sought in 20 very obese patients whose average age was 53 years. Some of them weighed over 17 stone (108 kg.). Only one among these 20 showed the S2S3 pattern. This patient complained of pain in the upper chest spreading into the neck and the left arm, but unrelated to exertion.

Although Ashman (1946) stated that strong counter-clockwise rotation of a horizontal heart can give rise to an S2S3 configuration, Schlomka (1948) found that obesity of itself does not cause sufficient axis deviation to the left to produce this cardiographic change. Moreover, it is likely that pregnancy with its considerable elevation of the diaphragm does not cause the appearance of this pattern.

Hypertension. We assembled 50 patients with systemic hypertension. In all these the blood pressure was significantly raised and the evidence of cardio-arterial derangement consisted of whipcord brachial arteries, narrowed retinal arterioles, variable enlargement of the left ventricle which was often very great, and always left ventricular preponderance in the electrocardiogram.

Only 8 of the 50 patients showed the S2S3 pattern. In six of these, cardiac pain was present and the cardiogram provided evidence of cardiac infarction in addition to left ventricular preponderance (Fig. 4). One of the remaining two gave no history of cardiac pain, but the cardiogram showed signs of cardiac infarction: in the other both history and cardiogram failed to confirm the presence of infarction.

The relation between the S2S3 pattern and the level of the blood pressure was examined in these patients. The average blood pressure was 220/120 in the eight showing this pattern compared with 220/130 for the whole group. Again, among 22 patients with a diastolic pressure of 135 mm. or

FIG. 4.—The S wave is deep in leads II, III, and III R, and the T wave is inverted in leads I and CR4. From a woman aged 43 years with cardiac pain and a blood pressure of 240/120.

FIG. 5.—The S wave is deep in leads II, III, and III R. The T wave is inverted in the same leads and is low in I and in CR7. From a man, aged 44 years, with coarctation of the aorta, in whom patchy fibrosis of the left ventricle was present at necropsy. Death had taken place from rupture of the aorta.

FIG. 6.—The S wave is deep in leads II, III, and III R, and the T wave is inverted in I and CR7. From a man, aged 50 years, with cardiac pain.

over, in whom the average blood pressure was 240/140, only one showed an S2S3 pattern, while none among the six with the highest values (300/130, 260/160, 260/150, 250/150, 250/145 and 260/140) showed it.

We then sought the pattern in 85 subjects who, though the blood pressure was raised, were regarded as examples of systemic hypertonia rather than hypertension in that they showed no clinical, electrocardiographic, or radiological signs characteristic of the condition (Evans, 1957). Seven of these, whose average blood pressure was 190/115 compared with 170/105 for the whole group, showed the S2S3 pattern. As many as six of the seven patients had suffered cardiac infarction, while in the remaining one an artificial pneumothorax, resulting in great mediastinal distortion, had been carried out in the treatment of pulmonary tuberculosis.

Among 26 patients with *coarctation hypertension* whose average age was 27 years, two showed the S2S3 pattern. The significance of this finding is not properly understood. In the only case with this pattern examined at necropsy (Fig. 5), isolated streaks of fibrosis were seen in the antero-lateral aspect of the left ventricle.

The findings in these 161 patients with a raised blood pressure allow us to deduce that the S2S3 pattern is not caused by either a raised blood pressure or the enlargement of the left ventricle that may accompany it. A significant finding was cardiographic evidence of cardiac infarction in 13 of the 17 who showed the pattern.

Cardiac Infarction. The S2S3 pattern was next sought in the electrocardiograms of 200 patients with cardiac infarction, in whom hypertension was absent. It was present in 32 or 16 per cent of them.

FIG. 7.—The S wave is deep in leads II, III, and III R, and the T wave is diphasic in I, CR4, and CR7. From a man, aged 55 years, with cardiac pain.

FIG. 8.—The S wave is deep in leads II, III, and III R, and the T is inverted in I, CR4, and CR7. From a man, aged 47 years, with cardiac pain.

FIG. 9.—The S wave is deep in leads II, III, and III R, and the T wave is inverted in I and diphasic in CR7. The QRS complex is wide and there is no Q in I. From a man, aged 49 years, with an obscure cardiomyopathy and hear failure.

In that the site of the infarct can be told by reference to changes in a comprehensive cardiogram, the location of a lesion producing the distinctive S2S3 tracing was next determined by examining its incidence in the main anatomical kinds of cardiac infarction. Such incidence is shown in Table II. Reference to this table makes it clear that the pattern was associated almost exclusively with infarction situated in the antero-lateral aspect of the left ventricle (Fig. 6, 7, and 8).

