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SUDDEN DEATH, CORONARY HEART DISEASE, ATHEROSCLEROSIS AND MYOCARDIAL LESIONS IN YOUNG MEN

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A standardized method for examining hearts and coronary arteries was applied in a study of sudden deaths in 227 autopsies of New Orleans men aged 25–49 years. Of these autopsies, 102 deaths were due to external violence, 125 were from natural causes and 45 occurred suddenly. Of the sudden natural deaths, 14 (31%) were due to coronary heart disease (CHD), seven (16%) were possibly due to CHD and 24 (53%) were due to other causes without evidence of CHD. Large recent myocardial lesions (>1 cm) were present in 19 (95%) of 20 CHD deaths and in 16 (18%) of 91 non-CHD deaths. The myocardial lesions in sudden CHD deaths were subendocardial involving preferentially the posterior septum and the apex of the heart. The lesions in non-sudden CHD deaths were transmural involving preferentially the anterior, lateral and posterior left ventricle and the base of the heart. Men who died of CHD had extensive coronary atherosclerosis, while those who died of other causes had minimal coronary atherosclerosis. The large proportion of CHD deaths which occurred suddenly (70%) reaffirms the need for primary prevention and the need for improved predictive factors for early detection of CHD.

atherosclerosis; coronary disease; death, sudden

Sudden natural deaths are the focal point of much controversy concerning coronary heart disease (CHD). Two points of controversy are the proportion of sudden natural deaths that are due to CHD and the proportion of CHD deaths that are sudden. Whether sudden death is defined as instantaneous, occurring in one hour, two hours or 24 hours, sudden deaths have been found to constitute from 50 to 70 per cent of all deaths due to CHD (1–3). Fifty to 90 per cent of sudden deaths are ascribed to CHD (4). These studies have generally been restricted to deaths certified by the medical examiner, who is frequently under pressure to find a cause of death. The lack of standard definitions of both sudden death and CHD have plagued those studying these problems (5).

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Abbreviations: ALV, anterior left ventricle; AS, anterior septum; CHD, coronary heart disease; ?CHD, questionable CHD; $\overline{\text{CHD}}$, not CHD; LLV, lateral left ventricle; PLV, posterior left ventricle; PS, posterior septum; RV, right ventricle.

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We know of no description of specific pathologic lesions in studies where sudden and non-sudden CHD deaths have been evaluated along with other deaths by pathologists who have had no information concerning age, race, diagnosis or manner of death. Thus, the question of whether sudden CHD deaths differ from non-sudden CHD deaths remains unanswered.

Information concerning the frequency of sudden deaths that are sudden, due to CHD, the proportion of CHD deaths that are sudden, and the specific pathologic findings in CHD deaths, is almost nonexistent for men less than 45 years of age. Because premature CHD is so important from the standpoint of prevention and control, we began an investigation of sudden death, CHD and atherosclerosis by studying 227 young black and white men 25–49 years of age who died and underwent autopsy in New Orleans.

Specifically, we were interested in the following questions:

1. What proportion of sudden unexpected natural deaths is due to CHD? Does the proportion differ when the definition of sudden death is changed?
2. What proportion of CHD deaths is sudden?
3. Are "sudden CHD" deaths really sudden based on pathological evidence of myocardial necrosis and of scars indicating previous myocardial injury?
4. Do sudden CHD deaths differ from non-sudden CHD deaths in frequency of thrombi or intramural hemorrhage of the coronary arteries, or amount of coronary artery atherosclerosis?
5. Do sudden CHD deaths differ from non-sudden CHD deaths in location of myocardial lesions?

Materials and methods

Sample. This report presents results of an analysis of data on 227 autopsies performed in the laboratory of the Coroner of Orleans Parish and in New Orleans hospitals (table 1). This sample represents 72 per cent of all autopsies and 54 per cent of all deaths of black and white New Orleans men aged 25–49 years during the period November 1, 1966, through November 30, 1968. (The major reason for "excluding" autopsies was descrepancies between the age or residence stated at time of autopsy and that on the death cer-

Table 1

Classification of 227 men ages 25–49 years by cause of death, type of death and race, New Orleans, Louisiana, November 1, 1966–November 30, 1968

Cause of* and type of death	Race		
	Black	White	Total
Violent	58	44	102
CHD			
Sudden	7	7	14
Non-Sudden	1	5	6
?CHD			
Sudden	4	3	7
Non-Sudden	3	4	7
$\overline{\text{CHD}}$			
Sudden	19	5	24
Non-Sudden	43	24	67
Total with autopsy specimen	135	92	227
Total deaths	248	173	421

* CHD = coronary heart disease; ?CHD = questionable CHD; $\overline{\text{CHD}}$ = not CHD.

tificate.) These materials were used to test standardized objective methods for independent pathological examination of the myocardium and coronary arteries.

