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Electrocardiographic Criteria of Normality

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Address communications to: Staff Bulletin, 3330 Powell Hall, University of Minnesota, Minneapolis 14, Minn.

I. CRITERIA OF NORMALITY IN CLINICAL ELECTROCARDIOGRAPHY

Ernst Simonson, M. D.

1. Introduction

There are three steps in electrocardiographic interpretation: (1) differentiation between normal and abnormal; (2) differentiation between physiological causes such as positional effects and pathological changes; and (3) correlation to specific types of pathology.

We define as normal all electrocardiographic patterns which can be found in the majority of a normal population. In that sense, a definition of "abnormal" means unusual in a normal person, but not necessarily "pathological." For instance, dextrocardia produces an abnormal ECG without pathological involvement. The differentiation between positional and pathological effects has always been, and still is, a major problem in electrocardiography; but the present topic is concerned with the first step, the differentiation between "normal" and "abnormal" which is, perhaps, the most important decision in the routine of clinical interpretation.

It may be useful, before presenting our material, to discuss briefly some general concepts in the definition of normality. If we have a large sample of normal population and a large sample of patients, there will be a certain overlapping of the distribution in any electrocardiographic or other item which can be measured.

TABLE 1

Overlapping distribution of normal population and of patients

Amplitude mm. lower than	Percentage of Population Normals	Patients
1.5	20	1
1.0	10	3
0.5	1	6
0.0	0.5	12
-0.5	0.05	25

This is illustrated in Table 1, which is, however, not based on actual figures. If the item shown in the table is smaller than 0.5, it will occur only in a small minority of normal people, in our example, 1 per cent. If the item decreases to zero, it will be found in only 0.5 per cent of normal people, and will be extremely rare if negative. On the other hand, 6 per cent of patients will have amplitudes larger than 0.5 mm. in our example. Therefore, any arbitrary dividing line, defined as normal limits, will include a certain percentage of abnormal population. If we set the normal limit at -0.5 in our example, the chance that lower, i.e. more negative values, will occur in a noraml person is only 1:5000 which seems to be very satisfactory for assurance of a positive diagnosis. However, such limit would be impractical because every fourth patient could not be diagnosed as abnormal. If we set the limit at 1.0, 10 per cent of normal population would be classified as abnormal, and this also would be impractical for clinical diagnosis. Actually this has happened with the normal range limits for low voltage of the QRS complex and the T wave, as can be found in many textbooks, or the more recent data by Sokolow and Friedlander[1] for the precordial leads.

It is apparent then that the so-called "normal limits" must necessarily be placed at an arbitrary point. It has been customary to place the boundaries of normality at a point which will include 98 per cent of the population. This will mean that on one occasion in 100 a normal individual will be classed as abnormal. It also means that a definite number of patients with cardiac pathology will be classed as normal, but statements as to the probability of this occurrence cannot be made at the moment. In most cases the number of cardiacs who will be classed as normal will be larger than the number of normals who will be classed as abnormal. As an example, in patients with clinical angina pectoris the electrocardiogram may be normal for several years. It is wise to set the normal limits in this way since every

effort should be made to avoid the creation of cardiac neurosis in normal individuals. Our tables, therefore, refer to the 98 per cent range limits, but narrower or wider limits could be easily calculated from the standard deviation or percentile distribution.

It is clear that the separation between patients and normals will be the better, the less the overlapping. Any improvement of procedure -- technical, biological, or statistical -- which will reduce the normal variability, will reduce the overlapping and thus improve the reliability of prediction.

It has been a rather common misunderstanding in electrocardiography, that experience in a large clinical material can replace a proper normal standard material. Actually, the reverse is the case, the definition of abnormality depends only on the evaluation of a normal population. Unfortunately, the normal electrocardiographic standard material has been inadequate both in regard to size and/or statistical evaluation for nearly three decades, since electrocardiography was introduced as a clinical method. It is only in the last ten years that larger samples have been collected. In 1942, Viscidi and Geiger[2] found that 40 per cent in a sample of 500 normal working population had electrocardiograms which exceeded the normal limits such as frequently used in clinical routine. This can only mean that these normal standards are absurd. While these authors criticized the conventional normal standards, they failed to provide corrections from their material. Graybiel et al.[3] studied a large normal material of 1000 young pilots. The standard deviations were not calculated in the original communication, but Drs. Graybiel and McFarland were kind enough to send us their material for calculation of the standard deviations. Although their sample was undoubtedly preselected in regard to physical fitness, compared to average and even more so to hospital population, it was found that the conventional normal range limits were too narrow for some items.

Without going into any further detail it may be summarized that, prior to our investigations, adequate data for normal young men and children[4, 5] but not for older men and women or for precordial leads or unipolar limb leads were available. We found that about 10 per cent of healthy, normal men in our experimental group would have to be classified as ventricular preponderance on the basis of Sokolow and Friedlander's normal limits for the precordial leads[1]. The same authors gave normal standards for aVR, aVL and aVF, but failed to consider the electrical heart position, which makes these standards nearly meaningless.

Our main purpose, to improve the normal standards by elimination or reduction of sources of normal variability, can be approached in two separate ways: improvement of the standardization of method and procedure, and analytical breakdown of constitutional variables. We have worked in both directions, and in addition provided normal standards for the conventional precordial leads V_1 to V_6, for nine additional leads around the chest at the fifth intercostal level, and for the Wilson unipolar limb leads. This material is quite large, so that only a brief summary can be given.

2. Standardization of Procedure

Wilson et al.[6] suggested, in 1934, that a junction of the three limb electrodes RA, LA and LL each with 5000 Ohm resistors be used as an indifferent reference electrode. This was a very definite imporvement and the closest approximation to a neutral reference electrode obtainable with comparatively simple means. Goldberger[7] suggested a two lead terminal to replace Wilson's three lead terminal, in order to obtain larger amplitudes in the unipolar limb leads VR, VL, and VF. Goldberger's terminal electrode is far from neutral, but he predicted that the augmentation ratio is constant, i.e. his augmented leads aVR (augmented VR), aVL, and aVF would equal 150 per cent of VR, VL and VF, or, ex-

pressed as equation: $\frac{aVR}{VR}$, $\frac{aVL}{VL}$ or $\frac{aVF}{VF}$ x 100 = 150. Goldberger offered no experimental proof for this prediction. We tested[8] this claim (Table 2) and found that the means of the ratio of 26 normal subjects are reasonably close to the prediction, but that the variability is so large that the augmentation could be anywhere between 100, i.e. no augmentation, and 300. Also, the augmentation between different leads in an average person was found to be highly variable; for instance, the augmentation of aVR might equal from 1/4 to nearly 2 times that of aVF. This means that the use of Goldberger's augmented leads introduces an uncontrolled and rather large source of variability, which will tend to make differentiation between normals and patients less accurate. Finally the greater amplitude which is the only advantage of the aV-leads could be easily attained by increasing the sensitivity of the galvanometer.