TABLE II
INCIDENCE OF S2S3 PATTERN AMONG 200 PATIENTS WITH CARDIAC INFARCTION OF VARYING SIZE AND SITE

The infarct		Cardiographic pattern†	Number of cases	Number with S2S3 pattern	Incidence expressed as percentage
Kind	Site				
Salient*	Dominantly anterior	TI, T4	29	3	10
	Dominantly lateral	TI, T7	34	12	35
	Antero-lateral	TI, T4, T7	33	11	33
	Postero-inferior and lateral ..	TIII, T7	13	1	8
	Postero-inferior and medial ..	QTIII	22	0	0
Limited* or Restricted	Anterior	I and 4	5	0	0
	Lateral	I and 7	33	4	12
	Antero-lateral	I, 4 and 7	5	1	20
	Postero-inferior and lateral ..	III and 7	21	0	0
	Indeterminate	Varied	5	0	0
Total ..			200	32	16

* In *salient* infarction the T wave was frankly inverted, but in the *restricted* kind the indicated leads showed the lesser signs of cardiac infarction.

† Roman numerals in the second column refer to limb leads, and Arabic numerals to chest leads.

In that the cardiograms of right ventricular preponderance and of right bundle-branch block almost always exhibit an obvious S wave in lead I, they do not come within the scope of this discussion for they do not conform to the definition we have set out for the S2S3 pattern. The problem of the association of this pattern with tracings of the left bundle-branch block kind is to be discussed later when the mechanism underlying this cardiographic deformity is considered.

In the patients with limited or restricted cardiac infarction where the cardiographic changes were less obvious (Evans and McRae, 1952; Evans and Pillay, 1957), the distribution of those irregularities again placed the injury in the antero-lateral portion of the left ventricle. Thus, the addition of the S2S3 pattern in these patients gave further emphasis to the significance of such signs.

Cardiomyopathy. In 53 patients in whom there was affection of the left ventricle, usually with enlargement of varying degree, the common causes like hypertension, valvular disease, and coronary arterial disease had been excluded. As many as 23 or 43 per cent of them demonstrated the S2S3 configuration in the electrocardiogram.

Thus, it was present in 3 out of 9 cases of *familial cardiomegaly*, in 2 out of 14 cases of *Friedreich disease*, in 1 out of 3 with *cardiac amyloidosis*, and in single cases of *myotonia atrophica* and of *hæmochromatosis*. In the remaining 25 with *obscure cardiomyopathy*, the S2S3 pattern was present in as many as 16 patients, or 64 per cent. (Fig. 9, 10, and 11). Usually the deformity was associated with a wide QRS complex of from 0.10 to 0.16 sec. duration, but in five it was 0.09 sec. or less. A Q wave in lead I often accompanied the S2S3 pattern.

The investigation, therefore, emphasizes the high incidence of this pattern in patients with cardiomyopathy. It would have been even higher were it not for the exclusion of some patients in whom a deep S wave in leads II and III was associated with right bundle-branch block with its expected S wave in lead I; such exclusion is in keeping with our definition of this pattern.

Emphysema. Other authors (Grant, 1956; Duchosal and Jornod, 1958) have found the S2S3 pattern in patients with emphysema, and Grant considered that it resulted from an altered electrical

FIG. 10.—The S wave is deep in leads II, III, and III R, and the T is inverted in I and CR7. From a woman, aged 71 years, with an obscure cardiomyopathy. At necropsy the left ventricle showed hypertrophy with moderate replacement fibrosis and some small foci of lymphocytes.

FIG. 11.—The S wave is deep in leads II, III, and III R, and there is left bundle-branch block. From a man, aged 52 years, with myotonia atrophica.

FIG. 12.—A deep S wave in leads II, III, and III R was the only change in this tracing from a man, aged 46 years, with chest pain. Three months later, and three years after the onset of chest pain, the patient died during an attack. A cardiogram at the time showed characteristic changes of extensive cardiac infarction (necropsy control).

conductivity in the lungs, being present in 10 per cent of patients. We assembled 21 patients with emphysema for the present investigation, and in none did we find the S2S3 cardiographic pattern.

ASSOCIATED ELECTROCARDIOGRAPHIC SIGNS

The R/S Ratio in Leads II and III. In the S2S3 pattern that we describe, the S wave in lead III is deep and the R wave diminutive in comparison. Moreover, this disproportion is preserved in III R (lead III recorded during deep inspiration) and differs in this respect from that pertaining to left axis deviation as commonly defined, where a deep S in lead III becomes small or disappears in III R.

