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A STUDY OF PREMATURE BEATS BY ELECTROCARDIOGRAM

ANNIE MARY LYLE

IRREGULARITIES of the pulse were among the first impairments studied in the modern substandard era. The irregularities were detected by palpating the pulse at the wrist or listening to the heartbeat at the apex. Cases so obtained were divided into two groups: Intermittent Pulse, in which the break in rhythm came at regular intervals; and Irregular Pulse, in which it came irregularly. Each of these groups was subdivided by age, number per minute, and reaction to exercise.

With the advent of the electrocardiogram it became evident that these groups, although made up chiefly of premature beats, contained some of the more serious arrhythmias: namely, auricular fibrillation, auricular flutter with irregular ventricular response, second degree heart block with dropped beats, and a rare type of sinus arrhythmia (believed to be arteriosclerotic in origin) characterized by marked and abrupt changes in rate that are not phasic with respiration. Some cases of all these types slipped through, despite the fact that considerable progress had been made in excluding them on clinical examination. With the electrocardiogram they were easily excluded, and more recent studies have justified a small deduction from the usual rating for an irregularity of the pulse when the irregularity is identified by electrocardiogram as premature beats and the electrocardiogram is otherwise normal. A small addition to the rating is sometimes made for certain features: for example, if the premature beats arise from multiple foci. The distinction between irregular and intermittent pulse has been discarded. Aside from these modifications, the underwriting classification is still essentially as it was in the beginning—based primarily on age, number per minute, and reaction to exercise.

But the electrocardiogram does more than identify simple premature beats as such. In many cases it shows certain well-defined variations apart from and in addition to the identifying characteristics. Over the years we have gained the impression that the following variants should be looked upon unfavorably:

The occurrence of pairs or brief runs of premature beats.
Bigeminy, that is, premature beats alternating with sinus beats.
Failure of either bundle branch to conduct an auricular premature beat.
Depressed T wave in the first sinus beat following a premature beat.
Multifocal origin.

The first two can usually be detected on clinical examination but are sometimes confusing. The last three cannot be detected without an electrocardiogram.

We have also observed that when premature beats develop during the course of chronic cardiovascular disease of any type, the disease is likely to progress more rapidly thereafter.

The present investigation was designed to study the mortality on premature beats identified as such by electrocardiogram, to test our impression that the variants listed above represent more serious impairment, and to find out whether the combination of premature beats and chronic cardiovascular disease results in a higher extra mortality than do the same impairments occurring separately. Any additional light that can be thrown on these questions would enable us to improve our selection of risks still further, particularly from the standpoint of equitable treatment.

Individuals entered the study between the ages of 17 and 69 on the date that premature beats were first identified by electrocardiogram. No new entrants were admitted after December 31, 1959, and observations closed June 30, 1960. Expected mortality was computed on the 1946–1949 Basic Table, Select and Ultimate. The cases studied were culled from a file containing all of the electrocardiograms that we have made on Prudential home office employees from 1933 to date—a total of approximately 30,000 electrocardiograms on 11,000 or more individuals.

This file has certain advantages for investigative purposes. All pertinent information is given in full detail, so that intensive study can be carried out on a pilot project. The follow-up is unusually good, as few are lost by resignation at the middle and older ages, and those who retire remain on the Company's pension records until death. Most important of all, it is our practice to recall for repeated examinations those who have any evidence of cardiovascular disease, so that their progress can be observed.

But the file also has disadvantages. One is that it does not represent a cross-section of the Prudential population, so that conclusions as to the incidence or frequency with which an impairment occurs can seldom be drawn. This is partly because our examinations are voluntary, and many do not choose to come. It is also because there is a disproportionate amount of pathology as a result of our follow-up practice: we consistently make and repeat electrocardiograms on all who show any evidence of cardiovascular disease but do not consistently do so on those who are normal. Another disadvantage is that, with electrocardiographic abnormalities so numerous and occurring in so many combinations and in association with so many clinical impairments, it is impossible to set up groups of satis-

factory size that are reasonably homogeneous. Nevertheless, it is believed that the findings in this study do indicate some possible modifications of underwriting rules, as well as further studies that may be worth while.