TABLE 2

Augmentation of amplitudes in the Goldberger leads (aV) expressed as a percentage of the Wilson leads

	$\frac{aV_R}{V_R}$ x 100		$\frac{aV_L}{V_L}$ x 100		$\frac{aV_F}{V_F}$ x 100	
	QRS	T	QRS	T	QRS	T
Mean (26 subjects)	156	147	165	157	184	187
S.D.±	29.8	48.3	41.4	39.0	77.0	62.7
Expected upper limit*	205	220	233	221	311	290
Expected lower limit**	107	100**	100**	100**	100**	100**
Number of values	26	24	22	13	25	15

* The expected range refers to 90 per cent of the population.

** The lower limit of 100 is assumed where, according to the variability, the aV leads may show a smaller amplitude than the V leads.

In clinical routine electrocardiography, appointments are usually made at any time of the day, irrespective of the intake of meals. In a well controlled series of 12 young men[9] statistically significant changes after a meal were found for most electrocardiographic items, and this was confirmed in later series on 42 middle-aged men and 12 women[10]. In these series, a total of 36 items were measured and statistically evaluated. The changes after the meal were independent of the composition of the meal or of the calorie content which varied between 900 and 1500 Cal. (which approximates an ordinary lunch), and showed no significant decline from 30 to 60 minutes after the meal. The total duration of the meal changes was not determined. The changes after a meal were even greater in patients. In many cases, a normal ECG before the meal became abnormal after the meal, but also opposite responses were observed, for instance in first degree AV-block, which disappeared or diminished in four out of five cases (Table 3).

In some cases, the changes were so marked that the ECG before and after the meal did not seem to be from the same patient. The response to a meal was utilized as a tolerance test for

TABLE 3

Electrocardiographic changes after meal in patients with partial A-V block

Patient No.	Sex	Age	Group	P-R interval (sec.) Before	After meal
265	M	52	IV	0.24	0.20
495	M	47	VI	0.23	0.19
318	M	54	VI	0.29	0.25
496	M	47	VI	0.25	0.20
477	M	47	VI	0.23	0.23

detection of latent coronary insufficiency which will be discussed in somewhat greater detail later; presently it may suffice to point out that in ordinary routine electrocardiography the effects of the previous meal constitute a major uncontrolled factor. It seems likely that this was not considered in the collection of the normal standard material, so that the normal range might be wider, and consequently less precise, than need be. It is suggested, therefore, to take the ECG either before breakfast, or at least two hours after a light meal. For several years, all ECG's are taken in basal condition in this Laboratory.

3. Age and Weight Trends

The normal standards as used in present clinical electrocardiography do not provide for any age trends above 20 years or consideration of the body weight. The only item where the body weight has been taken into consideration is the electrical heart position or axis, but even here no precise data were available, and the same is true for the age trends.

We compared 40 electrocardiographic items in 157 young men (between 18 and 25 years), and 233 older men (between 45 and 55 years). Each age group was subdivided into five weight groups with reference to the normal standard weight as determined from height and age: sub-group A, marked underweight, less than 85 per cent standard weight; sub-group B, moderate underweight, between 85 and 96 per cent standard weight; sub-group C, normal weight, from 95 to 105 per cent standard; sub-group D, moderate overweight, from 106 to 115 per cent standard; and sub-group E, marked overweight, above 115 per cent standard weight. Our subjects were very carefully screened as to absence of disease; in addition to a clinical examination, the tolerance to several physiological stress situation was studied.

TABLE 4

Means (M) and standard deviations (S.D.) of several electrocardiographic items in normal young men. Comparison of two different samples.

	Minnesota Group 157 (18-25 years)		Graybiel Group 1000 (20-30 years)	
	Mean	S.D.	Mean	S.D.
R_1	4.9	2.42	5.9	2.57
R_2	11.6	3.63	11.6	3.88
R_3	7.9	4.35	7.10	4.20
QRS axis	66.7	21.3	64.2	23.3
T_1	2.5	0.91	2.5	0.88
T_2	3.2	1.13	3.3	1.18
T_3	0.9	1.22	1.20	1.19

The younger age group was also compared with Graybiel's et at.[3] large group of 1000 pilots. Table 4 shows the excellent agreement both in regard to means as well as to standard deviations. Since the groups were from different regions, and collected about eight years apart, there is confidence that the values in our younger group have general validity. Unfortunately, no other middle-age group was available for a similar comparison, but it might be expected that the values are applicable to average normal population at least in this area, and probably also in other regions of this country.

TABLE 5

Statistically significant mean differences (ΔM) of ECG items between 157 young and 233 older men.
t (0.01) = 2.62; t (0.001) = 3.37.

Item	ΔM	t	Item	ΔM	t
R_1	+ 1.2	4.7	T_1	-0.5	4.8
R_2	- 3.9	10.8	T_2	-1.0	8.6
R_3	- 4.3	10.6	T_3	-0.7	5.4
S_1	- 0.5	4.9	ΣT	-1.9	7.5
S_2	- 0.3	2.9	T-CF_4	-1.2	4.7
S_3	+ 0.9	8.3	T-axis	-9.9	4.3
ΣQRS	- 7.5	9.5	P-R int.	+0.7	3.2
QRS-axis	-29.9°	10.2	K_{QT}	+0.01	3.9
			Δ R-R	-5.1	5.7

(Amplitudes in mm. (= 0.1 mv): axis in degrees)

The investigation revealed statistically highly significant differences in the great majority of electrocardiographic items as partially listed in Table 5. Each age group of Table 5 included all weight groups, in nearly the same proportion. It could be assumed, therefore, that the effect of weight was cancelled out, and this was actually shown in the comparison of the normal weight groups C.

Forty-nine younger men and 66 older men were in weight group C, i.e. within ±5 per cent of the standard weight. The mean age differences in the various items in the total groups and in the partial weight groups C were practically identical; for instance, the mean amplitude of R_2 was 11.61 and 7.75 with a mean difference of -3.86 for the total younger and older age group, and 11.97 and 7.58 with a mean difference of -4.39 for the partial weight groups C. The age differences, therefore, are not concealed effects of body weight. One still might object that the normal weight increases with age, so that in a comparison of different age groups of the same standard weight the older group is actually somewhat heavier. For this reason, the older weight group B was compared with the younger weight group C; the mean absolute body weight was nearly identical, but the electrocardiographic age trends were essentially the same as in the total groups.

In general, the effect of age was in the direction of abnormality; lower voltage of all deflections, prolongation of the P-R interval, and relative left ventricular preponderance. It might be expected, therefore, that a larger percentage of older normal men would fall outside the conventional normal range limits, i.e. diagnosed as abnormal, and this is actually shown in Table 6.