Our insistence that the S wave in lead II of the distinctive S2S3 pattern should be larger than the R wave in this lead is naturally arbitrary, fixing as it does the mean electrical axis at -30° or farther to the left. Inevitably, there will be instances where S2, though deep, is less than R2 and the result of heart disease, but we have elected in the meantime to adhere to our definition so that healthy subjects shall not by this token become handicapped by the stigma of a doubtful abnormality. Duchosal and Jornod (1958) have adopted a more rigid definition, namely that S2 should

be more than twice the amplitude of R₂, and not unnaturally they have found this pattern to be invariably pathological.

Associated Q Waves. The meaning of the association of a Q wave in lead I with the S₂S₃ pattern will be discussed later, but naturally a prominent Q may appear in CR₇ and/or in CR₄ when the pattern has been produced by an extensive myocardial lesion. Should the injury have a postero-inferior disposition with resulting Q waves in leads II and III, the addition of the S₂S₃ pattern is not then expected, and indeed rarely appears. When a deformity of the T wave accompanies the S₂S₃ pattern, it usually takes the form of inversion in leads I and CR₄ or CR₇, and the relative incidence of these deformities has been given in Table II.

S₂S₃ as a Lone Cardiographic Sign. There were three patients in whom the pattern had been present one time as a lone sign and where cardiac infarction developed later; in two the diagnosis was confirmed at necropsy and in the other by the association of characteristic cardiographic signs. In two of the three patients chest pain was a feature when S₂S₃ was a lone sign, but in the third pain was absent (Fig. 12 and 13).

Mention has already been made of three cases among the older members of the series of 200 healthy subjects who showed this pattern in their tracings. Subsequent to this investigation six subjects, four being young adults, have come to our notice where the S₂S₃ pattern was present in the absence of any evidence of heart disease.

The question is posed as to whether such instances have a pathological lesion of the myocardium, antero-laterally disposed, or are examples of a small conduction defect in the left ventricle as suggested by Manning (quoted by Grant, 1958). Thus, if the sign is discovered fortuitously in one without cardiac symptoms, it may be the outcome of congenital agenesis of the distal radicle of the left bundle, and as such is an immaterial finding. In an elderly patient without pain but showing this sign, the presence of a small infarct cannot be excluded.

THE MECHANISM UNDERLYING THE S₂S₃ PATTERN

Our clinico-cardiographic investigation has established that the pattern signifies some abnormality in the antero-lateral sector of the left ventricle. This is in line with the work of Grant (1956) who analysed the records of 672 cases in which electrocardiograms were taken within five weeks of death and were subsequently related to the necropsy findings. He found that 131 showed an S₂S₃ pattern almost identical with the one we describe, but he defined it as left axis deviation: more than one-third of them had frank cardiac infarction: two-thirds of 47 cases with cardiographic signs of antero-lateral infarction presented the S₂S₃ pattern. When Grant excluded all instances of cardiac infarction or lung disease, he did not find that left axis deviation among the remaining cases was related to the degree of hypertension or to the body build. Hurwitz *et al.* (1943) and Moll and Lutterotti (1951) also stressed the frequency with which a deep S wave in leads II and III appeared in patients with infarction of the anterior wall of the left ventricle: they mentioned that this was sometimes the only change in the limb lead electrocardiogram and was not caused by left ventricular hypertrophy.

The deep S wave in leads II and III is determined by a terminal vector that points to the left shoulder: this sequence in activation constitutes the essential ingredient of the pattern.

Now that the pathology of the S₂S₃ pattern has been nailed to an abnormality of the antero-lateral sector of the left ventricle, and its mechanism explained by the resultant change in direction of the terminal vector of the QRS complex, it remains to describe the association of this distinctive pattern with the appearance of a Q wave in lead I and with events in the conducting bundle producing a prolongation of the QRS period.

S₂S₃ in Relation to QI and QRS Widening. The association of a significant Q wave in lead I with conspicuous S waves in patients with cardiac infarction was described by Wilson *et al.* (1933), who named it the *QI pattern*. Their paper illustrated tracings from seven patients, two of whom were examined at necropsy and were found to have occlusion of the anterior descending branch of the left coronary artery and infarction of the anterior and lateral wall of the left ventricle. The

authors, however, stated that a Q in lead I might be inconspicuous or even absent. Evans and Pillay (1957) failed to find a natural Q wave in lead I alone in the electrocardiogram of 500 healthy subjects. Moreover, in 200 consecutive cases with chest pain that in regard to site, character, and relation to exercise and to rest resembled cardiac pain, though the electrocardiogram had been reported as normal, a Q wave in lead I alone was present only once, and in this instance it was associated with the S2S3 pattern (Fig. 4 in their paper).

