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SIGNIFICANCE OF ABNORMALLY SMALL QRS DEFLECTIONS IN ONE OR MORE PRECORDIAL LEADS

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LOW voltage of the QRS deflections in the standard limb leads has engaged the attention of many investigators since the early days of electrocardiography, and the significance attributed to it has undergone many fluctuations. In recent years, since the use of precordial leads has become more widespread, various reports have appeared on low voltage occurring in these leads. Several attempts have been made to show some correlation linking low voltage in the two types of leads, but there has been little agreement among different writers as to whether any correlation of this kind exists. There is, furthermore, no consensus as to whether low voltage in the precordial leads is of any important significance. The present study was undertaken partly to ascertain whether low voltage in the precordial leads has, as a rule, the same or a different origin than low voltage in the limb leads and whether it should be regarded as more, or less, important.

Before proceeding to a consideration of our findings it may be profitable to review some of the earlier work on the significance of low voltage occurring in the limb leads, in the precordial leads, or in both.

EARLIER OBSERVATION ON LOW VOLTAGE

Low Voltage in the Standard Limb Leads.—Low voltage is commonly considered to be present in the standard limb leads if the greatest deflection of the

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QRS complex extends less than 5 mm. above or below the base line in all of them, when the electrocardiograph is so standardized that the introduction of 1 mv. into the circuit produces a deflection of 10 millimeters.

In 1926 three papers dealing with low voltage appeared in the American literature, and all three took a gloomy view of its significance. Sprague and White,[1] reviewing fifty-seven cases, concluded: "Excluding the temporary effect of hypothyroidism, low voltage has never been found in our experience in records from normal hearts." Hepburn and Jamieson[2] summed up their experience thus: "Low voltage (even when) unaccompanied by other electrocardiographic abnormalities is a prognostic sign of serious import." On statistical grounds they considered it to rank second only to bundle branch block as an electrocardiographic sign of ill omen. Master and Pardee[3] included low voltage among a number of specified graphic abnormalities that are "indicative of a diseased heart."

In the following year there appeared a paper by Willius and Killins,[4] whose conclusions were almost directly opposite to those of the previous investigators. They reported a series of 140 electrocardiograms showing low voltage as the sole deviation from the normal, and they concluded that low voltage not accompanied by other graphic abnormalities does not necessarily imply that serious myocardial damage is present. Later studies, likewise carried out on hospital patients, gave intermediate results. Turner,[5] in 1932, reported low voltage in the standard limb leads in about 3 per cent of the routine electrocardiograms taken at the Presbyterian Hospital in New York. He analysed 164 cases and found that 113 of the patients had manifest heart disease and sixty-three of these were in congestive failure; of the remaining fifty-one patients with no organic cardiac disease, twelve had edema or fluid collections in the serous sacs. Barrit,[6] in a hospital series of ninety-four patients, found that when low voltage was accompanied by abnormal T waves there was a much higher incidence of heart disease than when it was the sole graphic abnormality but that even in the latter case heart disease was present in 45 per cent of the patients.

One obvious reason for such divergent conclusions is that the different observers were studying records taken on selected cases and not on "random samples." Before the significance of low voltage could be critically appraised, it was necessary to know its incidence as a physiologic variant in the records of normal persons, and up to the time of the early studies mentioned, no large series of such records had been published. During the past ten years, however, a considerable number of electrocardiograms of healthy persons have been collected and studied by various authors: Table I gives in summary the incidence of low voltage in the standard limb leads in seven such series, totalling 5,500 normal persons. One study was made on a group of college students,[14] two on groups of military aviators,[9,11] others on business and professional men whose ages ranged from 30 to 60 years,[8,12] and one series included women,[13] so that both sexes and a wide range of ages were represented.

Of the total of 5,500 persons, only sixty, or 1.09 per cent, showed low voltage in the standard limb leads. The incidence in each of the separate series is in quite good agreement with this figure, ranging from 0 to 1.6 per cent.

TABLE I. INCIDENCE OF LOW VOLTAGE IN STANDARD LIMB LEADS OF 5,500 HEALTHY INDIVIDUALS

SERIES	TOTAL NUMBER	NUMBER WITH LOW VOLTAGE	PER CENT
Crawford [7]	1,000	14	1.4
Johnson[8]	2,400	26	1.09
Graybiel and co-workers[9]	1,000	16	1.6
Stewart and Manning[11]	500	1	0.2
Larsen and Skulason[12]	100	0	0
Shipley and Halloran[13]	200	3	1.5
Bellet and Kershbaum[14]	300	0	0
Total	5,500	60	1.09

It is apparent from these data that while low voltage in the limb leads may occur in the electrocardiograms of healthy persons, it is an uncommon finding, occurring not much oftener than once in one hundred observations.

One other series is mentioned separately, because its figures are quite different from the rest. This is the report of a study made on 173 civil airline pilots[10] in whose standard limb leads low voltage was found in fourteen, or 8 per cent of the subjects. These pilots were in the same age group as many of the persons in the other series reported in the foregoing discussion, and it is difficult to account for the much higher incidence of low voltage. If these figures are added to the totals in Table I, then of a total of 5,673 healthy persons, there are seventy-four with low voltage, an incidence of 1.3 per cent.

Low Voltage in the Precordial Leads.—With regard to low voltage in the precordial leads, the literature contains a number of reports,[12-16] but for various reasons it is not easy to compare one with another. The method of selecting cases varied from one series to another, and the several investigators did not use the same locations for either exploring or indifferent electrodes. It will probably suffice for our purpose to consider in detail only two of the papers that were published up to 1941.[14,16] Both of these deal specifically with the pathologic significance of low voltage in the precordial leads and both contain comprehensive reviews of earlier work on the subject.

Leach, Reed, and White[16] studied the relationship between the amplitudes of the deflections in the standard limb leads and their size in a single unspecified precordial lead from the region of the apex impulse (probably CF_4). They considered low voltage to be present in this lead when the voltage of the largest QRS deflections in either direction did not exceed 0.5 millivolts. They collected 100 cases with low voltage in the standard leads and normal voltage in Lead IV (Group 1); 100 cases with low voltage in Lead IV and normal voltage in the standard leads (Group 2) and 100 cases with low voltage in both (Group 3). They found evidence of heart disease in 57 per cent of the total composite series of 300 cases: in 65 per cent of Group 1 and in 63 per cent of Group 3 but only in 47 per cent of Group 2 (low voltage in the precordial lead alone). They con-

cluded that not only heart disease, but also general debilitating diseases, changes in the position of the heart, and changes in the conductivity of the adjacent tissues are factors in the production of low voltage; and that, in addition, the thickness of the chest wall and the position of the precordial electrode (they were using only one precordial lead) influenced the voltage of QRS in Lead IV. It was their opinion that the finding of low voltage QRS deflections, whether in the limb or the precordial leads, is, by itself, of little diagnostic value. Since 10 per cent of their entire series of 300 individuals were perfectly healthy, it did not appear to furnish a valuable clue as to the presence or absence of cardiac or noncardiac disease.

Bellet and Kershbaum,[14] studying the same problem, arrived at a rather different conclusion. They collected twenty cases showing low voltage in both limb and precordial leads. In each of these, three precordial leads had been taken, and they required that all show small deflections before low voltage was considered to be present. Most of these cases had been studied before the American Heart Association and the Cardiac Society of Great Britain and Ireland had announced the joint recommendations of their Committees on Standardization of Precordial Leads,[17] and the chest leads used were not, for the most part, those later recommended. They consisted of one from apex to left leg, one from apex to back, and one from left scapula to left leg, so that only one of the precordial positions specified by the Committees on Standardization was included, namely, the region of the apex impulse. In all twenty patients there was evidence of severe myocardial damage, and in eleven myocardial infarction was present. In a control group of fifty patients with low voltage in the limb leads, but normal voltage in the precordial leads, the incidence of severe myocardial damage was much less. It was concluded that whereas the limb leads might show low voltage as a result of extracardiac factors such as edema, serous effusion, emphysema, or an unusual position of the heart in the thorax, the precordial leads are not, as a rule, affected by such factors, and low voltage in these leads is due in almost all cases to serious myocardial disease.