TABLE 6
Percentage of normal men exceeding accepted normal limits

Item	Limit	Percentage Younger Men	Percentage Older Men
P-R	0.21"	0.6	3.4
ΣQRS	15.0 mm.	6.4	11.2
Q_3	25% max. R.	3.2	9.0
$ST_{1,2}$	-0.5 mm.	2.5	7.3

The results were essentially the same when more conservative or more liberal screening levels were used, so far as the ratio between "abnormal" younger and "abnormal" older men is concerned. At the same time, Table 6 shows that the presently used normal standards are derived mainly from younger people, and that their use for older normal population will lead to diagnostic errors. This explains also Viscidi and Geiger's results[1] referred to in an earlier part of this paper. Age trends toward electrocardiographic abnormality are probably due to gradual, slowly progressive myocardial degeneration, and are an excellent correlate to the results of Clawson[11], obtained in a large autopsy material, and those of White et al.[12]. It seems that a certain degree of slow myocardial degeneration is a normal ageing process, and these age changes should be considered in electrocardiographic normal standards.

In the material discussed so far a possible effect of weight was balanced in the comparison of the total weight groups or eliminated in the comparison of partial weight groups. This does not mean, of course, that there are no weight trends. On the contrary, statistically significant weight trends were found in both age groups, but much more so in the older age group. In the young group continuous, significant, age trends were observed in four items in the standard leads and CF_4, and in twelve items in the older group. Several items in the younger group showed some continuous weight trends,, similar to those in older men, but they were too slight to attain statistical significance.

TABLE 7

Means, standard deviations (S.D.), differences (Δ) between the means of the extreme weight groups A and E, and slope with body weight for several electrocardiographic items in young and older men. Statistical significance: * = P 0.05; ** = P 0.01; *** = P 0.001. Amplitudes in 0.1 mv., axes in degrees.

Item	Group A Mean	Group A S.D.	Group E Mean	Group E S.D.	Δ	(slope)
			Young Men			
R_1	3.80	1.82	6.55	3.08	2.75**	0.053***
T_3	1.84	1.08	0.31	1.25	-1.53***	-0.033***
T axis	62.00	10.88	31.87	21.77	-30.13***	-0.576***
			Older Men			
R_1	3.86	2.41	7.44	2.57	3.58***	0.090***
R_2	8.95	3.24	6.59	2.47	-2.36**	-0.965***
T_2	2.80	0.88	1.65	0.76	-1.15***	-0.029***
T_3	1.03	1.01	-0.63	0.66	-1.66**	-0.041***
QRS axis	61.61	24.24	31.42	8.06	-30.19***	-0.787***
T axis	51.77	17.50	11.12	19.15	-40.65***	-0.941***
CF_4 - T	5.99	2.22	4.03	1.50	-1.96***	-0.036*

Table 7 shows the effect of body weight in a few items selected as typical examples in terms of mean differences between the extreme weight groups A and E, and of the slope, which was calculated for the total age groups. A positive slope means an increase, and a negative slope means a decrease of the value per unit (per cent) standard weight. In regard to the axes, "minus" means shift to the left. The mean differences as well as the slopes are significantly larger in the older group.

It is of interest that weight is a more important factor in older than in younger men. At the same time it shows that the effect of weight cannot be

interpreted as positional effect alone. It may also be mentioned, that age and weight trends were not identical in many respects.

The presence of statistically significant age and weight trends makes a correction for these constitutional variables for normal electrocardiographic standards necessary. It may be expected that the age and weight corrected standards, as presented in Tables 8 and 9, will contribute to reduce the overlapping of patients and normals, and thus aid in the differentiation between normal and abnormal.

TABLE 8

Expected range limits for 98 per cent of normal population, calculated from the percentile distribution.

Group		R_1	R_2	R_3	S_1	S_2	S_3	Σ QRS	QRS Axis
Younger Men	upper	11.72	21.43	18.02	4.57	5.29	3.51	51.65	99.56
	lower	1.10	5.19	0.00	0.17	0.01	0.00	12.62	-14.30
Older Men	upper	13.42	18.67	11.21	4.67	4.27	6.66	41.63	93.35
	lower	1.29	1.58	0.00	0.01	0.01	0.00	10.20	-28.9

Group		T_1	T_2	T_3	ΣT	$T\text{-}CF_4$	T Axis	P-R Int.	K_{QT}
Younger Men	upper	4.91	6.22	3.73	11.43	13.47	79.3	22.21	.462
	lower	0.00	0.39	-1.95	3.08	1.77	-21.15	11.11	.338
Older Men	upper	4.67	4.81	2.88	9.67	10.94	79.45	21.92	.458
	lower	0.23	0.55	-2.44	1.58	1.05	-14.25	12.13	.351

Table 8 shows the expected age corrected range limits for young and middle-aged men, as calculated from the percentile distribution, for the standard leads and CF_4. It is expected that V_4 will show a similar age trend of the T-wave as CF_4. Items not included do not show significant differences with age. Table 9 shows the age corrected limits for the unipolar limb leads. The vertical (V) and semi-vertical (SV), as well as the horizontal (H) and semi-horizontal (SH) positions are combined. It may be mentioned that this is the first normal standard material for the VR, VL, VF leads available, differentiated according to the electrical heart position.

TABLE 9

Expected normal limits (U = upper, L = lower) for unipolar limb leads. Calculated from the percentile distribution for 98 per cent normal population; younger men (Y) 18 to 25 years; older men (O) 45 to 55 years. The intervals (1/100 sec.) and amplitudes (standardized mm, 1 mm = 0.1 mv) are rounded to 0.5 units. The Q-wave refers to left ventricular surface leads, the QS deflection to VR. Symbols used for heart position: V = vertical; SV = semivertical; I = intermediate; H = horizontal; SH = semihorizontal.