The characteristic effect of a sizable cardiac infarct on the QRS complex is the appearance of a Q wave, whose disposition among the leads determines the site of the infarct. In about one-half of the cases of infarction with deformity of QRS, however, the terminal portion of the complex is also altered and, as in the case of the initial moiety, all body-surface leads are affected. A study of these changes in the terminal forces or vectors led First *et al.* (1950) to propound the concept of *peri-infarction block*. The changes identified with it follow a prescribed pattern. Thus, the initial vectors point away from the site of the lesion and the terminal vectors towards it. When an antero-lateral infarction, therefore, is associated with the so-called peri-infarction block, the distribution of the electrical forces is such that their terminal components point towards the affected area, causing the S2S3 pattern to appear in the electrocardiogram.

The pattern may accordingly appear when the initial vector is either normal or abnormal. In the former there is no Q wave in lead I, and in the latter there is. In each case the resultant pattern is abnormal, the latter signifying a lesion of the left ventricle large enough to make the initial vector point away from it. Obviously, the larger the Q wave, the more does the initial vector point to the right, and should it be 0.04 sec. in duration it is directed to the right of the vertical throughout this time. When the S2S3 pattern is present, however, the initial and terminal vectors may be widely separated, in the fashion of antero-lateral peri-infarction block, without the mean vector for the first 0.04 sec. being farther to the right than $+90^\circ$. In this event the Q wave will be less than 0.04 sec. in duration. Thus, a Q of 0.03 sec. or even 0.02 sec. can signify in the presence of the S2S3 pattern antero-lateral infarction of considerable size. In this connexion Grant (1956) has emphasized that peri-infarction block can exist with or without widening of the QRS.

Wilson *et al.* (1931) and Widran and Lev (1951) have suggested that the left bundle divides into two main portions, one lying anteriorly and superiorly and the other posteriorly and inferiorly. Grant (1956) considered that when interruption of the anterior fascicle occurs, the muscle normally activated along this path receives its innervation by the posterior fascicle and thence through a syncytium of conducting tissue. Such modification in the terminal phase of ventricular activation causes these forces to be directed upwards, forwards, and to the left.

With a wide QRS, when the axis is directed to the left, it is customary to allude to it as left, or atypical left, bundle-branch block, and since the S2S3 pattern is a common addition, there is now need to analyse this association in greater detail. It would appear that a deformity of the QRS complex caused by a lesion of the left ventricular conducting system is determined first by the site of the interruption, and secondly by the pre-existing state of the muscle. In that the onset of left bundle-branch block itself is usually unaccompanied by great changes in the direction of the initial, terminal, or mean vectors, it follows that when a lesion of the main left bundle has caused the widening of the QRS, the main features of the complex remain much the same as before the onset of the block. Naturally, if the widening is due to peri-infarction block and occurs concurrently with the onset of infarction, the vectors change in direction at that time. Thus, the initial vector may point away from the infarct and the terminal vector towards it. Again, the change in the vectors may take place at once and the widening of the QRS later on. Having regard to these circumstances, and in the absence of adequate histological evidence, our conception of what may be the pathology and pathogenesis underlying some common electrocardiographic patterns is illustrated in Fig. 14 and 15.

Left Axis Deviation. We have accepted the earlier definition of left axis deviation as showing a dominant R wave in lead I and S wave in lead III. The S wave in lead II is small or absent and the amplitude of S in III R is much reduced or even annulled.

FIG. 13.—A deep S wave in leads II, III, and IIIr is the only change in (A) recorded in a patient, aged 75, without chest pain, but the subject of paroxysmal auricular fibrillation through many years. Later he developed right bundle-branch block (B), and succumbed to cardiac infarction at the age of 82. Necropsy showed old and recent antero-lateral infarction.

FIG. 14.—Three cardiograms, showing a wide QRS complex. (A) Left bundle-branch block where both a Q in lead I and the S2S3 pattern are absent; from a woman, aged 51 years, whose only complaint was palpitation. (B) Left bundle-branch block with S2S3; from a man, aged 54, with cardiac pain, in whom there was enlargement of the left ventricle. (C) Peri-infarction block with widening of the QRS: a Q is present in lead I and is associated with the S2S3 pattern: from a man, aged 30 years, with obscure cardiomyopathy.