Collection of specimens. The specimens were obtained by technicians from the study laboratory who were present at the time of autopsy to aid the prosecutor. The specimens were taken to the central laboratory and processed according to the study protocol.

Protocol. Details of the methods of study and the precoded forms used for recording findings have been previously published (6). Briefly, hearts are x-rayed, coronary arteries injected with $BaSO_4$-gelatin, and angiograms taken. Coronary arteries are opened, flattened, stained grossly with Sudan IV and sealed in laminated plastic bags. Hearts are sliced at 1.0 cm intervals, x-rayed, fixed and sealed in plastic bags after taking standardized histologic sections. Five pathologists independently examine angiograms, coronary arteries, hearts and histologic slides and record their findings on precoded forms suitable for computer processing. Each specimen is evaluated independently of any other specimen and without reference to case identification or clinical data. A consensus of individual evaluations is used for definitive analyses. Details of the methods for deriving the consensus, which vary for different specimens, are available from the authors upon request.

Definition of terms. Deaths were classified as violent when the coroner's records indicated that they were due to accident, suicide or homicide. All other deaths were classified as natural. Sudden natural deaths were defined as deaths occurring within 24 hours after the onset of acute illness in individuals who had no history of restriction to home, hospital or institution immediately prior to onset of symptoms. Sudden death cases were sub-divided into those occurring within less than one hour, one to 24 hours, or unwitnessed but within 24 hours. Relevant information was obtained from coroner's records, hospital records, physician records and next of kin or close associates of the deceased.

The basis for the assignment of cases to CHD, questionable CHD (?CHD) and not CHD ($\overline{\text{CHD}}$) was the consensus of five pathologists with all available information—objective myocardial and coronary artery evaluation, other autopsy data and available clinical data.

The findings based on these 227 cases are not meant to provide definitive evidence on the pathogenesis of sudden death and CHD. They have been used as the basis for hypotheses for further testing in an ongoing study of deceased men in New Orleans.

Results

In all, 102 of the 227 deaths were classified as violent deaths and 125 were classified as natural deaths. Eighty of the 125 natural deaths did not occur suddenly (non-sudden deaths). Twenty-nine of the 45 sudden deaths occurred almost instantaneously, 12 were found dead (unwitnessed death) and four survived for several hours (table 2).

The 125 sudden and non-sudden natural deaths were classified as CHD, ?CHD, and $\overline{\text{CHD}}$. Only 14 (31 per cent) of the 45 sudden deaths cases were unquestionably due to CHD, seven (16 per cent) were ?CHD, while over one half (24 cases or 53 per cent), were definitely not due to CHD according to the consensus of the five pathologists. Sixteen of the 24 sudden deaths not due to CHD had sufficient evidence to establish the causes of death as alcoholism, pneumonia, acute narcotism, carcinoma, hypertensive cardiovascular disease, heart disease other than CHD, or encephalomalacia. The eight remaining sudden death cases in this group had uncertain causes even after autopsy, but had no evidence of CHD.

Only six (7 per cent) of 80 non-sudden natural deaths were classified as CHD

TABLE 2

Classification of 125 natural deaths of men ages 25–49 years by cause of death and by time interval from the onset of symptoms to death, New Orleans, Louisiana, November 1, 1966–November 30, 1968

Cause of death*	Sudden deaths (≤24 hrs)					Non-sudden deaths (>24 hrs)		All natural deaths	
	No. <1 hr	No. 1–24 hrs	No. unwitnessed	Sub-total No.	Sub-total %	No.	%	No.	%
CHD	8	3	3	14	31	6	7	20	16
?CHD	7			7	16	7	9	14	11
$\overline{\text{CHD}}$	14	1	9	24†	53	67	84	91	73
TOTAL	29	4	12	45	100	80	100	125	100

* CHD = coronary heart disease; ?CHD = questionable CHD; $\overline{\text{CHD}}$ = not CHD.