MATERIAL AND METHODS EMPLOYED IN THE PRESENT STUDY

Our material consisted of 100 electrocardiograms showing low voltage in the standard limb leads, drawn in chronological order from the files of the Heart Station and each accompanied by a full set of six precordial leads. These 100 cases were then separated into two main groups, depending on whether or not low voltage occurred in any of the precordial leads as well. The precordial leads were taken from the chest positions C_1 to C_6 specified by the Committees on Standardization,[17] with Wilson's central terminal[19] as the indifferent electrode.

In a series of this type, collected in a hospital, an element of special selection is inevitably introduced, because it is the practice in most clinics to order electrocardiograms only on patients who are strongly suspected of having heart disease; moreover, patients with certain types of cardiac disease are more likely to have precordial leads ordered than are others. The resulting series can therefore hardly be considered a random sample of the general population, or even

of the hospital population. This defect in sampling is freely acknowledged, but it need not be given too much weight in the case of the present study, since we do not intend to draw any broad conclusions as to the numerical incidence of specific diseases associated with low voltage but rather to ascertain what physiologic changes are common to the various types of case in which low voltage occurs.

Criteria for Low Voltage.—In the standard limb leads the specifications of the Criteria Committee of the New York Heart Association[31] were adopted, low voltage being considered to be present when the greatest deflection of the QRS complex does not extend more than 5 mm. above or below the base line in any of the three leads.

It was more difficult to arrive at a satisfactory definition of low voltage in the precordial leads. Others have applied to these leads the same criteria as in the case of the standard limb leads, but it seemed to us that this was hardly justifiable since the mean size of the deflections in the precordial leads is normally so much greater. It was necessary as a preliminary step to consider the range in amplitude of the ventricular deflections in the precordial electrocardiograms of healthy individuals whose curves had been taken with the same technique as that used in our cases. In this way a standard minimal voltage could be set for each lead, and any considerably smaller voltage could reasonably be designated as low. Two studies satisfactory for our purpose have been reported.

Kossmann and Johnston,[18] using Wilson's central terminal and the chest positions C_1 to C_5, studied the precordial Leads V_1, V_2, V_3, V_4, and V_5 in thirty normal students. Table II represents a condensation of some of their findings: it lists the minimum, maximum, and mean values for the amplitude of the R, S, and RS deflections in each lead. The RS, or intrinsic deflection in any lead, is measured by adding the voltages of the R and S deflections; this gives a truer representation of the magnitude of the potential variations than does the largest QRS deflection measured from the base line. The minimum values found for the RS deflection were 1.5 mv in Lead V_2, 1.26 mv in Lead V_3, 1.8 mv in Lead V_4, and 1.12 mv in Lead V_5.

TABLE II. MEASUREMENTS OF THE QRS DEFLECTIONS IN THE PRECORDIAL LEADS, EXPRESSED IN TENTHS OF A MILLIVOLT*

LEAD	R			S			RS		
	MIN.	MAX.	MEAN	MIN.	MAX.	MEAN	MIN.	MAX.	MEAN
V_1	1.0	9.6	4.16	3.4	24.0	11.05	6.6	26.8	15.21
V_2	4.0	20.8	9.05	3.0	38.8	16.23	15.0	46.0	25.27
V_3	6.0	54.6	16.70	0.0	22.0	9.05	12.6	54.6	25.75
V_4	12.2	46.0	22.31	0.0	16.0	5.32	18.0	51.6	27.63
V_5	8.8	33.0	18.78	0.0	9.6	1.93	11.2	33.2	20.70

*Adapted from Kossmann and Johnston.[18]

Bryant,[20] using the same technique, measured the RS deflection in Leads V_2 and V_4 in the electrocardiograms of 103 normal persons. In each of these leads the minimum voltage was 1.0 millivolts.

If the findings in these two series are combined, then the minimum values for the RS deflection in each lead are: 1.0 mv in Lead V_2, 1.26 mv in Lead V_3, 1.0 mv in Lead V_4, and 1.12 mv in Lead V_5.

Neither Kossmann and Johnston nor Bryant included Lead V_6 in their studies. This is a lead from the left midaxillary line at the level of the apex impulse. It has perhaps a closer relationship to the standard limb leads than have most of the other precordial leads, since it is influenced chiefly by electrical forces more nearly in the frontal plane. In order to ascertain the range of its amplitude in normal individuals as well as to add to the number of cases in which the other precordial leads had been studied, we measured the intrinsic deflections of Leads V_2, V_3, V_4, V_5, and V_6 in 100 cases with normal amplitude in the limb leads. These were not necessarily all normal records, but we did exclude cases of anterior, lateral, and posterolateral infarction, since these are commonly the cause of small QRS deflections in Leads V_4, V_5, and V_6. In these 100 cases, an RS deflection of 0.9 mv or less occurred only once in Lead V_2, twice in Lead V_3, once in Lead V_4, and in no case in Lead V_5, and an RS of 0.7 mv or less occurred only twice in Lead V_6. Accordingly it appeared reasonable to accept as arbitrary values for low voltage a total RS deflection of 0.9 mv or less in Leads V_2, V_3, V_4, V_5 and of 0.7 mv or less in Lead V_6. These figures are all well below the minimum values found by Kossmann and Johnston and by Bryant.

Low voltage in Lead V_1 was not considered to have any significance for our purpose, since there is quite a wide variation in the amplitude of its deflections in normal persons.

RESULTS

Using the criteria specified, we found that of the main group of 100 patients showing low voltage in the standard limb leads (to which we shall for convenience hereafter refer as Group A), sixty-five (to be designated subgroup B) showed low voltage in one or more of the precordial leads as well. Table III shows the distribution by diagnosis of the patients in both main group and subgroup. In both, the commonest diagnosis was myocardial infarction, which was present in forty-three of the 100 in the main group and thirty-five of the sixty-five in the subgroup. Of the remaining fifty-seven patients in the main group, thirty-seven had extracellular collections of fluid (hydrothorax, hydropericardium, ascites, marked congestion or edema of the lungs, or subcutaneous edema) and two had pronounced pulmonary emphysema. In the subgroup there were thirty patients without myocardial infarction and of these, nineteen had extracellular collections of fluid (twelve had hydrothorax, six had marked congestion or edema of the lungs, one had subcutaneous edema only) and one had pulmonary emphysema.

In the main group there were six patients and in the subgroup four patients with no evidence of heart disease and without extracellular fluid accumulations, obesity, emphysema, or hypothyroidism.

TABLE III. GROUP A, 100 PATIENTS IN WHOM LOW VOLTAGE OCCURRED IN STANDARD LIMB LEADS; SUBGROUP B, 65 OF PATIENTS IN GROUP A IN WHOM LOW VOLTAGE OCCURRED IN PRECORDIAL LEADS ALSO

	GROUP A			SUBGROUP B		
Myocardial infarction						
With extracellular fluid						
Hydrothorax, etc.	7			7		
Pulmonary congestion or edema	11			7		
Peripheral edema only	2			0		
Total with extracellular fluid		20			14	
Without extracellular fluid		23			21	
Total myocardial infarcts			43			35
Arteriosclerotic heart disease						
With extracellular fluid						
Hydrothorax, etc.	7			4		
Pulmonary congestion or edema	10			6		
Peripheral edema only	9			1		
Total with extracellular fluid		26			11	
Without extracellular fluid		8			4	
Total arteriosclerotic heart disease			34			15
Constrictive pericarditis, with hydrothorax			4			3
Hodgkin's disease, with hydrothorax			1			1
Carcinoma of liver, with ascites			1			0
Foreign body in heart, with hydrothorax			1			1
Metastatic carcinoma of heart, with hydrothorax			1			1
Massive pulmonary embolus with gangrenous infarct			1			0
Rheumatic heart disease, with hydrothorax			3			2
Rheumatic heart diease, peripheral edema only			1			0
Idiopathic cardiac hypertrophy			2			2
Pulmonary emphysema, severe			2			1
No heart disease (no emphysema, serous effusion, edema, obesity, or myxedema)			6			4
Total			100			65

Table IV lists in detail the sixty-five cases included in subgroup B, all showing low voltage in one or more of the precordial leads as well as in the standard limb leads. It gives the age, sex, diagnosis, height of the P and T waves, presence or absence of serous effusion, congestion of the lungs, and peripheral edema; and it gives the amplitude of the RS deflection in each of Leads V_4, V_5, and V_6, as well as in the lead in which it is greatest.