Almost immediately difficulties began to appear. The volume of material available was less than had been anticipated. There were only 1,139 individuals who had premature beats recorded at any time, out of the 11,000 or more individuals in the file. From this it would appear that premature beats are less common than they are generally believed to be. There were, all told, 968 cases with premature beats that were simple at the onset—that is, premature beats that had none of the variants listed above when they were first recorded. There were 171 cases with premature beats that showed variants on the first record. Of the 968 that began as simple premature beats, 180 later developed one or more variants and were included in both groups. These 180 were placed first in the Simple group and remained there until their death or the close of observation; they were entered in the Variant group when the variant features appeared, at the then attained age and at select duration one, and remained there until the end of observation or prior death. There were, then, a total of 968 Simple premature beats and 351 Variant premature beats. Among the 1,139 individuals studied, there were 258 deaths.

There were 429 out of 1,139 individuals with no clinical or electrocardiographic abnormalities other than the premature beats. The remaining 710 cases had a variety of associated impairments. The latter obviously posed a difficult problem of meaningful and significant subgrouping.

There was also the question of how best to classify the premature beats themselves. The effect of exercise unfortunately could not be studied, as we do not perform an exercise test routinely for premature beats on employee examinations. Subdivision by age showed no definite trend in mortality, nor did subdivision by number per minute. The amount of material available for these studies was too small to show a trend if one exists, but it is noted that the 1951 Impairment Study, with a larger amount of material, showed no trend by age or number per minute.

The present study by number per minute did, however, yield the interesting observation that a high frequency is more often associated with Variant than with Simple premature beats. Of the 968 cases of Simple premature beats, only 64 (6.6%) had a frequency of more than ten a minute at any time. Of the 351 cases of Variant premature beats, 147 (41.9%) had a frequency of more than ten a minute at some time. Some allowance must be made for the fact that one of the features used in classifying premature beats as Variant was bigeminy, which implies (and usually produces) a high count, but a review of the cases showed that

bigeminy alone was responsible for the Variant classification in only 30 of the 147 cases. This observation suggests that the variants that are undetectable on clinical examination may have been a significant factor in the increase in mortality with increase in frequency that has been found in the past by counting the pulse.

The historic method of counting the number per minute has worked very well in practice but, as one follows the course of premature beats, one wonders why it has. Premature beats are very erratic. They are present on one examination and not on another; almost no one in this study had premature beats on every examination. They vary greatly in frequency, too—numerous on one examination and rare on another. In fact, it is not unusual to find marked variation on the same examination—bigeminal rhythm in one portion of the electrocardiogram and uninterrupted sinus rhythm in another. They may occur regularly or irregularly. They may come in scattered single beats, or they may come in showers. At best the count is inaccurate, because a premature beat is visible in the electrocardiogram only when the ectopic focus discharges while the heart is susceptible to stimulation, that is, between sinus beats; when it discharges while the heart is refractory during a sinus beat, the premature beat is not perceptible.

The variant features, likewise, come and go from day to day and even in the same record. An impaired bundle branch may conduct one early auricular impulse and not another. Two separate foci may be active on the same examination, or one of them on one examination and the other on another. Examinations showing Variant premature beats are often scattered among examinations showing Simple premature beats or no premature beats at all. A given case may show any one, two, three, four, or all five of the variants at one time or another.

These characteristics raised the secondary question of whether or not to include cases which showed the pertinent findings only rarely. For example: Should cases on which a single premature beat was recorded on only one of several examinations be included in the study? Should a single instance of two premature beats with one sinus beat between be considered bigeminy? It was concluded that the rarity of the finding should be no bar, on the theory that if the potential is there, the finding must often occur undetected, since we can observe a person for so small a portion of his time. An individual who has had ten electrocardiograms spread over a period of twenty years might be said to have been kept fairly well under observation. But an electrocardiogram as routinely made in our office comprises runs totaling approximately two minutes, so that he has actually been under electrocardiographic observation for only twenty minutes in twenty years.

It was decided, therefore, to classify the premature beats merely as Simple or Variant—Simple if none of the five electrocardiographic variants previously listed is present, Variant if any one or more of them are present.