† These cases included the following causes of death: alcoholism, pneumonia, narcotism, carcinoma of lung, hypertensive cardiovascular disease, encephalomalacia and uncertain other causes.

and seven (9 per cent) as ?CHD. Sixty-five of the 67 $\overline{\text{CHD}}$ had well documented causes of death and two cases had an uncertain cause of death without evidence of CHD. The proportion of sudden deaths due to CHD (31 per cent) was significantly greater than the proportion of non-sudden deaths due to CHD (7 per cent), $t = 3.22; p \leq 0.005$. Twenty-nine (64 per cent) of the 45 cases classified as sudden death died within one hour of developing symptoms (table 2). Of 20 CHD deaths in this young age group, 14 (70 per cent) were classified as sudden deaths, and eight (40 per cent) occurred less than one hour after the onset of symptoms. Similarly, half of the 14 ?CHD deaths were sudden and all occurred within one hour of the onset of symptoms. On the other hand, only 24 (26 per cent) of 91 $\overline{\text{CHD}}$ deaths were sudden and 14 (15 per cent) occurred within one hour. There was no significant difference ($t = 0.68$) in the proportion of deaths due to CHD in the group dying within one hour (eight out of 29 deaths, or 28 per cent) and those dying in 1–24 hours plus the unwitnessed deaths (six out of 16 deaths, or 38 per cent).

Coronary atherosclerosis. The mean per cent of intimal surface involved with raised atherosclerotic lesions in the three main branches of the coronary arteries (table 3) was high and almost identical for CHD deaths classified as either sudden (55 per cent) or non-sudden (54 per cent); the ?CHD sudden and non-sudden deaths had average raised lesions of 46 and 36 per cent. In contrast, the $\overline{\text{CHD}}$ natural sudden and non-sudden deaths, and the violent deaths showed less raised lesion involvement averaging 6, 11 and 6 per cent, respectively.

Stenosis and occlusion. The frequency of stenosis and occlusion determined from coronary angiograms and inspection of the open flattened vessels is also shown in table 3. Seven of the 14 sudden CHD deaths had occlusions, and seven cases had stenosis of at least one major coronary branch but no areas of complete occlusion. Thirteen of these 14 cases had stenosis and/or occlusion of multiple vessels. Three of the six non-sudden CHD deaths had occlusion, and three had stenosis only. Four of these six cases had stenosis and/or occlusion of multiple vessels. Of the seven ?CHD sudden deaths, two had occlusion and five had stenosis only. The seven questionable CHD non-sudden deaths had stenosis without occlusion. None of the $\overline{\text{CHD}}$ deaths, whether sudden or non-sudden, had occlusion, but one of 24 sudden and 17 of 67 non-sudden deaths in this category had areas of stenosis. Two of the 102 violent deaths had occlusion and 21 had areas of stenosis.

Thrombosis and hemorrhage. Thrombot-

TABLE 3

Classification of 227 autopsied men ages 25–49 years by cause of death, type of death and type of coronary artery lesions based on consensus of five pathologists. New Orleans, Louisiana, November 1, 1966–November 30, 1968

Coronary artery lesions, by characteristics	Cause of* and type of death							
	CHD		?CHD		$\overline{\text{CHD}}$		Violent death (N = 102)	Total (N = 227)
	Sudden (N = 14)	Non-sudden (N = 6)	Sudden (N = 7)	Non-sudden (N = 7)	Sudden (N = 24)	Non-sudden (N = 67)		
Occlusion	7	3	2				2	14
Stenosis only	7	3	5	7	1	17	21	61
Stenosis or occlusion	14	6	7	7	1	17	23	75
Thrombus								
Occlusive	4	3						7
Non-occlusive	5	1		1		1		8
Hemorrhage								
In areas of occlusion	4	2						6
In areas of stenosis	1	1		2		2		6
Thrombus or hemorrhage†	11	4		3	1	3	2	24
Mean % raised atherosclerotic lesions in 3 coronary arteries	55%	54%	46%	36%	6%	11%	6%	14%

* CHD = coronary heart disease; ?CHD = questionable CHD; $\overline{\text{CHD}}$ = not CHD.