It will be noted that in only three instances were the deflections low in all six precordial leads. One of these patients had a hydropneumopericardium; one had cardiac hypertrophy of undetermined etiology, without congestive failure; the third was a young woman, 31 years of age, without evidence of any organic disease.

Low voltage occurred in three precordial leads in twelve cases; in two leads in twenty-six cases; and in a single lead in twenty-four cases.

It occurred in Lead V_2 in three cases, in Lead V_3 in four cases, in Lead V_4 in twenty-two cases, in Lead V_5 in forty cases, and in Lead V_6 in fifty-six cases.

TABLE IV. MEASUREMENTS OF RS, P, AND T DEFLECTIONS IN TENTHS OF A MILLIVOLT

CASE	SEX	AGE	RS V_4	RS V_5	RS V_6	LARGEST RS DEFLECTION	P	T	CLINICAL DIAGNOSIS	PLEURAL OR PERICARDIAL EFFUSION OR ASCITES	CONGESTION OR EDEMA OF THE LUNGS	PERIPHERAL EDEMA	OTHER CLINICAL FEATURES
1	M	38	10	6	7	V_2 25	1.5	4.0	Myocardial infarction	0	0	0	
2	M	54	9	4	4	V_3 14	0.75	3.0	Myocardial infarction	0	0	0	
3	F	41	6	1	4	V_2 22	2.0	2.5	Myocardial infarction	0	0	0	
4	M	49	14	14	7	V_5 14	1.0	2.5	Myocardial infarction	0	0	0	
5	M	57	17	7	5	V_3 30	1.0	0.5	Myocardial infarction	0	0	0	
6	F	55	7	4	5	V_2 17	2.0	1.0	Myocardial infarction	0	0	0	
7	M	45	23	14	2	V_4 23	1.5	0.0	Myocardial infarction	0	0	0	
8	M	46	8	8	7	V_2 18	1.5	0.5	Myocardial infarction	0	0	0	
9	M	53	13	7	9	V_2 17	1.0	2.0	Myocardial infarction	0	0	0	
10	M	40	20	18	7	V_3 20	1.0	0.75	Myocardial infarction	0	0	0	
11	M	45	26	11	5	V_4 26	1.0	0.2	Myocardial infarction	0	0	0	
12	M	48	10	5	5	V_2 20	0.5	0.75	Myocardial infarction	0	0	0	
13	M	58	15	12	4	V_3 20	1.5	2.0	Myocardial infarction	0	0	0	
14	M	46	10	7	6	V_2 30	1.5	1.0	Myocardial infarction	0	0	0	
15	M	57	15	14	4	V_2 17	1.5	0.75	Myocardial infarction	0	0	0	
16	M	42	22	10	5	V_4 22	1.5	2.0	Myocardial infarction	0	0	0	
17	M	52	8	6	6	V_2 20	1.5	1.5	Myocardial infarction	0	0	0	
18	M	61	16	12	6	V_4 16	1.5	3.0	Myocardial infarction	0	0	0	
19	F	78	15	13	6	V_4 15	0.25	0.25	Myocardial infarction	0	0	0	
20	M	63	9	3	5	V_3 15	2.5	0.5	Myocardial infarction	0	0	0	
21	M	37	14	5	4	V_2 20	2.0	1.0	Myocardial infarction	—	—	0	Bilateral hydrothorax
22	M	68	10	9	5	V_2 13	1.0	1.0	Myocardial infarction	—	—	—	Ascites
23	M	29	16	13	4	V_2 40	0.5	0.75	Myocardial infarction	—	0	0	Left hydropneumothorax
24	M	64	11	7	5	V_1 25	0.1	0.1	Myocardial infarction	—	0	—	Hydrothorax and ascites
25	M	46	20	5	5	V_2 23	1.5	0.1	Myocardial infarction	—	0	—	Bilateral hydrothorax
26	M	65	8	8	8	V_3 25	1.0	0.1	Myocardial infarction	—	—	—	Bilateral hydrothorax
27	M	58	15	5	8	V_2 25	1.5	1.0	Myocardial infarction	—	—	—	Hydrothorax and ascites

28	M	58	20	2	3	V_3	22	0.1	0.1	Myocardial infarction	—	—	—	Hydrothorax
29	M	51	4	4	3	V_2	11	1.5	1.5	Myocardial infarction	0	0	—	Emphysema
30	F	53	11	8	4	V_2	14	1.0	0.5	Myocardial infarction	0	—	—	Pulmonary edema
31	M	51	7	3	4	V_3	10	1.2	0.1	Myocardial infarction	0	—	—	Pulmonary edema
32	M	55	10	3	6	V_2	30	1.5	1.0	Myocardial infarction	0	—	—	
33	M	56	18	6	5	V_2	20	0.75	0.5	Myocardial infarction	0	—	—	
34	M	63	9	3	5	V_3	15	2.5	0.5	Myocardial infarction	0	—	0	Pulmonary infarct and pulmonary edema
35	M	66	3	14	10	V_4	17	1.5	1.75	Myocardial infarction	0	—	—	Pulmonary infarct with gangrene
36	F	60	6	7	6	V_2	12	0.0	0.25	Arteriosclerotic heart disease	0	0	0	Obesity
37	F	47	9	12	10	V_3	12	0.5	1.75	Hypertensive heart disease	0	0	0	
38	M	49	15	8	4	V_4	15	1.2	1.2	Arteriosclerotic heart disease	0	0	0	Emphysema and obesity
39	M	68	15	12	5	V_4	15	1.0	2.0	Arteriosclerotic heart disease	0	0	0	Carcinoma of sigmoid
40	M	53	15	7	4	V_3	21	0.5	0.5	Arteriosclerotic heart disease	—	—	—	Bilateral hydrothorax
41	M	71	25	20	5	V_4	25	0.25	0.25	Arteriosclerotic heart disease	—	—	—	Hypertension and bilateral hydrothorax
42	M	49	15	7	5	V_3	21	0.0	0.1	Arteriosclerotic heart disease	0	—	—	Hypertension
43	M	48	7	8	8	V_2	18	0.75	0.5	Arteriosclerotic heart disease	0	—	—	Hypertension
44	M	67	6	10	7	V_5	10	0.5	1.0	Arteriosclerotic heart disease	0	—	0	Carcinoma of esophagus; infarct right lung
45	M	44	19	5	4	V_3	21	1.5	0.5	Arteriosclerotic heart disease	0	—	—	
46	M	58	18	12	6	V_2	25	0.0	0.1	Arteriosclerotic heart disease	0	—	—	Auricular fibrillation
47	M	55	15	13	3	V_3	15	1.5	0.75	Arteriosclerotic heart disease	0	0	—	Emphysema
48	M	63	14	5	7	V_3	28	0.0	0.1	Arteriosclerotic heart disease	—	—	—	Hydrothorax, autirular fibrillation
49	M	54	13	12	7	V_3	25	0.75	0.1	Arteriosclerotic heart disease	0	—	—	Hypertension
50	M	76	19	11	4	V_4	19	1.0	1.25	Arteriosclerotic heart disease	—	—	—	Bilateral hydrothorax; carcinoma of prostate