			Amplitudes							
Heart Position	Age Group	Lead	P		Q		R		QS or S	
			U	L	U	L	U	L	U	L
All	Y	VR	-1.0	0.0	-	-	2.0	0.0	-11.5	-2.5
	O	VR	-1.0	0.0	-	-	1.5	0.0	-9.0	-1.0
V + SV	Y	VL	+0.5	-0.5	-	-	2.5	0.0	-9.7	-0.0
	O	VL	+0.5	-0.5	-	--	2.5	0.0	-7.0	0.0
	Y	VF	+1.0	0.0	-1.5	0.0	13.0	1.5	-2.0	0.0
	O	VF	+1.5	0.0	-1.0	0.0	11.5	1.0	-1.5	0.0
I	O	VL	+0.5	-0.5	-1.5	0.0	6.0	0.5	-2.0	0.0
	O	VF	+1.2	0.0	-1.0	0.0	9.0	1.0	-1.5	0.0
H + SH	O	VL	+0.5	-0.5	-2.0	0.0	7.5	1.0	-1.5	0.0
	O	VF	+1.0	0.0	-0.5	0.0	2.5	0.0	-3.0	0.0

			Amplitudes				Intervals			
			ST		T		QR		QRS	
			U	L	U	L	U	L	U	L
All	Y	VR	-1.0	0.0	-4.5	-0.5	9.0	3.0	12.5	6.0
	O	VR	+0.5	-0.5	-3.5	0.0	8.0	4.0	12.0	5.0
V + SV	Y	VL	+0.5	-0.5	+2.0	-2.5	9.0	2.0	13.0	6.0
	O	VL	+0.5	-0.5	+1.5	-2.5	8.5	2.0	12.0	5.0
	Y	VF	+0.5	-1.0	3.0	0.0	7.0	2.0	6.5	2.0
	O	VF	+0.5	-1.0	3.5	-0.5	12.5	6.0	12.5	5.5
I	O	VL	+0.5	-0.5	+2.5	-0.5	8.0	2.0	12.5	5.0
	O	VF	+0.5	-0.5	+2.8	0.0	7.5	2.0	12.5	5.0
H + SH	O	VL	+0.5	-0.5	2.5	0.0	7.0	3.0	13.0	5.5
	O	VF	+0.5	-0.5	1.5	-0.5	8.5	2.0	12.0	5.0

Table 10 gives the weight-corrected standards for each age span, for three weight categories: normal weight, definite underweight, and definite overweight. For the use of these standards, only an approximate estimate of the body weight is necessary, such as a crude classification of lean, normal, and

TABLE 10

Upper (U) and lower (L) limits for 98 per cent for underweight (A: below 86 per cent of normal weight), normal weight (C: 95 to 105 per cent of normal weight) and overweight (E: above 114 per cent normal weight) normal men, calculated from the standard deviation. Standard leads and CF_4.

Weight Group	A		C		E	
Item	U	L	U	L	U	L
			Young Men			
R_1	8.04	0.00	9.92	0.00	13.73	0.00
T_1	3.75	0.77	4.25	0.71	5.17	0.61
T_3	4.36	-0.67	3.30	-1.46	3.22	-2.60
T-axis	87.35	36.35	80.27	5.85	82.59	-18.85
QRS-axis	115.62	24.98	110.23	25.56	108.03	11.57
Δ R-R	21.54	0.00	18.90	3.70	40.48	0.00
QRS-int.	10.40	6.26	12.07	5.07	12.70	4.64
R_2	18.22	3.54	19.28	5.12	16.97	4.35
R_3	18.20	0.00	17.34	0.00	13.73	0.00
			Older Men			
R_1	9.47	0.00	12.93	1.09	13.45	1.45
R_2	16.50	1.40	14.48	0.68	12.35	0.83
R_3	12.78	0.00	9.11	0.00	8.57	0.00
S_3	5.15	0.00	7.48	0.00	6.96	0.00
T_2	4.85	0.75	4.04	0.18	3.42	-0.12
T_3	3.38	-1.32	2.69	-2.91	0.91	-2.17
rT	9.68	1.80	9.02	1.38	8.09	1.01
QRS-axis	118.09	5.13	103.04	-33.60	96.38	-33.54
T-axis	92.31	11.23	66.50	-18.32	55.74	-33.50
R-CF_4	18.90	0.00	21.32	0.00	23.27	0.00
T-CF_4	11.16	0.82	9.00	1.46	7.52	0.53
S_2	3.40	0.00	2.88	0.00	2.08	0.00
T_1	3.59	-0.27	4.07	0.53	3.81	0.37

obese. Table 10 includes only those items where consideration of body weight is of importance; for the other items, Tables 8 and 9 are to be used. It is regrettable that no data on women are yet available. We suggest, however, that the present data may be used also for women until specific information can be provided.

4. Day to Day Variability

Although serial electrocardiograms have become clinical routine for a long time, it is surprising that no adequate data on normal day to day variability were available. Knowledge of the normal day to day variability is important for any conclusion whether electrocardiographic changes in the same patient are due to chance variability or to a change of the condition of the heart. Thus, abnormally large changes, exceeding the expected normal limits, could serve as a criterion of abnormality, whether the changes are within or outside the wide

normal range limits.

We studied day to day variability in 12 normal young men[13] who were investigated two or three times a week for a period of about three months. Taken all in all, 11 repeats were made. Thirty-five electrocardiographic items were measured in three standard leads and CF_1, CF_2 and CF_4. There is every reason to believe that the results are valid also for V_1, V_2 and V_4. The location for the placement of the chest electrodes was marked by intracutaneous dye injection. The expected normal limits of variability were calculated from the intra-individual standard deviation and are shown for some items in Table 11.

TABLE 11

Normal limits of day to day variability of some electrocardiographic items compared to variations in a patient.

Item	Mean	Limits	Item	Mean	Limits	Patient D. B. I/11	I/14	I/24
P-R 1/100 sec.	14.7	±1.40	S-CF_1(V_1)	16.7	±2.2	8.5	7.0	8.2
R_2 mm.	12.8	±1.1	S-CF_2(V_2)	24.9	±3.7	4.5	2.0	4.0
S-T_2 mm.	0.1	±0.3	R-CF_4(V_4)	11.9	±2.6	18.5	19.5	17.5
T_2 mm.	3.8	±0.7	T-CF_1(V_1)	-3.1	±0.9	1.5	2.5	1.0
QRS axis°	69.2	±10.8	T-CF_2(V_2)	6.8	±1.4	4.8	4.0	2.8
T axis°	40.0	±16.4	T-CF_4(V_4)	7.3	±1.4	11.2	7.5	6.5

Table 11 shows also the variations in V_1, V_2 and V_4 of a patient (D. B.) on three occasions. The standard leads as well as the chest leads were within normal limits on all three days. The variations of the QRS complex were within normal limits of variability, but the decrease of the T-wave exceeded considerably the normal limits of variability, especially in V_4. Although each ECG was within normal limits, the variability was considered to be abnormal and tentatively interpreted as coronary insufficiency of the posterior wall. This was done independently from clinical information. Actually the patient*, a 63 year old woman, had had a typical attack of coronary insufficiency, which was so severe that myocardial infarct was suspected. Since, as a rule, the locations for the chest electrodes are not permanently marked in patients, a somewhat greater variability than in our experimental series can be expected. However, the normal variability of the QRS complex and the fact that the T-wave changes occurred in all precordial leads excludes different electrode position as an important factor in this case.

However, the placement of chest electrodes is a disturbing and uncontrolled source of variability. In order to eliminate it at least in hospitalized patients, it was suggested for the Veterans Hospital and the Mount Sinai Hospital to use idelible ink for the marks which should be renewed by the nurse in the ward whenever they become faint.