† A case may have thrombus and hemorrhage therefore total does not equal the sum of the two.

ic material was present in nine of the 14 CHD sudden deaths; hemorrhage in a plaque was found in five cases. Three cases had neither hemorrhage nor thrombus. Thrombotic material was present in four of the six CHD non-sudden deaths, and three of the four had hemorrhage into a plaque or thrombus. Neither thrombus nor hemorrhage was detected in the seven ?CHD sudden deaths. Of the seven ?CHD non-sudden deaths, one had a thrombus, two had hemorrhage in a plaque and four cases had neither hemorrhage nor thrombus. No thrombi were detected in the 24 $\overline{\text{CHD}}$ sudden deaths and only one had hemorrhage. Of the 67 $\overline{\text{CHD}}$ non-sudden deaths one had thrombotic material while two had hemorrhage. Twenty-three of the 24 sudden deaths and 64 of the 67 non-sudden deaths not due to CHD had neither hemorrhage nor thrombus and only two of 102 violent deaths had hemorrhage.

Frequency of myocardial lesions. The consensus grading of the five pathologists (table 4) revealed that all of the sudden CHD deaths, three of seven ?CHD and three of 24 $\overline{\text{CHD}}$ deaths had large ($\geq$1 cm) myocardial lesions. Thirteen of 14 sudden CHD deaths had recent myocardial lesions (necrosis) detected by gross and microscopic evaluation of coded specimens. Ten of these 14 sudden CHD deaths also had myocardial scars representing obvious previous episodes of injury to the myocardium, although none of these cases had a previous history of CHD. The consensus grading also showed that five of the six non-sudden CHD deaths, three of seven ?CHD and 13 of the 67 $\overline{\text{CHD}}$ deaths had large myocardial lesions. Four of the five non-sudden CHD deaths with large myocardial lesions had necrosis. The myocardial lesions found in men not dying of CHD were not the result of coronary stenosis or occlusion and were usually associated with other basic disease processes or terminal complications such as sickle cell disease, systemic lupus erythematosus, malignancy, rheumatic heart disease, hypertensive and cerebral vascular disease, thrombo-embolism, and gastrointestinal hemorrhage. The necrosis in four violent deaths was probably the result of trauma or severe shock during the terminal episode.

Location of myocardial lesions. The location of large myocardial lesions among CHD, ?CHD and $\overline{\text{CHD}}$ sudden and non-sudden deaths is illustrated in figure 1. All the myocardial lesions of the 14 CHD sudden deaths involved the posterior septum, the subendocardium and the apex. All the large myocardial lesions among the five CHD non-sudden deaths involved the base and the center of the heart. The myocardial lesions in cases dying of CHD were usually larger than lesions among natural deaths not due to CHD. Three of the 24 $\overline{\text{CHD}}$ sudden death cases had large myocardial lesions similar to the 13 $\overline{\text{CHD}}$ non-sudden deaths. The small number of cases in these comparisons, however, makes interpretation difficult.

Discussion

Classification of deaths. Among our cases, whether sudden death is rigorously defined as occurring in one hour, or less rigorously as occurring within 24 hours, a surprisingly low proportion of sudden deaths is the result of CHD. Even if all the questionable CHD deaths are considered to be CHD deaths, only 47 per cent of the sudden deaths in this young population can be assigned to CHD in sharp contrast with the 80–90 per cent commonly assumed (5). In this study, most of the cases not assigned to CHD had another well-documented cause of death.

A study of sudden death in Baltimore (7) has also shown a low per cent of sudden deaths assigned to atherosclerotic heart disease. Alcoholism and narcotism, which are considered to be more common in large metropolitan areas and in young age groups account for a large portion of the sudden deaths not related to CHD.