FIG. 15.—Diagrammatic representation of the vector and the considered underlying anatomical lesion in the separate electrocardiograms in which the presence of the S2S3 pattern is a special feature. (A) Left axis deviation. (B) Left bundle-branch block. (C) Focal block. (D) Left bundle-branch block with S2S3; here the lesion is peripherally disposed and may also be associated with a lesion astride the proximal portion of the main left bundle (P). (E) Peri-infarction block without QRS widening. (F) Peri-infarction block with QRS widening.

I=initial; T=terminal vector.

The mean QRS axis lies at $+15^\circ$ in the frontal plane, and both initial and terminal vectors point in a normal direction. Such a tracing (Fig. 15A) is physiological and is a frequent finding in health, especially when the diaphragm is elevated, as from obesity.

Left Bundle-Branch Block. Here the QRS complex is widened to 0.12 sec., there is no Q wave in lead I, and the S2S3 pattern is absent. The S wave in III is somewhat lessened in III(R) (Fig. 14A and 15B). The directions of the QRS vectors are similar to those of left axis deviation.

The lesion lies astride the main branch of the left bundle and, although conduction of the impulse is impeded, the myocardium need not show significant disease.

Focal Block. This has sometimes been referred to as left ventricular parietal block. The cardiogram shows no Q wave in lead I and no widening of the QRS complex, but the S2S3 pattern is present (Fig. 15C).

The mean QRS axis lies at -30° , the initial QRS vector is normal, and the terminal vector points towards the left shoulder. The significance of this finding as a lone sign has already been discussed.

Left Bundle-Branch Block with S2S3. Here the electrocardiogram shows a wide QRS complex and the S2S3 pattern in addition. There is no Q wave in lead I (Fig. 14B and 15D). Abnormalities of the S-T segment or T wave are usually present.

The QRS cardiac vectors are as of the focal kind, while a lesion of the left main bundle may indeed be present. The muscle itself is affected in the periphery, causing delay in terminal activa-

tion from its spread along slowly-conducting pathways. The disease, therefore, is diffuse, and the frequency of cardiac failure in patients exhibiting this cardiogram bears testimony to this.

Peri-infarction Block without QRS Widening. Here the electrocardiogram shows the S2S3 pattern in addition to a Q wave in lead I, but the QRS is not wide (Fig. 15E).

The vector for the first 0.04 sec. points away from the site of the lesion, and that for the last 0.04 sec. towards it. The injury causing this particular abnormality in the cardiogram is usually infarction from coronary arterial disease (Fig. 12).

Peri-infarction Block with QRS Widening. Here the QRS complex is wide in addition to the S2S3 pattern and a Q in lead I (Fig. 15F). The vectors are widely separated as in the previous kind, and the curve is not indicative of left bundle-branch block, but is the outcome of diversionary activation due to a large peripheral injury and delay in the terminal spread of conduction.

Inasmuch as the pattern is frequently seen in so-called cardiomyopathy (Fig. 14C), the term peri-infarction block should not connote infarction in its literal meaning. Thus, in cardiomyopathy considerable scarring and fibrosis of the myocardium is present in the absence of coronary arterial disease (Evans, 1949; Brigden, 1957). The term may be retained, therefore, as an electrocardiographic conception alone, for the pathology of the underlying lesion may be something other than infarction.

SUMMARY AND CONCLUSIONS

The electrocardiographic pattern S2S3 in which the amplitude of the S wave in leads II and III exceeds that of the R wave, in the absence of an S wave in lead I, was sought in 655 cases. These consisted of 200 healthy subjects, 20 with obesity only, 21 with emphysema, 161 with a raised systemic blood pressure, 200 with cardiac infarction, and 53 with cardiomyopathy.

This distinctive electrocardiogram indicates a fault within the myocardium in the antero-lateral aspect of the left ventricle. Muscular hypertrophy alone, as in systemic hypertension, did not produce the pattern, nor did an alteration in the position of the heart as found in many of the cases with obesity or emphysema.

Naturally, cardiac infarction provided the most frequent cause of the myocardial lesion, when the S2S3 pattern was usually accompanied by other cardiographic changes, but it was also common in patients with cardiomyopathy. Sometimes, the pattern was a lone index of a cardiac infarct.

Exceptionally, the S2S3 pattern was a solitary abnormality in a subject without symptoms, and especially without chest pain. In these rare instances we have regarded the sign, although without pathological proof, as expressing either an impedance of conduction in a peripheral radicle of the left bundle from a small lesion or an immaterial congenital agenesis of this distal portion.

In a patient with chest pain that suggests a cardiac origin the presence of this distinctive cardiographic sign, either alone or in company with changes not in the meantime universally accredited as being significant, is likely to be evidence of cardiac infarction. It is this that allocates to the S2S3 pattern an importance not generally appreciated.

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