5. Standards for Chest Leads

We mentioned already that the present standards for the chest leads are not adequate for several reasons. Recently, it has been suggested that additional chest leads be taken farther right from V_1 or left from V_6 for diagnosis of right ventricular preponderance or coronary insufficiency (14 to 18). No standards for such additional leads were ever provided, so that the patterns found in

* I wish to thank Dr. P. Gordon, Mount Sinai Hospital, for his kind permission to use this case.

other leads such as V_1, VR, V_6 or VF were used as a criterion also for the additional leads.

We studied the regional pattern distribution as well as the amplitudes of the major deflections in 15 leads around the chest at the level of the fourth and fifth intercostal space, following electrocardiographic routine. For the definition of patterns, we followed accepted clinical terminology [7, 19]. The material was subdivided into the following nine patterns: rS, T-; rS, T-iso; rS, T+; RS, T+; (q)R, T+; (q)R, T-iso; qR, T-; QR, T-; Qr, T- (or rSr', T-).

For the analysis of the regional distribution of patterns around the chest, a heart center was carefully determined by Dr. C. Chapman in measurements of 72 chest plates. Although the definition was arbitrary to a certain extent, the symmetrical arrangement of the patterns which was found with respect to the center proved that the position of the center must have been quite accurate.

Thus, the location of the chest leads could be expressed in terms of angles from the center, for instance $V_1 = 125^\circ$, $V_2 = 82^\circ$, $V_3 = 63^\circ$, etc., with a transversal line through the center taken as 0° (left side) and 180° (right side). This provided the possibility of defining the location of each pattern in terms of its midpoint, endpoints, and extent (zone). The normal limits for each of these items were calculated from the standard deviations. In addition, the regional trends of the amplitudes of the various deflections were statistically analysed. The experimental group, 103 normal middle-aged men, was subdivided into five positional groups (V = vertical, SV, = semi-vertical, I = intermediate, SH = semi-horizontal, H = horizontal) from the unipolar limb leads, using Wilson's et al.[19] and Myer's et al.[20] criteria. The statistical analysis was made separately for each group. Taken all in all, 600 items were statistically analysed, which is probably the largest electrocardiographic material yet analysed. The analysis took well over one year, and the tabulated results were so voluminous as to be not manageable for publication, so that a substantial part will be deposited in the American Institute of Documentation. It is, therefore, possible only to discuss some points of more general interest.

There is a continuous trend from one pattern to the other, so that any definition of patterns is arbitrary. In a general way, there is a mirror-like arrangement of patterns around the chest, for instance the qR, T+ pattern on the left side of the chest corresponds to the rS, T- pattern on the right side of the chest, or the RS, T+ pattern usually found between V_2 and V_4 corresponds to a QR, T- pattern on the back. The geometrical range of most patterns is quite wide and there is a large degree of overlap. It was impossible, in a substantial proportion of our material, to relate consistently the pattern distribution to local patterns assumed to be produced by specific parts of the heart. As a whole, the results do not support the so-called "unipolar electrocardiographic theory" based on synthesis of local patterns as a sound basis of electrocardiographic interpretation. The electrical position in the frontal plane affects the pattern distribution in the horizontal plane. The distribution of the qR or QR, T- pattern is of some diagnostic consequence because this is usually interpreted as right ventricular preponderance when found in V4R to V6R, and as coronary insufficiency or infarct when found in V7 to V9. The normal midpoint of these patterns is on the back, but it may extend normally to V5R or to V7, simulating right ventricular preponderance or coronary insufficiency, respectively. There is little coubt that such misinterpretations have been made in the absence of normal standards.

6. Tolerance Tests

While the new normal standard material

as corrected to age and weight, and enlarged as to the number of leads, will contribute to a better differentiation between normals and patients, still a certain proportion of patients will fall into the normal range limits. This is especially true for the initial phase of coronary artery disease. Even in patients with clinical angina pectoris, the ECG may remain within the normal limits for several years. The coronary blood supply may be sufficient for resting conditions, but becomes inadequate for superimposed physiological stress, resulting in angina pectoris attacks.

If patients can be investigated during typical angina episodes, or very shortly after, pronounced ST- depressions will be found. However, these changes persist only for a few minutes, as a rule, and therefore escape recognition until the pathology is more advanced. For about two decades, several types of superimposed physiological stress have been used for the diagnosis of latent coronary insufficiency: exercise[21], anoxia[22], and ergonovine[23] or related drugs. In none of these tests the existing normal standard material can be considered as adequate, and this is especially true for the so-called Master's two step test. In view of the absence of valid standards, the interpretation of these tolerance tests has become quite arbitrary, although it is not doubted that a definitely abnormal response can be diagnosed.

One of the objections against the three types of tolerance tests is the poor definition of the physiological load. The two-step test is actually quite strenuous work, but of so short duration that no steady state is attained. Therefore, the physiological load in the two-step test will be quite variable from patient to patient. Breathing 10 per cent oxygen mixtures does not produce a constant depression of arterial oxygen saturation; and in drug tests the individual sensitivity to drugs is a complicating factor. None of these tests is without risk; it is, indeed, curious to observe that the proponents of one type of these tolerance tests claims safety for this particular test and a definite risk for the other types.

We have been using as exercise tolerance tests walking on a treadmill at moderate speed and grade, for a duration of ten minutes, at the Veterans Hospital for several years. This type of exercise is much safer, since it is moderate and similar to the ordinary activity, and the physiological load can be quite accurately predicted from body weight, speed and grade[24]. Another treadmill of the same type is being built now for the University Heart Hospital. At the present time, we are collecting at our Laboratory a large normal standard material. The ECG is taken before, immediately after (about 30 sec.), and three minutes after the exercise.

The accumulation of the material is still in progress and no normal standards are yet available. However, several observations favor a more conservative interpretation than has been previously suggested. We found ST- depressions of 1 mm. or even somewhat larger quite common in healthy middle-aged men. In addition, we found also that changes of the QRS- complex are quite frequent. This is of interest, because only changes of the ST segment and the T wave are diagnostically evaluated in the present routine of tolerance tests. The occurrence of QRS changes in transient coronary insufficiency, produced by moderate exercise, is also of interest in view of the current practice of differentiating between myocardial infarct and coronary insufficiency by the presence or absence of QRS- changes. It is currently assumed that coronary insufficiency does not produce changes of the QRS complex. If transient coronary insufficiency produced by moderate exercise in normal men may change the QRS complex, the validity of this concept is questionable. It is understood that we are not suggesting that a large Q-wave may be produced by transient coronary insufficiency.