TABLE 4

Classification of 227 autopsied men ages 25–49 years by type of death, cause of death and stage of myocardial lesions based on consensus of five pathologists, New Orleans, Louisiana, November 1, 1966–November 30, 1968

Stage of myocardial lesion	Type of and cause of* death							
	Sudden death			Non-sudden deaths			Violent death (N = 102)	Total (N = 227)
	CHD (N = 14)	?CHD (N = 7)	$\overline{CHD}$ (N = 24)	CHD (N = 6)	?CHD (N = 7)	$\overline{CHD}$ (N = 67)		
Large lesions (≥1 cm)	14	3	3†	5	3	13†	7	48
Recent only	4	2			1	2	3	12
Healing only						1		1
Recent, healing, scar	9	1		3		3		16
Recent, healing				1	1	3	1	6
Recent, scar			1					1
Healing, scar	1		2	1	1	4	3	12
Small lesions only (<1 cm)		3	5		1	15	15	39

* *CHD = coronary heart disease; ?CHD = questionable CHD; $\overline{CHD}$ = not CHD.*

† These cases included the following disease processes or terminal complications: sickle cell disease, systemic lupus erythematosus, malignancy, rheumatic heart disease, hypertensive and cerebral vascular disease, thrombo-embolism and gastrointestinal hemorrhage.

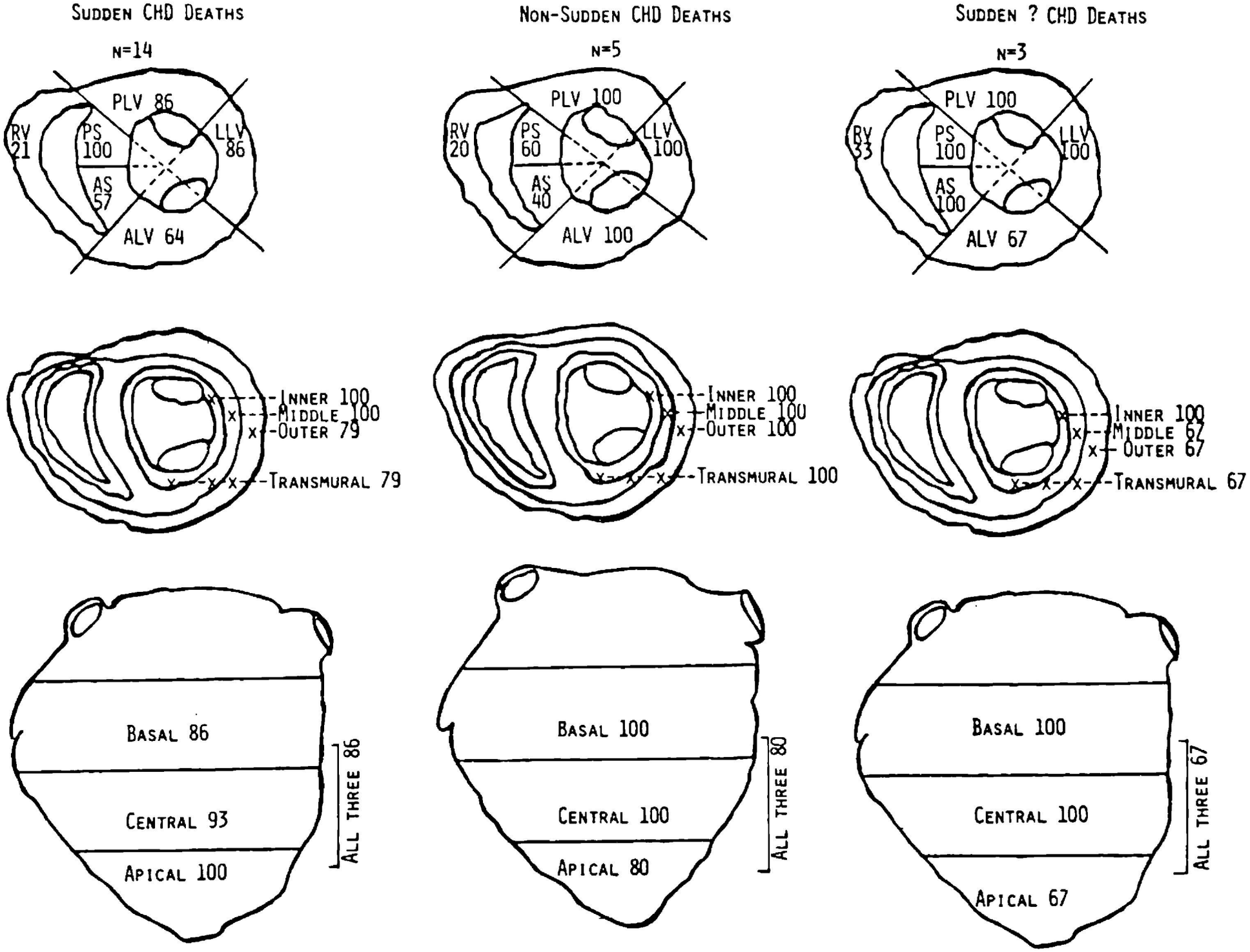
Under such conditions, the use of the sudden death category as an index of CHD in epidemiologic studies would obscure any effect under study because of the inclusion of many $\overline{CHD}$ deaths. This problem may be particularly crucial in community-wide investigations in large metropolitan areas.