TABLE IV. MEASUREMENTS OF RS, P, AND T DEFLECTIONS IN TENTHS OF A MILLIVOLT—CONT'D

CASE	SEX	AGE	RS V_4	RS V_5	RS V_6	LARGEST RS DEFLECTION	P	T	CLINICAL DIAGNOSIS	PLEURAL OR PERICARDIAL EFFUSION OR ASCITES	CONGESTION OR EDEMA OF THE LUNGS	PERIPHERAL EDEMA	OTHER CLINICAL FEATURES
51	F	22	5	5	4	V_2 8	2.0	0.5	Tuberculous pericarditis	—	0	0	Pneumohydropericardium and pneumohydrothorax
52	M	52	11	7	4	V_2 10	1.5	0.1	Constrictive pericarditis	—	—	—	Bilateral hydrothorax and ascites
53	M	52	12	7	4	V_3 15	0.1	0.1	Constrictive pericarditis	—	—	—	Hydrothorax and ascites
54	M	54	6	6	5	V_1 18	0.5	0.5	Hodgkin's disease (mediastinal)	—	0	0	Massive left hydrothorax
55	M	56	9			V_2 17	0.6	0.5	Foreign body in ventricular wall	—	0	0	Hydrothorax and hydropericardium
56	M	41	9	10	7	V_3 23	1.5	0.1	Rheumatic heart disease; mitral stenosis	—	—	—	Bilateral hydrothorax
57	M	59	22	18	7	V_4 22	0.0	0.1	Rheumatic heart disease; mitral stenosis	—	—	—	Hydrothorax, right; auricular fibrillation
58	M	64	13	7	4	V_4 13	1.2	1.0	Metastatic carcinoma to heart and pericardium	—	—	0	Hydrothorax, right; carcinoma of trachea
59	F	36	11	10	4	V_4 11	1.75	0.10	Idiopathic hypertrophy	0	0	0	
60	F	20	4	7	5	V_2 8	1.5	0.75	Idiopathic hypertrophy	0	0	0	
61	M	47	12	11	4	V_4 12	1.5	2.5	Pulmonary emphysema	0	0	0	Bronchial asthma
62	F	31	9	7	4	V_4 9	1.0	1.0	No heart disease	0	0	0	Psychoneurosis, neurocirculatory asthenia
63	M	55	6	10	9	V_2 17	0.7	0.5	No heart disease	0	0	0	Carcinoma of stomach
64	F	53	9	5	4	V_2 12	1.75	1.0	No heart disease	0	0	0	Parosysmal tachycardia
65	M	30	15	9	2	V_2 17	0.75	0.75	No heart disease	0	0	0	

In many instances the deflections were very large in some leads although small in one or more of the others. In twenty-nine of the sixty-five cases the largest RS deflection was 2.0 mv or more, and in one case it was 4.0 millivolts. The largest deflection occurred in Lead V_2 in twenty-seven cases, in Lead V_3 in eighteen cases, in Lead V_4 in fourteen cases, in Lead V_1 in three cases, and in Lead V_5 in three cases. This is in contrast to the usual finding in normal persons, where the mean amplitude of RS tends to increase from Lead V_1 to V_4. This shift of the largest deflection toward the right (from Lead V_4 to V_2) suggests that the electrical axis was rotated from its normal position toward the sagittal plane.

PHYSIOLOGIC CONSIDERATIONS

Wilson pointed out in 1930, in a discussion of the factors theoretically capable of leading to a reduction in the amplitude of the electrocardiographic deflections, that such reduction might be accomplished in one or more of three ways:

1. By conditions that affect the efficiency of the myocardium itself, preventing it from developing an electromotive force of normal magnitude.
2. By conditions that alter the electrical conductivity of the tissues surrounding the heart.
3. By conditions that alter the direction of the mean electrical axis of the heart, thereby changing the size of its projection upon any given lead.

These three principles will be developed more fully later in this paper, when the various types of conditions that were found to be associated with low voltage in this series are discussed.

Conditions Affecting the Total Electromotive Forces Developed by the Myocardium.—Under this heading we shall discuss in particular myocardial infarction and arteriosclerotic heart disease. Myocardial infarction was the most common cardiac abnormality in both groups shown in Table III; it was present in forty-three of one hundred cases in Group A and in thirty-five of the sixty-five cases in subgroup B. To understand how this condition may give rise to low voltage it is instructive to compare a normal precordial electrocardiogram with those from typical cases of myocardial infarction.

The precordial leads of a healthy young man of 30 years are shown in Fig. 1,*A*. An R wave is present in all leads; it is relatively small in Lead V_1 and grows progressively larger in the leads from points farther to the left until it attains its maximum voltage of 2.1 mv in Lead V_4; it then becomes smaller again in Lead V_5, and in Lead V_6 it is only 1.2 millivolts. The S wave is relatively large in Leads V_1, V_2, and V_3, then rapidly diminishes until in Lead V_6 it is merely vestigial.

Fig. 1,*B*, represents the precordial electrocardiogram of a 46-year-old physician who had had an acute coronary occlusion five days before it was taken. It is entirely typical of anteroseptal myocardial infarction. In contrast to the normal record, *A*, the R wave is completely absent in Leads V_1, V_2, and V_3 and is only rudimentary in Lead V_4. In each of these leads there is now only a deep

QS wave, reflecting the negative potential of the ventricular cavity which is transmitted passively through the electrically inert infarcted muscle. The fifth and sixth precordial positions were apparently somewhat lateral to the infarct and the still-healthy myocardium beneath them produced a positive potential as shown by the emergence of the R waves in Leads V_5 and V_6, but the infarcted area was close enough to transmit some cavity negativity to the exploring electrode; moreover, the positivity previously contributed by the adjacent now-infarcted muscle has been withdrawn, with the result that these R waves did not attain their usual size. The RS-T elevation and late inversion of the T waves complete the electrocardiographic picture of recent infarction. The standard limb leads and unipolar extremity leads are shown in Fig. 1,*C*. They exhibit unusually small deflections but show no diagnostic features.

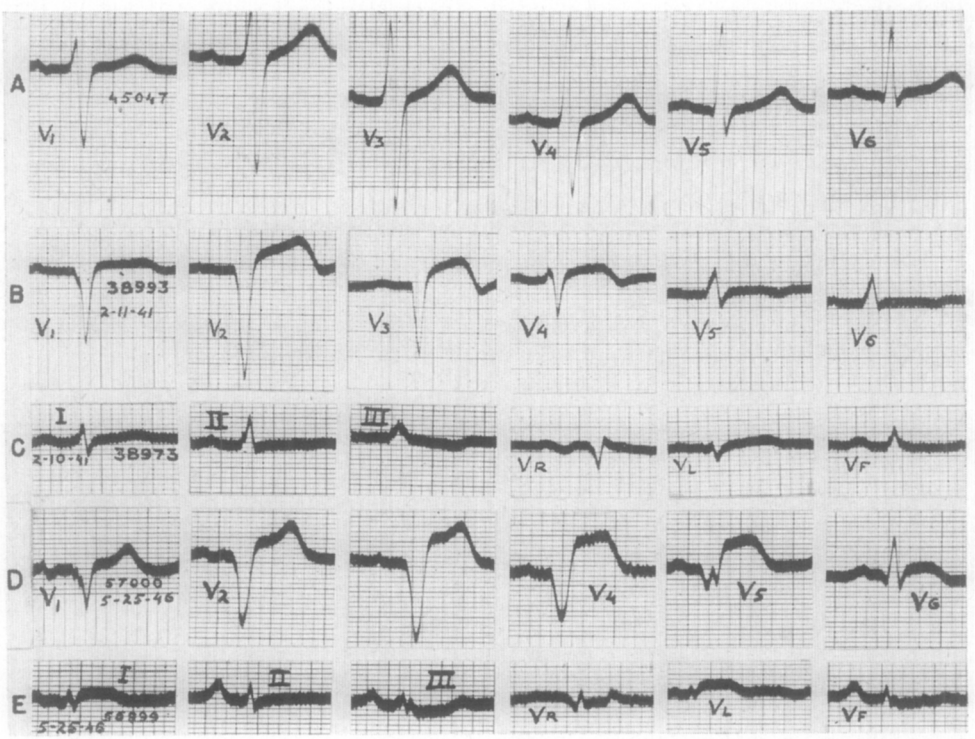

Fig. 1.—*A*, Normal precordial electrocardiogram. *B*, Precordial electrocardiogram of patient with recent anterior myocardial infarction, showing low voltage in Leads V_5 and V_6. *C*, Low voltage in limb leads; same patient as in *B*. *D* and *E*, Electrocardiogram of another patient with anterior infarction, showing low voltage in Leads V_5 and V_6 and in the limb leads.