We utilized the electrocardiographic changes after the meal, which we discussed briefly in an earlier part of this paper, as a basis for a tolerance test. The underlying physiological mechanism is quite different from that involved in exercise or anoxia; the increase of the blood flow is only slight, and the arterial oxygen saturation is not changed. Probably, the changes are of a reflex nature, and the abnormal response to meal in patients would indicate an exaggerated reflex irritability. Recently, evidence for increased reflex irritability in patients with coronary disease has been reported[25]. It may be mentioned, that probably even after exercise a part of the changes are of reflex origin since an abnormal exercise tolerance test in patients can be converted to normal by ergotamine[26]. Whether this response can be utilized to differentiate between latent coronary insufficiency and emotional effects, as suggested by Master et al.[26], is still debatable.

An advantage of the meal test is its safety, in clinical as well as in legal aspects, and its simplicity. The ECG is first taken before breakfast, or before lunch at least three hours after a light breakfast. The calorie content may vary between 900 and 1500 cal., and the composition of the meal is of no consequence. The ECG is repeated about 30 minutes after the meal, but the time is not critical; the changes can be observed from 20 to 60 minutes after the meal.

The normal limits of the response to meal were calculated from the data of 12 young men, 42 middle-aged men and 12 middle-aged women[9, 10]. Table 12 gives the range limits for 98 per cent of normal population of a few items which frequently were found abnormal in patients.

TABLE 12

Normal range limits of changes of some electrocardiographic items after a meal. "+" means an increase and "-" means a decrease compared to the pre-meal value.

Item	Middle-aged Men		Middle-aged Women	
	from	to	from	to
Heart Rate	+6.2	+16.0	-5.4	+13.5
R_2	-1.6	+5.6	-1.1	+4.3
ST_2	-1.0	+0.6	-1.0	+0.6
T_1	-2.3	+0.6	-1.5	+0.6
T_2	-2.4	+0.6	-1.8	+0.2
$T\text{-}V_4$	-5.6	+0.5	-2.5	+0.3
QRS-axis°	-24	+35	-19	+32
T-axis°	-17	+23	-51	+36

These standards for a normal response were used as a criterion for analysis of the response in 99 patients, subdivided into the following categories: (I) suspected coronary insufficiency; (II) coronary insufficiency; (III) arterial hypertension; (IV) miscellaneous cardiac pathology other than coronary insufficiency or hypertension; (V) patients with borderline ECG, but clinically normal; (VI) patients with abnormal ECG, but clinically normal[27].

TABLE 13

Incidence of abnormal electrocardiographic changes after meal in six clinical groups

Clin. Categ.	No. Patients Tot.	Abn. Resp.	$T_{1,2}$, CF_4, V_4 Decr.	Incr.	T Axis	S-T Dep.	Heart Rate -	0	+	Other Changes	Abn. Items per Pat.
I	16	12	10	0	0	3	2	3	1	2	1.75
II	23	19	14	7	0	3	5	3	2	4	1.89
III	21	16	10	3	5	3	2	2	4	1	1.88
IV	6	5	3	0	1	0	1	0	2	1	1.60
V	10	5	1	1	0	2	1	0	3	0	1.60
VI	23	18	8	2	3	0	2	1	4	6	1.45
Total	99	75	46	13	9	11	13	9	16	14	--

Table 13 summarizes the results. The high incidence of abnormal responses is encouraging for the diagnostic use. In most patients more than one item was found to be abnormal. Abnormal changes of the T-waves were more important than changes of the ST- segment. In patients with subjective discomfort after the meal the response was always abnormal, but only a very small minority of patients (about 5 per cent) experienced discomfort. The abnormal response, therefore, does not depend on subjective discomfort.

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II. MEDICAL SCHOOL NEWS

Coming Events

Mar. 18 Minnesota Pathological Society Meeting; "Studies on Breast Cancer Patients Receiving Estrogen Therapy," Dr. Robert A. Huseby; Owre Amphitheater; 8:00 p.m.
Mar. 20 E. Starr Judd Lectureship in Surgery; "Some Observations on the Treatment of Carcinoma of the Pancreas," Dr. Thomas G. Orr, Professor of Surgery, University of Kansas; Owre Amphitheater; 8:15 p.m.
Mar. 24-26 Continuation Course in Therapeutics for General Physicians
Apr. 7-9 Continuation Course in Surgery for General Physicians
Apr. 8 George E. Fahr Lectureship; "Coarctation of the Aorta," Dr. Robert E. Gross, Ladd Professor of Children's Surgery, Harvard Medical School, and Surgeon-in-Chief; Children's Hospital, Boston; Owre Amphitheater; 8:15 p.m.
Apr. 14-19 Continuation Course in Proctology for General Physicians
Apr. 17-19 Continuation Course in Obstetrics for Specialists
Apr. 21-23 Continuation Course in Pediatrics for Specialists

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Continuation Course in Surgery

A continuation course in Surgery will be held at the Center for Continuation Study from April 7 to 9, 1952. The course is intended primarily for physicians engaged in general practice and will deal largely although not exclusively with pediatric surgery. We are very fortunate that Dr. Robert E. Gross, Ladd Professor of Children's Surgery, Harvard Medical School, and Surgeon-in-Chief, Children's Hospital, Boston, will join us as a member of the faculty for the course. Dr. Gross will also deliver the Annual George E. Fahr Lecture on Tuesday, April 8, at 8:15 p.m. in Owre Amphitheater. The subject of the Fahr Lecture will be "Coarctation of the Aorta."

Faculty News

Dr. Leo G. Rigler, Professor and Head, Department of Radiology and Physical Medicine, was the recent guest lecturer for the Los Angeles Western Conference on Radiology. On February 23 he discussed "The Possibilities and Limitations of Roentgen Diagnosis," and "Early Diagnosis of Carcinoma of the Stomach."

Dr. C. J. Watson, Professor and Head, Department of Medicine, recently visited the University of Oregon Medical School in Portland, Oregon, where he delivered the Jones Lectures in Medicine. On Monday, March 3, he discussed "The Erythrocyte Porphyrins with Special Reference to the Anemias," and the following day he talked on "Clinical and Fundamental Studies of Porphyria."

Dr. Wesley W. Spink, Professor, Department of Medicine, recently spoke on "The Recognition and Management of Human Brucellosis," at the Chicago Medical Society Meeting.