There was still unsolved the problem of dealing with the associated clinical and electrocardiographic impairments. As a trial measure, the associated impairments (the premature beats themselves being ignored) were classified as Slight to Moderate (+30% to +200%) and Marked to Very Marked (+201% up, without limit), based roughly on our underwriting practice.

The results are of little practical use in underwriting, and have been relegated to the appendix. They are of interest in that they do support our day to day impressions. The mortality ratios are consistently higher in the Variant groups than in the Simple groups. They show an upward trend as the degree of associated impairment increases. The cardiovascular death rate is high. Males had a higher mortality than females.

But the results do not tell us what we need to know for underwriting purposes. They do not tell us whether the combination of premature beats and cardiovascular disease produces a higher extra mortality than the same impairments do when they occur singly. They do not tell us what the additional mortality is on the variant features. The difficulty is, of course, that we cannot tell how much of the extra mortality is due to the associated impairments, nor is there any assurance that the degree of impairment is, on the average, the same in all the Slight to Moderate groups or in all the Marked to Very Marked groups.

To provide a more satisfactory control, it was necessary to use a single, definite impairment that could be subdivided into an acceptably narrow range. Hypertension fulfills these requirements and is present in our file in workable volume. And so, individuals who had hypertension with premature beats and no other impairment were measured against individuals who had hypertension without premature beats and no other impairment.

The hypertension was classified by the table shown on page 250, *TSA*, VI, as:

Slight	+ 30% to + 80%
Moderate	+ 81% to +200%
Marked	+201% to +400%
Very Marked	Over +400%

In grading the hypertension, blood pressures were averaged in such a way as to determine whether the individual was, on the whole, a slight, moderate, marked, or very marked hypertensive during any given part of the observation period. When the degree of hypertension increased, or when premature beats made their appearance or took on variant fea-

TABLE 1

ELEVATED BLOOD PRESSURE AND PREMATURE BEATS

BLOOD PRESSURE	PREMATURE BEATS	NUMBER OF CASES (1)	ACTUAL DEATHS (2)	EXPECTED DEATHS (3)	MORTALITY RATIOS (4) = (2) ÷ (3)	CARDIOVASCULAR DEATHS (5)	CARDIOVASCULAR DEATHS (6) = (5) ÷ (2)
		Males					
Normal.....	None	1,805	145	187.1	78%	73	50%
	Simple	267	25	26.1	96	13	52
	Variant	63	7	6.5	108	3	43
Slight.....	None	472	58	45.4	128%	38	66%
	Simple	82	17	7.3	233	12	71
	Variant	27	9	3.0	297	7	78
Moderate....	None	341	61	31.0	197%	47	77%
	Simple	59	10	6.7	149	10	100
	Variant	23	9	3.0	300	8	89
Marked.....	None	126	32	9.3	342%	27	84%
	Simple	27	5	1.3	391	5	100
	Variant	9	2	.5	425	1	50
Very Marked.	None	220	95	16.9	563%	79	83%
	Simple	61	28	4.4	635	23	82
	Variant	28	14	2.0	713	10	71
		Females					
Normal.....	None	902	29	102.8	28%	10	35%
	Simple	109	6	12.5	48	2	33
	Variant	28	1	1.5	66	0	0
Slight.....	None	247	14	36.5	38%	10	71%
	Simple	51	4	4.0	100	4	100
	Variant	9	1	.7	139	0	0
Moderate....	None	234	26	36.1	72%	17	65%
	Simple	46	4	3.5	115	2	50
	Variant	9	1	.6	165	1	100
Marked.....	None	71	12	10.4	115%	10	83%
	Simple	19	4	2.2	185	3	75
	Variant	6	0	.4	0	0	.....
Very Marked.	None	179	51	26.6	192%	43	84%
	Simple	58	16	7.4	217	16	100
	Variant	18	10	2.5	405	10	100

tures, the case was entered again in the appropriate classification (in the same manner that the 180 cases of Simple premature beats which later became Variant were entered in both groups), so that some cases were included in two or more groups. Nothing was added for electrocardiographic abnormalities (mostly T wave changes) that are often associated with hypertension. It is recognized that these do add something to the risk, but the added risk is presumably included in the blood pressure ratings, since hypertension is usually underwritten without electrocardiogram. To bring the resulting mortality ratios into better perspective, there were added to the hypertensive groups the premature beats with blood pressure normal (those without other impairments) taken from the