On the other hand, in this study the proportion of CHD deaths which occurred suddenly (70 per cent) is higher than reported in many other studies (1–3), and even when all of the ?CHD cases are included, 62 per cent of the CHD deaths are sudden. This high proportion of sudden CHD deaths may be due in part to the younger age of this study population.

Coronary atherosclerosis, thrombosis and hemorrhage. The finding of coronary thrombi in such a large proportion of cases of CHD sudden deaths (nine out of 14 cases, or 64 per cent) is also counter to the data of others (8–11). These thrombi were identified by gross inspection of the opened arteries and confirmed histologically. Perhaps the sudden deaths classified as ?CHD in this study, with all seven cases lacking coronary thrombi, account for this apparent difference. If they were included in the CHD category, the proportion of cases with thrombi would then be nine of 21 (43 per cent).

There seemed to be no difference in the sudden CHD deaths and those not occurring suddenly in amount of coronary atherosclerosis, frequency of thrombi or occlusion, or presence of recent myocardial lesions.

Frequency of myocardial lesions. Many of the sudden CHD deaths had well established myocardial infarcts and advanced, complicated coronary artery lesions, although none of the 14 cases had a previous history of CHD. These myocardial and coronary artery findings, based on observations made by five pathologists without knowledge of the circumstances of death, are contrary to prevailing impression and findings (8–13). Our findings, however,


SUDDEN CHD DEATHS
N=14
PLV 86
RV 21
PS 100
LLV 86
AS 57
ALV 64
INNER 100
MIDDLE 100
OUTER 79
TRANSMURAL 79
BASAL 86
CENTRAL 93
APICAL 100
ALL THREE 86
NON-SUDDEN CHD DEATHS
N=5
PLV 100
RV 20
PS 60
LLV 100
AS 40
ALV 100
INNER 100
MIDDLE 100
OUTER 100
TRANSMURAL 100
BASAL 100
CENTRAL 100
APICAL 80
ALL THREE 80
SUDDEN ? CHD DEATHS
N=3
PLV 100
RV 33
PS 100
LLV 100
AS 100
ALV 67
INNER 100
MIDDLE 67
OUTER 67
TRANSMURAL 67
BASAL 100
CENTRAL 100
APICAL 67
ALL THREE 67