Fig. 1,*D*, represents the precordial electrocardiogram in another typical case of anterior myocardial infarction. The patient, a 63-year-old man, had had a coronary occlusion ten days before this record was taken. The precordial leads are very similar to those in the previous case, showing absence of the R waves and deep QS waves in Leads V_1, V_2, V_3, and V_4. In Lead V_5 the Q wave

is still present but is not very deep, and a small upward deflection that barely reaches the base line represents the R wave produced by surviving muscle under the exploring electrode. Lead V_6 shows a tiny Q wave also, but this lead was far enough lateral to the main area of infarction to have an R wave 7 mm. tall. The standard limb leads are shown in Fig. 1,*E*. In these the QRS complexes are small in all leads, reflecting the low amplitudes of Leads V_5 and V_6; there is a small Q wave in Lead I with upward displacement of RS-T and terminal inversion of the T wave. There is S-T depression in Lead III. The deflections in the unipolar extremity Leads V_R, V_L, and V_F are very small.

In both cases it will be seen that while the deflections in Leads V_5 and V_6 and in the limb leads are small, the deflections in Leads V_2 and V_3 are well within the normal range of amplitude. Because these leads from the right and mid-precordium are influenced chiefly by forces acting in the sagittal plane, they have little relationship to the limb leads. The latter lie in the frontal plane and are accordingly more likely to resemble Leads V_5 and V_6.

Fig. 2 is particularly instructive as an example of conditions that may lead to a definite increase in QRS amplitude in certain precordial leads while at the same time producing a decrease in others and in the limb leads. It shows serial

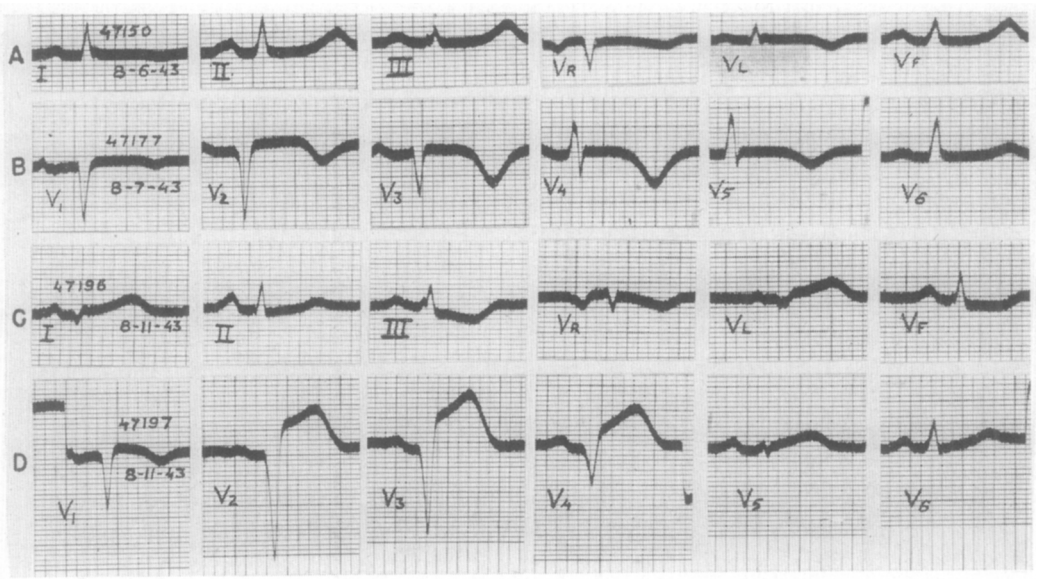

Fig. 2.—*A* and *B*, Electrocardiogram taken within twenty-four hours after the patient had had a small anterior infarct. *C* and *D*, Electrocardiogram of same patient four days later. With extension of the infarcted area, there has been an increase in the size of the deflections in Leads V_2 and V_3 but a decrease in Leads V_5 and V_6 and in the limb leads.

curves of a 41-year-old woman who had been known to have hypertensive and arteriosclerotic heart disease for some time. On Aug. 6, 1943, she had a sudden attack of severe persistent substernal pain. The standard limb leads shown in Fig. 2,*A*, were taken a few hours later and the precordial leads, *B*, the next day. The precordial leads indicate a small anteroseptal infarct; they show complete

absence of the R wave in Lead V_2, an embryonic R wave in Lead V_3, and deep inversion of the T waves in Leads V_2, V_3, V_4, and V_5. The patient continued to have pain, and it is most interesting to compare the first set of records described with those taken four days later, Aug. 11, 1943. In the case of the latter, Lead I,*C*, shows a Q wave not previously present and reduced QRS amplitude. The precordial leads, *D*, show changes suggesting that there has been a lateral extension of the infarct. The R wave, previously absent in Lead V_2 only, is now absent in Leads V_1, V_2, V_3, and V_4. The amplitude of the QS wave in V_2 and V_3 is much greater than it was on August 7, whereas in Leads V_5 and V_6 and in the limb leads the QRS amplitude has become distinctly smaller. The reason for these rather paradoxical changes is that because of the wider extent of infarcted and therefore electrically inert muscle, more of the cavity negativity was transmitted to the electrodes in the C_1 and C_2 positions, and hence the (negative) amplitude in these leads is greater than it was when the infarct was smaller. Leads V_5 and V_6 are affected in the opposite way. Being lateral to the actual infarct, they tend to have positive R waves derived from the surviving healthy muscle underlying the exploring electrode. These R waves do not, however, attain their previous amplitude, partly because some of the forces formerly contributing to them had originated in muscle subsequently infarcted, and were accordingly abolished, and partly because the wider transmission of cavity negativity through the more extensive infarct tends to neutralize those that remain. The limb leads, subject to the same influences as Leads V_5 and V_6, were likewise reduced in amplitude with extension of the infarct.

The examples we have presented have all been from cases of anterior infarction, but it can be shown that posterolateral infarction may affect the limb leads and the leads from the left precordium in similar fashion.

We have pointed out that anterior infarction very frequently leads to low voltage in Leads V_5 and V_6, but it is not our intention to suggest that it invariably does so or that when it does it is necessarily accompanied by low voltage in the limb leads. For example, the tracings shown in Fig. 3,*A* and *B*, are those of a patient with anteroseptal infarction. The precordial leads, *B*, show all the characteristic QRS changes and yet the deflections of Leads V_5 and V_6 and of the limb leads, *A*, are of normal amplitude. Lead V_4, it is true, shows low voltage. Fig. 3,*C* and *D*, represents the limb and precordial leads from another case of anterior infarction. In this case Leads V_4 and V_6 do indeed show low voltage, and yet the deflections in the limb leads are quite large. These two cases are included to show how variable are the combinations that can occur in the different leads, and it is emphasized again that while we have indicated certain general relationships between the QRS amplitudes in Leads V_5 and V_6 and those in the standard leads, it is with full realization that these relations are not always present.

Arteriosclerotic Heart Disease: This is another diagnosis which occurred frequently among the cases in this series (Table III). The term does not have a very precise significance and is often used in cases in which the heart does not show any gross abnormality when examined post mortem. On histologic ex-

amination, however, the myocardium may exhibit extensive streaky and patchy replacement of the muscle by fibrous tissue. Each muscle fiber thus replaced represents the loss of a functioning unit normally contributing to the total electromotive force developed by the heart, and the elimination of a large number of such units may well lead to a pronounced reduction in the size of the electrocardiographic deflections through its effect on the mass of the cardiac muscle. An autopsy was performed in only one of the cases of our series falling into this category. Diffuse fibrosis and scarring of the myocardium was found. A pulmonary infarct was also present in this instance.