TABLE 2

ANGINA PECTORIS AND PREMATURE BEATS

Premature Beats	Number of Cases (1)	Actual Deaths (2)	Expected Deaths (3)	Mortality Ratios (4) = (2) ÷ (3)	Cardiovascular Deaths (5)	Cardiovascular Deaths (6) = (5) ÷ (2)
	Males					
None	56	16	7.2	223%	12	75%
Simple	16	5	1.8	284	4	80
Variant	7	2	.6	308	1	50

tables in the appendix, and normal control groups with blood pressure normal and no premature beats. The normal control groups consist of men and women taken from the same file, ages 40 to 69 and for the same observation period, who had no premature beats at entry, whose electrocardiograms were normal, and who had no other impairments. The results of this study are given in Table 1.

Similar comparisons were made on angina pectoris and coronary occlusion, Tables 2 and 3. All of the cases used were normotensive prior to onset and had no other impairments except premature beats. Some of those classified as angina pectoris had a subsequent coronary occlusion and were included in both groups. None classified as angina pectoris had had a previous occlusion. These two studies were confined to males, since there were too few females to study.

And finally, a study was made of rheumatic heart disease, Table 4. Many of these cases had lesions of both the aortic and the mitral valves. Only 7 of them (3 men and 4 women) had mitral insufficiency without a dias-

TABLE 3

CORONARY OCCLUSION AND PREMATURE BEATS

PREMATURE BEATS	NUMBER OF CASES (1)	ACTUAL DEATHS (2)	EXPECTED DEATHS (3)	MORTALITY RATIOS (4) = (2) ÷ (3)	CARDIOVASCULAR DEATHS (5)	CARDIOVASCULAR DEATHS (6) = (5) ÷ (2)
	Males					
None	110	47	11.4	414%	42	89%
Simple	54	22	6.1	360	21	96
Variant	28	14	2.1	654	13	93

TABLE 4

RHEUMATIC HEART DISEASE AND PREMATURE BEATS

PREMATURE BEATS	NUMBER OF CASES (1)	ACTUAL DEATHS (2)	EXPECTED DEATHS (3)	MORTALITY RATIOS (4) = (2) ÷ (3)	CARDIOVASCULAR DEATHS (5)	CARDIOVASCULAR DEATHS (6) = (5) ÷ (2)
	Males					
None	39	14	4.0	351%	13	93%
Simple	15	8	1.2	654	8	100
Variant	6	3	.7	420	3	100
Auricular fibrillation*	27	12	1.0	1,186	12	100
	Females					
None	41	13	2.7	484%	11	85%
Simple	18	8	1.1	702	8	100
Variant	10	2	.4	470	2	100
Auricular fibrillation*	26	12	1.4	830	11	92

* Some of these cases had occasional "premature" beats (that is, ectopic beats from a ventricular focus), but they were ignored as being overshadowed in importance by the auricular fibrillation. The cases classified as premature beats in this table had not had auricular fibrillation at any time prior to entering these groups, although some of them later developed it.

tolic murmur at the apex, but they were well-documented rheumatics. In this group auricular fibrillation was added for comparison with the premature beats.

The results again support our day-to-day impressions, but a much larger amount of material is needed for dependable mortality ratios. In the absence of sufficient material it is of some interest to see what the present study would have looked like without accidental fluctuations. Accordingly, the male mortality ratios in Table 1, as calculated, were smoothed by the graphic method, not to produce the closest fit possible but to produce a reasonably close fit along with a smooth progression from group to group. The ratios for blood pressure without premature beats were left unchanged, being already smooth enough. The adjusted mortality ratios were then applied to the calculated expected mortality to test the fit. The total adjusted deaths so obtained were 520 compared with actual deaths of 517, an error of 0.6%. Since different underwriting schedules for males and females would be impractical, the smoothed mortality ratios for females were constructed by adding the male increase in mortality due to premature beats to the female mortality ratios without premature beats at corresponding blood pressure levels. The females' adjusted deaths were 186 compared with 179 actual deaths, an error of 3.9%. The results are shown in Table 5.