* * *

New Minnesota Medical Foundation Members

Howard M. Frykman, M.D., Minneapolis
Marguerite Booth, M.D., Minneapolis
Fred B. Riegel, M.D., St. Croix Falls
Mancel T. Mitchell, M.D., Minneapolis
William J. Focke, M.D., Poynette, Wisc.
Mr. George E. Schaffer, Ada
Miss Gertrude Gilman, Minneapolis
William T. Peyton, M.D., Minneapolis
Mary Saunders Bulkley, Minneapolis
E. J. Engberg, M.D., Faribault
Frank R. Gratzek, M.D., Minneapolis
Harry Medovy, M.D., Winnipeg, Canada
Francis Roach, M.D., California
Anthony F. Rozycki, M.D., Pine River
O. A. Brines, M.D., Detroit, Michigan
Osmund J. Baggenstoss, M.D., Minneapolis
Carlton L. Ould, M.D., Fresno, California
Dagfinn Lie, M.D., Webster, South Dakota
Mrs. Bonita W. Abbott, Tooele, Utah

III. UNIVERSITY OF MINNESOTA MEDICAL SCHOOL
WEEKLY CALENDAR OF EVENTS

Physicians Welcome

March 17 - 22, 1952

Monday, March 17

Medical School and University Hospitals

9:00 - 9:50 Roentgenology-Medicine Conference; L. G. Rigler, C. J. Watson and Staff; Todd Amphitheater, U. H.

9:00 - 10:50 Obstetrics and Gynecology Conference; J. L. McKelvey and Staff; W-612, U. H.

10:00 - 12:00 Neurology Rounds; A. B. Baker and Staff; Station 50, U. H.

11:30 - Tumor Conference; Doctors Kremen, Moore, and Stenstrom, Todd Amphitheater, U. H.

11:30 - Physical Medicine Seminar; 142 Chemical Engineering Bldg.

12:15 - Obstetrics and Gynecology Journal Club; Staff Dining Room, U. H.

1:30 - 2:30 Pediatric-Neurological Rounds; R. Jensen, A. B. Baker and Staff; U. H.

4:00 - Pediatric Seminar; The Premature Infant; R. Novick; Sixth Floor West, U. H.

4:30 - 5:30 Dermatological Seminar; M-346, U. H.

4:30 - Public Health Seminar; 15 Owre Hall.

5:00 - 6:00 Urology-Roentgenology Conference; C. D. Creevy, O. J. Baggenstoss, and Staff; Eustis Amphitheater.

Minneapolis General Hospital

7:30 - Fracture Grand Rounds; Dr. Zierold; Sta. A.

10:30 - 12:00 Tuberculosis and Contagion Rounds; Thomas Lowry; Station M.

11:00 - Pediatric Rounds; Franklin H. Top; 7th Floor.

12:30 - Surgery Grand Rounds; Dr. Zierold; Sta. A.

1:00 - X-ray Conference; Classroom, 4th Floor.

1:30 - Pediatric Rounds; Robert Ulstrom; 4th Floor.

Veterans Administration Hospital

9:00 - G. I. Rounds; R. V. Ebert, J. A. Wilson, Norman Shrifter; Bldg. I.

11:30 - X-ray Conference; Conference Room; Bldg. I.

2:00 - Psychosomatic Rounds; Bldg. 5.

3:30 - Psychosomatic Rounds; C. K. Aldrich; Bldg. I.

Tuesday, March 18

Medical School and University Hospitals

9:00 - 9:50 Roentgenology-Pediatric Conference; L. G. Rigler, I. McQuarrie and Staff; Eustis Amphitheater, U. H.

9:00 - 12:00 Cardiovascular Rounds; Station 30, U. H.

12:00 - 1:30 Selected Topics, Permeability and Metabolism; Nathan Lifson; 129 Millard Hall.

12:30 - 1:20 Pathology Conference; Autopsies; J. R. Dawson and Staff; 102 I. A.

4:00 - 5:00 Pediatric Rounds on Wards; I. McQuarrie and Staff; U. H.

4:30 - 5:30 Clinical-Medical-Pathological Conference; Todd Amphitheater, U. H.

5:00 - 6:00 X-ray Conference; Presentation of Cases by Veterans Hospital Staff; Drs. Fink, O'Loughlin, et al., Eustis Amphitheater, U. H.

* 8:00 p.m. Minnesota Pathological Society Meeting; Studies on Breast Cancer Patients Receiving Estrogen Therapy; Robert A. Huseby; Owre Amphitheater.

Ancker Hospital

8:00 - 9:00 Fracture Conference; Auditorium.

1:00 - 2:30 X-ray Surgery Conference; Auditorium.

Minneapolis General Hospital

8:00 - Pediatric Rounds; Spencer F. Brown; 5th Floor.

10:30 - 12:00 Medicine Rounds; Thomas Lowry and Staff; Station F.

11:00 - Pediatric Rounds; Erling S. Platou; 7th Floor.

Veterans Administration Hospital

7:30 - Anesthesiology Conference; Conference Room, Bldg. I.

8:30 - Infectious Disease Rounds; Dr. Hall.

8:45 - Surgery Journal Club; Conference Room, Bldg. I.

9:00 - Liver Rounds; Drs. Nesbitt and MacDonald.

9:30 - Surgery-Pathology Conference; Conference Room, Bldg. I.

10:30 - Surgery Tumor Conference; Conference Room, Bldg. I.

1:00 - Surgery Chest Conference; T. Kinsella and Wm. Tucker; Conference Room, Bldg. I.

2:00 - 2:50 Dermatology and Syphilology Conference; H. E. Michelson and Staff Bldg. III.

3:30 - 4:20 Autopsy Conference; E. T. Bell and Donald Gleason; Conference Room, Bldg. I.

Wednesday, March 19

Medical School and University Hospitals

8:00 - 8:50 Surgery Journal Club; O. H. Wangensteen and Staff; M-109, U. H.

8:00 - 9:00 Roentgenology-Surgical-Pathological Conference; Norman Jacob and L. G. Rigler; Todd Amphitheater, U. H.

11:00 - 12:00 Pathology-Medicine-Surgery Conference; Pediatrics Case; O. H. Wangensteen, C. J. Watson and Staff; Todd Amphitheater, U. H.

5:00 - 5:50 Urology-Pathological Conference; C. D. Creevy and Staff; Eustis Amphitheater, U. H.

5:00 - 6:00 Vascular Conference; Todd Amphitheater, U. H.

5:00 - 7:00 Dermatology Clinical Seminar; Dining Room, U. H.

7:00 - 8:00 Dermatology Journal Club; Dining Room, U. H.

8:00 - 10:00 Dermatological-Pathology Conference; Review of Histopathology Section; R. Goltz; Todd Amphitheater, U. H.

Ancker Hospital

8:30 - 9:30 Clinico-Pathological Conference; Auditorium.

3:30 - 4:30 Journal Club; Surgery Office.

Minneapolis General Hospital

8:00 - Pediatric Allergy Rounds; Lloyd Nelson; 4th Floor.

10:30 - 12:00 Medicine Rounds; Thomas Lowry and Staff; Station D.

11:00 - Pediatric Rounds; Franklin H. Top; 7th Floor.

12:00 - Surgery-Physiology Conference; Dr. Zierold and Dr. E. B. Brown; Classroom.

12:30 - Pediatric Staff Meeting; Factors of Importance in Breast Milk; Theresa Haddy; 4th Floor Annex.