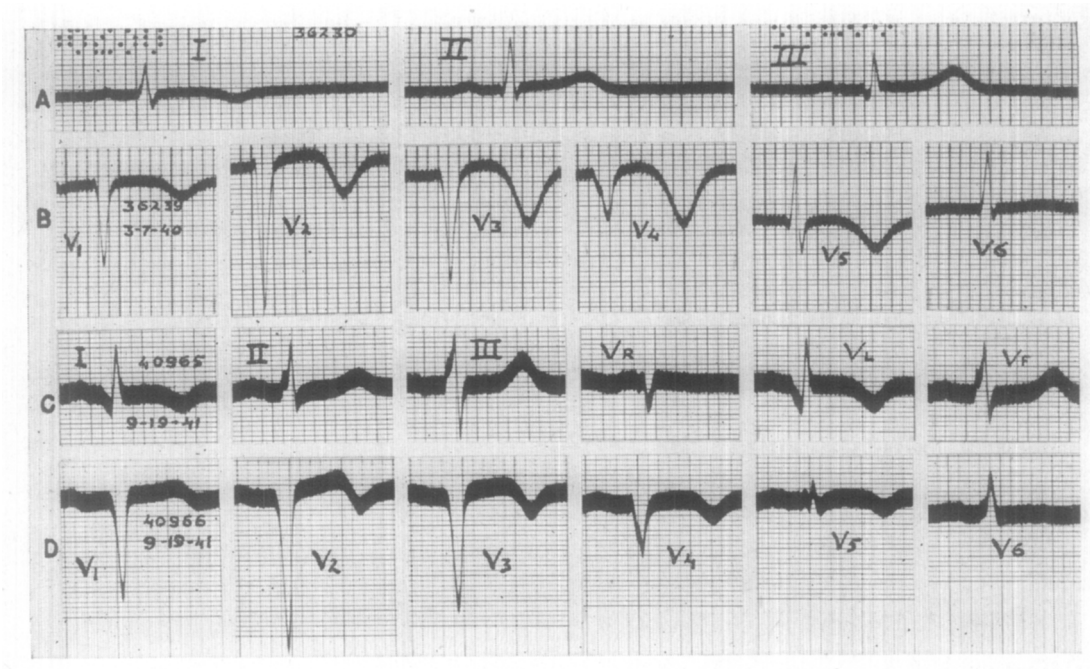

Fig. 3.—*A* and *B*, Electrocardiogram of a patient with an anterior infarct, showing normal voltage in Leads V_5 and V_6 and in the limb leads. *C* and *D*, Electrocardiogram of another patient with an anterior infarct, showing low voltage in Leads V_5 and V_6 but normal voltage in the limb leads.

Conditions That Affect the Conductivity of the Tissues Surrounding the Heart.—It will be seen from Table III that after exclusion of the thirty-five cases of myocardial infarction there remain thirty instances of low voltage in one or more precordial leads. In twelve of these the patient had a pleural or a pericardial effusion. There were also six examples of pronounced pulmonary congestion or edema of the lungs, one of marked pulmonary emphysema, and one instance of peripheral edema alone in this group. A number of experimental studies have a bearing upon the manner in which these conditions tend to produce low voltage, even in the absence of primary heart disease.

Eyster and associates[22] found that when extensive edema was produced in animals by infusion of the tissues with isotonic salt solution, a conspicuous fall in total body impedence was recorded under certain experimental conditions.

In their impedence experiments, however, these writers employed alternating currents of a frequency (4×10^4 cycles per second) far above any of those found in the electrocardiogram. The bearing of this work upon the impedence offered by the tissues to currents of electrocardiographic frequency is therefore open to serious question. In the experiments of Katz and co-workers,[23] parts of the heart surface were short-circuited by means of lead or tinfoil, or insulated by means of glass and rubber. Both of these procedures naturally reduced the voltage of the electrocardiogram. Since the conductivity of metals on the one hand and of dielectrics, such as glass or rubber, on the other are of an order entirely different from that of tissue or tissue fluid, such experiments probably have little practical bearing on the problems under consideration here.

Theoretically, an increase in the amount of extracellular fluid might be expected to reduce the voltage of the electrocardiogram by its short-circuiting effect upon the cardiac currents, since such fluid, compared to organized tissues, offers a relatively low resistance to low-frequency current. Kaufman and Johnston[24] in experiments on animals measured the specific resistances of the tissues surrounding the heart and found values approximately as follows for the different tissues (expressed in ohms per cubic centimeter): muscle, 575; liver, 506; lungs (normal inspiration), 744; lungs (superinflated), 1,227; pericardium, 405; serum, 98; blood, 185; fat, 1,808.

In the healthy individual the tissues chiefly concerned are pericardium, muscle, liver, and normally inflated lung, and the differences in specific resistance between these tissues are sufficiently small to justify the assumption that the cardiac currents are distributed in accordance with the principles that govern current flow in a homogeneous volume conductor.[29] Since the specific resistance of serum is only 98 ohms per cubic centimeter, it is probable that collections of fluid near the heart, whether in the form of massive effusion into the pericardial or pleural spaces or of abnormal extracellular accumulations in the alveoli or connective tissue of the lungs (as, for example, in pulmonary congestion or pulmonary edema), have a more or less pronounced short-circuiting effect upon the cardiac currents and reduce the potential variations recorded at the body surface.

On the other hand, air-containing spaces, such as are present in pneumopericardium and pneumothorax, must act as insulators and would be expected to diminish the size of the electrocardiographic deflections in some leads if not in all. In severe emphysema the lung tissue is thin, atrophic, and relatively avascular and the alveolar spaces are abnormally large, so that it may be that in this condition the pulmonary resistance approaches that for superinflated lung, with consequent insulating effect. It must be conceded, however, that without more accurate knowledge of the actual magnitude of the changes in specific resistance that occur under these various circumstances it is difficult to estimate their effects upon the electrocardiogram with any pretense to accuracy.

The smallest precordial deflections observed in our series occurred in the electrocardiogram of a 22-year-old girl with tuberculous polyserositis. This patient had effusions of fluid into the pericardial and left pleural spaces. When the two upper records of Fig. 4 were taken Feb. 24, 1939, both of these had been aspirated and replaced with air. Roentgenographic examination February 23

had been reported as showing air and fluid in the pericardial sac, with marked thickening of the pericardium, and air and fluid in the left pleural space. The QRS deflections in the limb leads were very small (Fig. 4,*A*), but those of the precordial leads were even smaller. This is the sole instance in which the precordial deflections were smaller than those of the limb leads. The explanation appears to be that the combination of fluid, air, and thickened pericardium interposed between the chest electrodes and the precordium, with the short-circuiting effect of the one added to the insulating effect of the other, largely prevented the transmission of the potential variations of the cardiac surface to