In appraising the results, it must be borne in mind that the mortality ratios in this table, and in all others, should be compared with the normal control group's 78%, and not with 100% of the Basic Table. Thus, in column (4), the 93% on Simple premature beats and 108% on Variant premature beats without other impairments represent some increase in mortality over standard. These ratios, in the section on normal blood pressure, show the mortality to be expected on Simple and Variant premature beats *per se*. When the excess mortality on premature beats *per se* is added to the mortality ratios for hypertension without premature beats in the following sections of the table, it can be seen that the combination of premature beats and elevated blood pressure results in an extra mortality in excess of the sum of the extra mortality on the two separate impairments, that the excess increases as the degree of hypertension increases, and that it is greater for Variant premature beats than for Simple premature beats.

Although no attempt was made to smooth the mortality ratios on angina pectoris, coronary occlusion, and rheumatic heart disease because of the paucity of data, the combination of premature beats and these forms of cardiovascular disease obviously produces an extra mortality in excess of that on the separate impairments.

I believe the present study suggests a reasonable approach to the selection of premature beats. With sufficient material it might be profitable to study, in addition, the electrocardiographic variants individually and in combinations, to find out whether or not all of them should have equal weight; whether two of them constitute a greater risk than one, three a greater risk than two, etc. There are other questions, too, not touched upon in this study. Are multifocal premature beats more serious when they come from two ventricular foci than when one focus is ventric-

TABLE 5

ELEVATED BLOOD PRESSURE AND PREMATURE BEATS AFTER SMOOTHING

BLOOD PRESSURE	PREMATURE BEATS	ACTUAL DEATHS (1)	EXPECTED DEATHS (2)	CALCULATED MORTALITY RATIOS (3) = (1) ÷ (2)	SMOOTHED MORTALITY RATIOS (4)	ADJUSTED DEATHS (5) = (2) × (4)
	Males					
Normal.....	None	145	187.1	78%	78%	146
	Simple	25	26.1	96	93	24
	Variant	7	6.5	108	108	7
	Totals	177				177
Slight.....	None	58	45.4	128%	128%	58
	Simple	17	7.3	233	163	12
	Variant	9	3.0	297	193	6
	Totals	84				76
Moderate....	None	61	31.0	197%	197%	61
	Simple	10	6.7	149	257	17
	Variant	9	3.0	300	302	9
	Totals	80				87
Marked.....	None	32	9.3	342%	342%	32
	Simple	5	1.3	391	432	6
	Variant	2	.5	425	492	2
	Totals	39				40
Very Marked.	None	95	16.9	563%	563%	95
	Simple	28	4.4	635	688	30
	Variant	14	2.0	713	763	15
	Totals	137				140
	Grand Totals	517				520

TABLE 5—*Continued*

Blood Pressure	Premature Beats	Actual Deaths (1)	Expected Deaths (2)	Calculated Mortality Ratios (3) = (1) ÷ (2)	Smoothed Mortality Ratios (4)	Adjusted Deaths (5) = (2) × (4)
	Females					
Normal.....	None	29	102.8	28%	28%	29
	Simple	6	12.5	48	43	5
	Variant	1	1.5	66	58	1
	Totals	36				35
Slight.....	None	14	36.5	38%	38%	14
	Simple	4	4.0	100	73	3
	Variant	1	.7	139	103	1
	Totals	19				18
Moderate....	None	26	36.1	72%	72%	26
	Simple	4	3.5	115	132	5
	Variant	1	.6	165	177	1
	Totals	31				32
Marked.....	None	12	10.4	115%	115%	12
	Simple	4	2.2	185	205	4
	Variant	0	.4	0	265	1
	Totals	16				17
Very Marked.	None	51	26.6	192%	192%	51
	Simple	16	7.4	217	317	23
	Variant	10	2.5	405	392	10
	Totals	77				84
	Grand Totals	179				186

ular and the other auricular? Do unifocal premature beats of supraventricular origin have a significantly different mortality from those of ventricular origin?