12:30 - EKG Conference; Boyd Thomes and Staff; 302 Harrington Hall.

1:30 - Pediatric Rounds; E. J. Huenekens and Robert Ulstrom; 4th Floor.

Veterans Administration Hospital

8:30 - 10:00 Orthopedic X-ray Conference; Conference Room, Bldg. I.

8:30 - 12:00 Neurology Rehabilitation and Case Conference; A. B. Baker.

2:00 - 4:00 Infectious Disease Rounds; Conference Room, Bldg. I.

4:00 - 5:00 Infectious Disease Conference; W. Spink; Conference Room, Bldg. I.

7:00 p.m. Lectures in Basic Science of Orthopedics; Conference Room, Bldg. I.

Thursday, March 20

Medical School and University Hospitals

Thursday, March 20

Medical School and University Hospitals

8:00 - 9:00 Vascular Rounds; Davitt Felder and Staff Members from the Departments of Medicine, Surgery, Physical Medicine, and Dermatology; Heart Hospital Amphitheater.

9:00 - 11:50 Medicine Ward Rounds; C. J. Watson and Staff; E-221, U. H.

11:00 - 12:00 Cancer Clinic; K. Stenstrom and A. Kremen; Todd Amphitheater, U. H.

1:30 - 4:00 Cardiology X-ray Conference; Heart Hospital Theatre.

3:30 - Medicine-Pediatric Infectious Disease Conference; Heart Hospital Auditorium.

4:00 - 5:00 Physiology-Surgery Conference; Todd Amphitheater, U. H.

4:30 - 5:20 Ophthalmology Ward Rounds; Erling W. Hansen and Staff; E-534, U. H.

5:00 - 6:00 Radiology Seminar; Some Aspects of the Action of X-Rays on Living Cells; Halvor Vermund; Eustis Amphitheater, U. H.

7:30 - 9:30 Pediatric Cardiology Conference and Journal Club; Review of Current Literature 1st hour and Review of Patients 2nd hour; 206 Temporary West Hospital.

* 8:15 p.m. E. Starr Judd Lectureship in Surgery; "Some Observations on the Treatment of Carcinoma of the Pancreas," Dr. Thomas G. Orr; Professor of Surgery, University of Kansas, Kansas City; Owre Amphitheater.

Minneapolis General Hospital

8:00 - Pediatric Rounds; Spencer F. Brown; 5th Floor.

8:30 - Neurology Rounds; William Heilig; 4th Floor.

11:00 - Pediatric Rounds; Erling S. Platou; 7th Floor.

1:00 - Fracture-X-ray Conference; Dr. Zierold; Classroom.

Veterans Administration Hospital

8:00 - Surgery Ward Rounds; Lyle Hay and Staff; Ward 11.

9:15 - Surgery Grand Rounds; Conference Room, Bldg. I.

11:00 - Surgery Roentgen Conference; Conference Room, Bldg. I.

Friday, March 21

Medical School and University Hospitals

8:30 - 10:00 Neurology Grand Rounds; A. B. Baker and Staff; Station 50, U. H.

9:00 - 9:50 Medicine Grand Rounds; C. J. Watson and Staff; Todd Amphitheater, U. H.

10:30 - 11:50 Medicine Rounds; C. J. Watson and Staff; Todd Amphitheater, U. H.

10:30 - 11:50 Otolaryngology Case Studies; L. R. Boies and Staff; Out-Patient Department, U. H.

Friday, March 21 (Cont.)

Medical School and University Hospitals (Cont.)

11:45 - 12:50 University of Minnesota Hospitals Staff Meeting; New Drugs in the Treatment of hypertension; Carleton B. Chapman; Powell Hall Amphitheater.

1:00 - 2:50 Neurosurgery-Roentgenology Conference; W. T. Peyton, Harold O. Peterson and Staff; Todd Amphitheater, U. H.

2:00 - 3:00 Dermatology and Syphilology Conference; Presentation of Selected Cases of the Week; H. E. Michelson and Staff; W-312, U. H.

3:00 - 4:00 Neuropathological Conference; F. Tichy; Todd Amphitheater, U. H.

3:30 - 4:30 Advanced Neurophysiology Seminar; E. Gellhorn; 111 Owre Hall.

4:00 - 5:00 Dermatology Seminar; W-321, U. H.

5:00 - Urology Seminar and X-ray Conference; Eustis Amphitheater, U. H.

Ancker Hospital

1:00 - 3:00 Pathology-Surgery Conference; Auditorium.

Minneapolis General Hospital

11:00 - Pediatric Rounds; Franklin H. Top; 7th Floor.

11:00 - Pediatric-Surgery Conference; Dr. Wyatt, Forrest Adams; Classroom, Sta. I.

12:00 - Surgery-Pathology Conference; Dr. Zierold, Dr. Coe; Classroom.

1:00 - 3:00 Clinical Medical Conference; Thomas Lowry; Classroom, Station M.

1:30 - Pediatric Rounds; Robert Ulstrom; 4th Floor.

Veterans Administration Hospital

10:30 - 11:20 Medicine Grand Rounds; Conference Room, Bldg. I.
1:00 - Microscopic-Pathology Conference; E. T. Bell; Conference Room, Bldg.I.
1:30 - Chest Conference; Wm. Tucker and J. A. Meyers; Ward 62, Day Room.
3:00 - Renal Pathology; E. T. Bell; Conference Room, Bldg. I.

Saturday, March 22

Medical School and University Hospitals

7:45 - 8:50 Orthopedic X-ray Conference; W. H. Cole and Staff; M-109, U. H.
9:00 - 10:30 Pediatric Grand Rounds; I. McQuarrie and Staff; Eustis Amphitheater,
9:00 - 11:50 Medicine Ward Rounds; C. J. Watson and Staff; Heart Hospital Amphitheater.
9:15 - 10:00 Surgery-Roentgenology Conference; L. G. Rigler, J. Friedman, Owen H. Wangensteen and Staff; Todd Amphitheater, U. H.
10:00 - 11:30 Surgery Conference; Todd Amphitheater, U. H.
10:00 - 12:50 Obstetrics and Gynecology Grand Rounds; J. L. McKelvey and Staff;, Station 44, U. H.

Minneapolis General Hospital

8:00 - Podiatric Rounds; George Lund; 5th Floor.
11:00 - 12:00 Medical - X-ray Conference; O. Lipschultz, Thomas Lowry, and Staff; Main Classroom.
11:00 - Pediatric Clinic; C. D. May and Floyd Denny; Classroom, 4th Floor.

Veterans Administration Hospital

8:00 - Proctology Rounds; W. C. Bernstein and Staff; Bldg. III.
8:30 - Hematology Rounds; P. Hagenaand E. F. Englund.

* Indicates special meeting. All other meetings occur regularly each week at the same time on the same day. Meeting place may vary from week to week for some conferences.