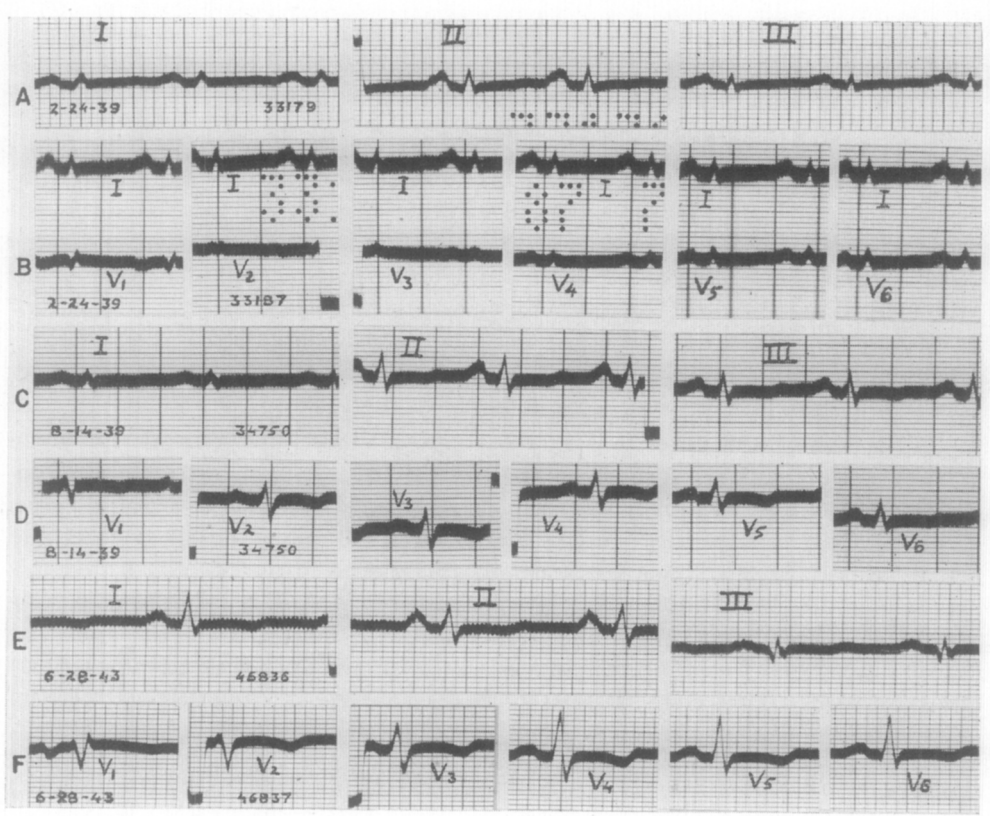

Fig. 4.—Electrocardiograms of a young woman with hydropneumopericardium of tuberculous origin. *A* and *B*, Extremely low voltage, especially in the precordial leads, in the presence of air and fluid in the pericardial sac. *C* and *D*, Increased voltage after absorption of air from pericardial space; fluid still present. *E* and *F*, Further increase in voltage after absorption of fluid.

the precordial electrodes. It is interesting that the smallest deflections occurred in Leads V_2 and V_3, possibly because with the patient in the supine position the enclosed air lay just beneath the part of the precordium from which these leads were taken. Apparently, the potential variations of the sides and back of the heart were better transmitted to the body surface, for the deflections in Leads V_5 and V_6 and in the limb leads were somewhat larger.

The standard and precordial leads taken Aug. 14, 1939, are shown in Fig. 4,*C* and *D*. Roentgenograms taken on this date show that the air but not all of the fluid had been absorbed from the pericardial sac and that the left hydropneumothorax was still present. The deflections of the standard limb leads are seen to have increased moderately and those of the precordial leads considerably, although both are still decidedly subnormal in size.

The two lowermost records in Fig. 4,*E* and *F*, were taken June 28, 1943. Roentgenograms on this date showed the lungs completely expanded and no air or fluid in the pericardial sac. The pericardium itself, however, was 5 to 8 mm. thick, and the roentgenkymogram showed diminished cardiac pulsations. Both limb and precordial leads showed a further increase in the size of the ventricular deflections, but it may be that the extreme thickness of the pericardium still prevented these deflections from attaining normal amplitudes.

Low Voltage With no Pathologic Changes in the Heart or Neighboring Structures.—In our main group of one hundred cases there were six and in the subgroup four patients with no evidence of heart disease, emphysema, or hypothyroidism who were not obese and had no increase in extracellular fluid. In the four last-mentioned cases the clinical diagnoses were (1) minimal pulmonary tuberculosis, arrested; (2) adenocarcinoma of the stomach; (3) neurocirculatory asthenia; (4) no disease. The last two cases will be discussed in detail below. As to the manner in which low voltage is produced in such cases, we can offer only some speculations which leave much to be desired.

The size of the deflections in the standard limb leads depends not upon the total electromotive force generated by the myocardium but upon the projection of this electromotive force, considered as a vector, upon the frontal plane. This component is large when the spatial electrical axis is nearly parallel and small when it is nearly perpendicular to this plane. Otto[26] demonstrated this experimentally by rotating the heart forward on a basal transverse axis. He found that the deflections in the limb leads were largest when the apex was most caudad and became progressively smaller as the apex was tilted anteriorly and cephalad. They were smallest when the long axis of the heart was perpendicular to the frontal plane.

Meek and Wilson[27] rotated the canine heart about its longitudinal and anteroposterior axes and produced marked alterations in the position of the cardiac electrical axis. In this way they obtained either pronounced left or pronounced right axis deviation with corresponding variations in the size of the ventricular deflections.

Cohn and Raisbeck[28] used another approach to the same problem. Instead of displacing the heart itself, they ingeniously rotated the apices of the "Einthoven triangle," and therefore the directions of the standard limb leads, through 360 degrees in the frontal plane. By this method they were able, both in the case of subjects with normal and those with hypertrophied hearts, to obtain tracings depicting either extreme right or extreme left axis deviation.

These and other experiments indicate that position of the cardiac electrical axis and the size of the deflections in the limb leads are determined, to a large

extent, by such factors as the position of the heart and the contour, symmetry, and thickness of the chest wall. In certain cases similar factors probably explain the occurrence of unusually small deflections in the precordial electrocardiograms of subjects who show no other evidence of disease. Thus low voltage is common in Leads V_3 or V_4 when these are from points in the transitional zone where the potential variations of one ventricular surface tend apparently to cancel those transmitted from the other.

There are cases, however, in which most or all of the precordial leads exhibit low voltage, and it is not easy to explain exactly how this happens. Two cases in our series fall into this category. Both displayed unusual features in addition to the low voltage and presented some initial diagnostic difficulties.

CASE 1.—T. M., a young woman, 31 years of age, complained of palpitation, dizziness, vague pains in the chest, choking sensations, and a feeling that she could not get enough air at each breath. There was no history of rheumatic or scarlet fever and there had been no dyspnea or edema. Physical examination was entirely negative; the body habitus was asthenic, the blood pressure 12 /88, the heart sounds normal. There was no edema, no pulmonary congestion, no emphysema, no evidence of hypothyroidism. The teleroentgenogram was normal. The final diagnosis was psychoneurosis and neurocirculatory asthenia. The electrocardiogram is shown in Fig. 5. The upper record shows the standard limb leads and unipolar extremity potentials. Low voltage is present in all these leads and the T wave is rather flat in Lead I. The precordial leads are shown in the middle record. The RS deflection is rather small in all leads; it is largest in Lead V_4, in which it measures only 1.0 millivolt. There is inversion of the T wave in Leads V_1, V_2, and V_3. Inverted T waves are commonly found in Lead V_1 in normal persons and occasionally in Lead V_2, but after early childhood they are very uncommon in Lead V_3. The lower record shows the precordial leads after the administration of amyl nitrite. The T wave has become diphasic in Lead V_2 and upright in Lead V_3, without a conspicuous change in heart rate.

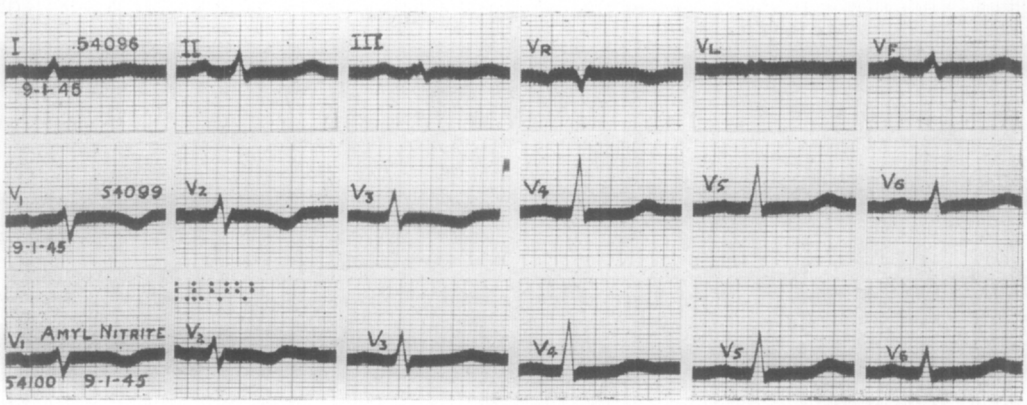

Fig. 5.— Electrocardiogram of a young woman with neurocirculatory asthenia, showing low voltage in almost all leads, flat T waves in Lead I, and inverted T waves in Leads V_2 and V_3. Lowest record shows tendency for T waves to become upright in Leads V_2 and V_3 after administration of amyl nitrite.