Considering the complexity of the problem and the disappointingly small amount of material on premature beats in past intercompany mortality studies, one must conclude that it may be a long time before sufficient material is available. Meanwhile, in day-to-day underwriting, it would seem reasonable to give some weight to the over-all results of the present study, which may be summarized as follows:

(1) The mortality ratios on premature beats without other impairments do represent some increase in mortality when compared with those of the normal control groups as standard, although the increase is small.

(2) Premature beats with the electrocardiographic variants described show a higher mortality than premature beats that do not have these variants. The variants are apparently more significant than age or number per minute.

(3) The combination of premature beats and some chronic cardiovascular impairment results in an extra mortality in excess of the extra mortality on the separate impairments. The excess increases as the severity of the associated impairment increases.

(4) The female mortality ratios show the same trend as the male ratios, but at a lower level.

One would, then, add something to the sum of the separate ratings for a combination of Simple premature beats and chronic cardiovascular disease, and add a somewhat larger amount in the case of Variant premature beats, with the amounts that are added graded upward as the degree of associated impairment increases.

APPENDIX

PREMATURE BEATS, SIMPLE AT ONSET, 180 OF WHICH LATER DEVELOPED VARIANT FEATURES, WITH A HETEROGENEOUS GROUP OF ASSOCIATED IMPAIRMENTS

Impairment	Number of Cases (1)	Actual Deaths (2)	Expected Deaths (3)	Mortality Ratios (4) = (2) ÷ (3)	Cardiovascular Deaths (5)	Cardiovascular Deaths (6) = (5) ÷ (2)
	Males					
None	267	25	26.1	96%	13	52%
Slight–Moderate	174	30	15.3	196	23	77
Marked–Very Marked	224	98	20.3	482	86	88
Totals	665	153	61.7	248%	122	80%
	Females					
None	109	6	12.5	48%	2	33%
Slight–Moderate	80	8	7.2	112	6	75
Marked–Very Marked	114	35	13.4	261	30	86
Totals	303	49	33.1	148%	38	78%

PREMATURE BEATS WITH VARIANT FEATURES, 180 OF WHICH BEGAN AS SIMPLE PREMATURE BEATS, WITH A HETEROGENEOUS GROUP OF ASSOCIATED IMPAIRMENTS

IMPAIRMENT	NUMBER OF CASES (1)	ACTUAL DEATHS (2)	EXPECTED DEATHS (3)	MORTALITY RATIOS (4) = (2) ÷ (3)	CARDIOVASCULAR DEATHS (5)	CARDIOVASCULAR DEATHS (6) = (5) ÷ (2)
	Males					
None	63	7	6.5	108%	3	43%
Slight–Moderate	62	19	6.9	274	17	90
Marked–Very Marked	130	57	11.4	501	48	84
Totals	255	83	24.8	335%	68	82%
	Females					
None	28	1	1.5	66%	0	0%
Slight–Moderate	20	4	1.8	224	2	50
Marked–Very Marked	48	17	4.3	400	16	94
Totals	96	22	7.6	291%	18	82%

The following list shows the impairments found associated with premature beats in this study, and the degree of impairment assigned to each in the preceding table. Combinations of impairments were treated as they are in underwriting, that is, additive with an additional debit for the combination where necessary.

No Impairment
- Functional murmur
- Non-cardiac chest pain
- Wandering pacemaker (by EKG)
- Sino-auricular block (by EKG)
- Incomplete right bundle branch block (by EKG)

Slight to Moderate Impairment
- Blood pressure elevated, slight to moderate
- Organic murmur, not rheumatic
- Albumin, blood, pus, casts, slight to moderate
- Diabetes mellitus
- Intermittent claudication
- Recurrent paroxysmal tachycardia, age 40 or over
- Displaced pacemaker, age 50 or over (by EKG)
- First degree heart block, over 0.22 sec. (by EKG)
- Wolff-Parkinson-White syndrome (by EKG)
- T wave abnormalities, minor or major (by EKG)

Marked to Very Marked Impairment
- Blood pressure elevated, marked to very marked
- Angina pectoris
- Coronary occlusion
- Rheumatic heart disease
- Albumin, blood, pus, casts, marked to very marked
- Senile type of sinus arrhythmia (by EKG)
- Intraventricular conduction defect (by EKG)
- Complete bundle branch block (by EKG)
- Left ventricular hypertrophy pattern (by EKG)
- Cardiac hypertrophy (by X-ray)

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DISCUSSION OF PRECEDING PAPER

ANDREW C. WEBSTER:

Miss Lyle deserves our thanks for an excellent study. The underwriter is always glad to have additional information to help him in classifying risks. Speaking as general chairman of the Committee on Mortality and Morbidity among Lives Individually Insured, I would like to make a plea for more studies from individual companies. This paper demonstrates the value of such investigations in that the author has, with apologies to our motto, replaced underwriting impressions with facts, and these are not obscured by the relatively small numbers involved. Only rarely can we erect an edifice like the Build and Blood Pressure Study, but we can always use building blocks such as this paper.

TABLE 1

Blood Pressure	Rating	Smoothed Mortality Ratios	Equivalent Rating
Normal	100	78%	100
Slight	130–180	128	164
Moderate	181–300	197	252
Marked	301–500	342	438
Very marked	500+	563	721

I have a comment on one point in the paper: The results of the analysis of Elevated Blood Pressure and Premature Beats (Table 5) bear, I think, upon another underwriting problem. In the Build and Blood Pressure Study the Experience on Entrants with Favorable Electrocardiogram showed mortality ratios lower in most instances than those for the corresponding blood-pressure classifications in the main study. The selective nature of this group was emphasized in interpreting the results, but the difference in the ratios has encouraged underwriters to allow credits for favorable electrocardiograms against their blood-pressure ratings.

It seems to me that in Table 5 we have an experience with elevated blood-pressure cases with favorable electrocardiograms. The figures where no premature beats were found are shown in Table 1.

If I read the paper aright, the mortality ratios correspond to the tentative ratings used in the classifications, and, assuming that these correspond to the regular blood-pressure ratings in use, it would suggest that the favorable electrocardiogram may not have too much effect upon the final results.

(AUTHOR'S REVIEW OF DISCUSSION)

ANNIE MARY LYLE:

Mr. Webster has raised a very interesting question. He is mistaken, however, in thinking that all the cases of hypertension that went into Table 5 had normal electrocardiograms. They had no other *clinical* impairments, but some of them did have electrocardiographic abnormalities of the kinds that are often associated with hypertension—most of these being T-wave changes. The mortality ratios in Table 5, then, represent hypertension selected without electrocardiogram, and so also do the debits that were used in grading the hypertension.

Mr. Webster has pointed out that the recent blood-pressure study appears to justify a credit against the blood-pressure rating for an electrocardiogram in which the basic pattern is normal. This was true, also, in a study on hypertension that I presented before this Society several years ago. Whether the same credit for a normal basic pattern is justified when premature beats are present, I do not know. We have seen that combinations of impairments sometimes show unexpected results. I suspect, however, that almost any cardiovascular impairment or combination of such impairments, especially at the middle and older ages, would show a lower mortality if the electrocardiogram were normal than if it were abnormal.

I should like to second Mr. Webster's plea for more studies from individual companies and to say a few words on behalf of small groups, since it is doubtless the paucity of material available that tends to discourage such studies. Small groups are inherently subject to accidental fluctuations, of course. But a small group, all from the same source, is more homogeneous than a large group gathered from many sources with no unified control, and its homogeneity tends to diminish fluctuations. Moreover, any group whose subdivisions give results that are consistent with one another and appear reasonable in the light of our previous knowledge is more convincing than the mathematical theories of probable error based on numbers alone would suggest.

There is a practical aspect, too. When a pilot study is being made to discover what the significant features of an impairment are, each case requires intensive study and careful judgment, and there are often many trials and reappraisals. This would be a formidable undertaking with thousands upon thousands of cases. Determining what the significant features are is the first and most important step in a mortality study made for underwriting purposes. After that has been done, the basic classification can be set up and the thousands upon thousands of cases put into it to obtain more dependable mortality ratios.