Inversion of the T wave in both the standard and precordial leads in cases of neurocirculatory asthenia, with reversion of the electrocardiogram to normal after the administration of ergotamine tartrate or of amyl nitrite, has been the subject of several recent studies.[30] It was of particular importance to recognize the benign nature of the T-wave changes in this case, for the combination of

low voltage and inverted T waves in the electrocardiogram of a patient with complaints referred to the cardiovascular system might have led to an erroneous and mischievous diagnosis of serious heart disease with resulting intensification of the cardiac neurosis already present.

CASE 2.—C. B., an Army medical officer, 30 years of age, was thought by his associates to be somewhat cyanotic about the lips and nail beds while he was testifying before a medical review board. Apart from the tension and anxiety natural to the situation, there were no subjective symptoms. He had always been strong and healthy, of athletic habits and physique; there was no history of rheumatic or scarlet fever or of diphtheria. There had been no undue dyspnea or chest pain, nor indeed was the cyanosis ever noted again. Physical examination and a teleroentgenogram showed no abnormalities and no further attention would have been paid to the episode had it not been for the electrocardiogram, which showed low voltage in the standard limb leads with inverted T waves in Lead I (Fig. 6, *A*). The precordial leads (Fig. 6, *B*) show normal voltage in Leads V_1, V_2, V_3, and V_4 but low voltage in Leads V_5 and V_6. The QRS complexes are of normal configuration, and the low voltage in the standard limb leads and in the leads from the left precordium, with normal voltage in the remaining precordial leads, could have resulted merely from a shift in the direction of the mean electrical axis toward the sagittal plane, in the absence of heart disease. The inverted T waves in Leads I, V_3, V_4, V_5, and V_6, however, also required explanation. Fig. 6, *C* shows the effect of exercise on these T waves in Leads V_4 and V_5. The control record was taken with the subject standing; Leads V_4 and V_5 show inverted T waves as previously. The same leads were then recorded, with the subject again in the upright position, but immediately after he had stepped briskly up and down a standard two-step stairway fifty times. In this record the T waves in Leads V_4 and V_5 are upright. Five minutes later a third record was taken, and the T waves in these leads were again both negative.

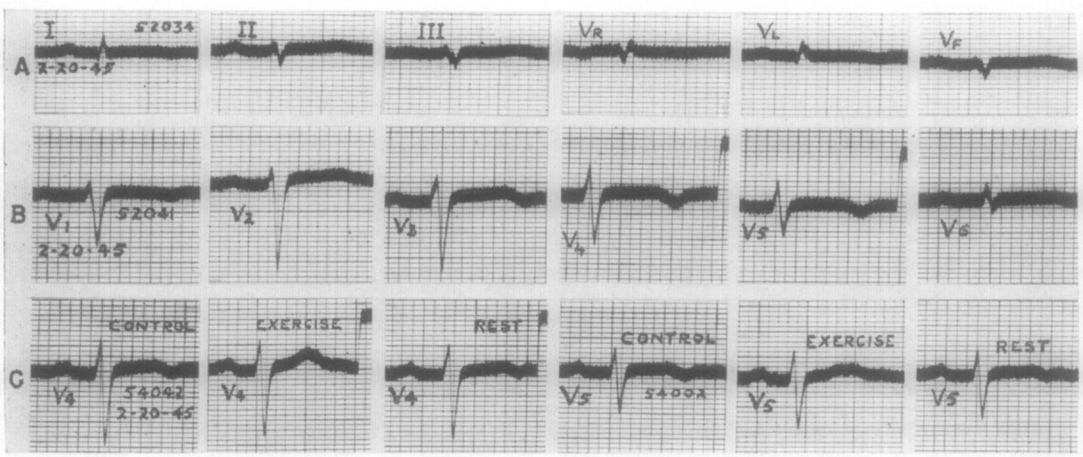

Fig. 6.—Electrocardiogram of an apparently healthy young man. *A*, Low voltage and flat T waves in the limb leads. *B*, Low voltage in Leads V_5 and V_6 and inverted T waves in Leads V_3, V_4, and V_5. *C*, Effect of exercise: T waves in Leads V_4 and V_5 become upright immediately after exercise and then revert to negativity with rest.

As in the previous case, the inconstant T-wave inversion was regarded not as a sign of disease but as a physiologic variant apparently related in some way to an unstable balance between sympathetic and vagal tonus. Whether or not this conclusion was correct is uncertain; the patient's health two years after the electrocardiographic peculiarity was first noted is still excellent.

Low Voltage of P and T Waves.—In a paper already referred to, Wilson[21] suggested that the QRS deflections might be low in the electrocardiograms of some normal persons because the electrical forces produced by one part of the heart were neutralized by those arising in other parts. In this case, he reasoned, the P wave and perhaps the T wave should be of normal amplitude. On the other hand, when the QRS deflections were diminutive because of altered tissue conductivity or myocardial degeneration, then the P and T deflections should also be small.

In this study the P and T deflections were classified as showing low voltage if both were less than 0.1 mv in amplitude in all three of the standard limb leads. In our total group of one hundred cases with low voltage of QRS in the limb leads, small P and T deflections occurred in thirty patients. Thirteen of the patients concerned had hydrothorax or hydropericardium and twelve had marked congestion of the lungs with or without peripheral edema. The remaining five had no extracellular fluid accumulations.

DISCUSSION

Several conclusions are suggested by an analysis of our cases and the comparison of our data with those collected by other investigators.

1. The electrophysiologic factors that produce low voltage in the standard limb leads tend to produce low voltage in the precordial leads also. This might be expected a priori, but certain authors[14] were led to the conclusion that, with few exceptions, no conditions other than severe myocardial disease produced low voltage in the precordial leads. Our own series, like theirs, is biased by the method of sampling used, for most of the subjects included in it must have been suspected of having myocardial disease before the electrocardiogram was ordered. It is not surprising, therefore, that most of them did have serious organic disease. Nevertheless, a certain number were found not to have heart disease and some had no organic disease of any kind.

2. It is very uncommon to find extremely low voltage in all six precordial leads even in the presence of extensive disease. Most often, when the deflections in the standard limb leads are small, those of the leads from the left side of the precordium are also small, since both reflect forces acting in the frontal plane. The forces acting in the sagittal plane may actually be very large in some of these cases, and this is suggested by the occurrence in some instances of large deflections in leads from the midprecordium, such as Leads V_2 and V_3. These sagittal forces are almost without effect upon the standard limb leads.

3. In the diagnosis of myocardial disease the configuration of the QRS deflections is of far greater significance than their size.

SUMMARY AND CONCLUSIONS

1. Low voltage occurred in one or more of precordial Leads V_2 to V_6 in sixty-five of one hundred cases selected because of the occurrence of low voltage in the limb leads.

2. Low voltage occurred in all six precordial leads in three cases only. It was most frequent in Lead V_6 (fifty-six cases) and least frequent in Lead V_2 (three cases).

3. In most instances in which certain leads showed low voltage, the size of the deflection of other leads was well within normal limits, and in many the chief deflection in the lead in which the voltage was largest was distinctly greater than the average for its kind. The largest deflection occurred most often in Lead V_2 in contrast to the normal situation in which it is most frequent in Lead V_4. This suggests that in our series the mean electrical axis, on the average, was shifted toward the sagittal plane.

4. By far the greater proportion of the patients in this series had serious heart disease. Myocardial infarction was present in thirty-five and arteriosclerotic heart disease in fifteen of the sixty-five cases. The manner in which these conditions may produce low voltage in certain leads is discussed in the text.

5. Low voltage did occur, however, in both precordial and limb leads of some patients with no intrinsic disease of the heart. Most of these had extracardiac disorders of a type that would be expected to change the electrical conductivity of the tissues surrounding the heart.

6. Low voltage also occurred in both standard limb leads and precordial leads of a small number of persons who exhibited no evidence of physical disease. In such cases it is probably the result of an unusual orientation of the anatomic and hence of the mean electrical axis of the heart.

The writer wishes to express his gratitude to Dr. Frank N. Wilson for his many valuable suggestions and for his help in the preparation of this paper.

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