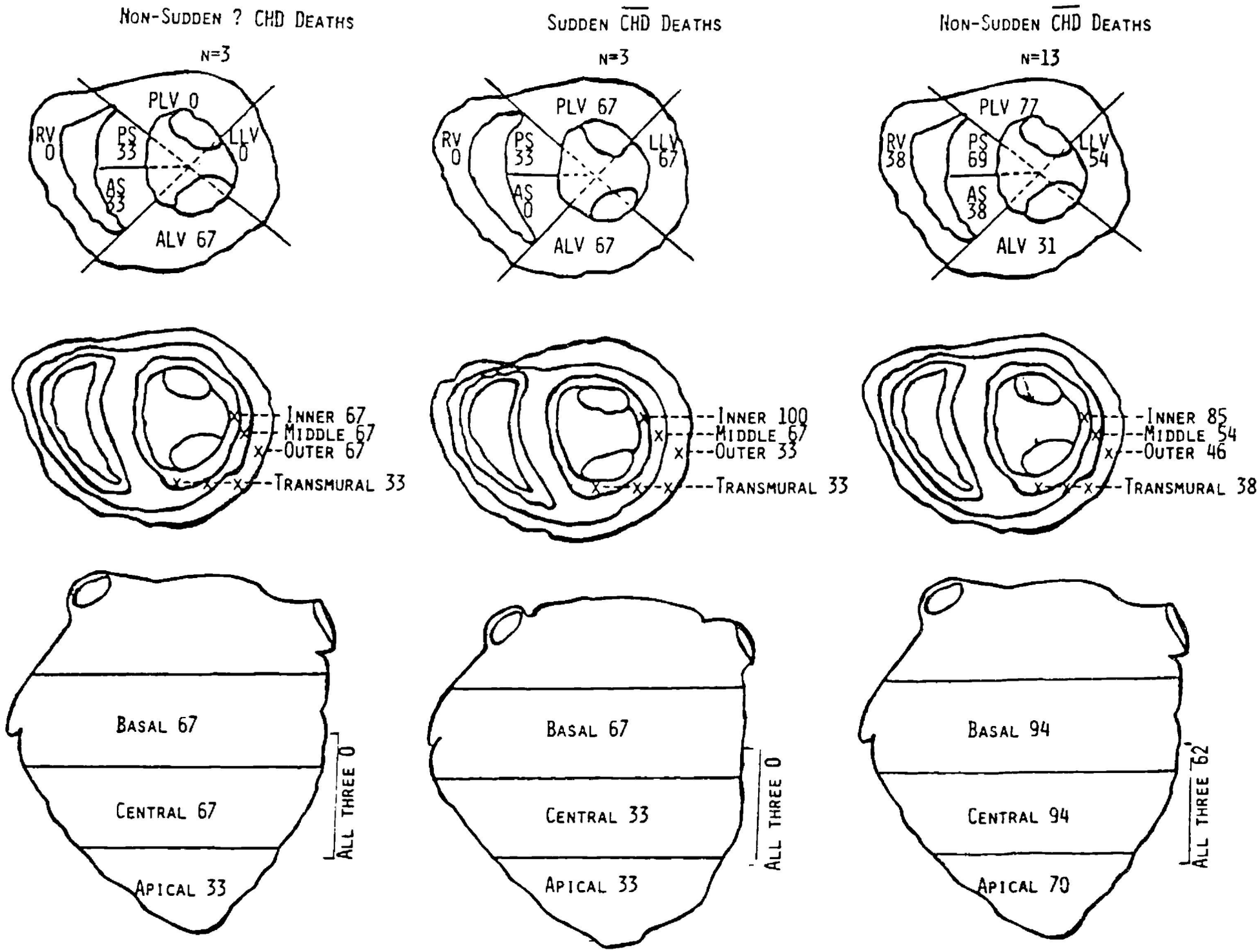


FIGURE 1. Location of large myocardial lesions (≥1 cm) in natural deaths with large lesions by cause and type of death: Proportion of cases involving each area of myocardium among autopsied men ages 25–49 years, New Orleans, Louisiana, November 1, 1966–November 30, 1968

agree with those of Scott and Briggs (14).

The high prevalence of large myocardial lesions in the deaths not due to CHD (17 per cent) and the violent deaths (7 per cent) is undoubtedly the result of the method of independent-blind grading by the five pathologists. Conditions have been identified which for the most part satisfactorily account for these myocardial lesions. The grossly visible lesions did not always have an easily recognized uniform pattern. In addition to the "usual" myocardial changes of CHD, several unusual lesions encountered in the myocardium when examined were: necrosis in the subendocardial zone of muscle throughout virtually the entire left ventricle; scattered small lesions of various sizes and ages, generally in a markedly hypertrophied left ventricle; recent necrosis and hemorrhage selectively within the conduction system as it descends along the subendocardial zone of the left ventricular septal wall; and recent or old necrosis exclusively within papillary muscles. Some of these cases had no significant atherosclerosis in the major coronary branches as revealed in angiograms and dissected coronary arteries. Lesions which resembled typical infarcts or small ischemic lesions were sometimes seen in the absence of severe coronary artery disease. These findings further emphasize that not every large myocardial lesion is due to coronary atherosclerosis or thrombosis. The relationship of these myocardial lesions to coronary atherosclerosis, hypertension or other variables is still under investigation. The magnitude of their importance in the overall impact of heart disease has not yet been determined. Cases of this kind can be studied best at autopsy. Conclusions derived chiefly from attention to vital statistics or clinical information may easily fail to distinguish such cases from those of true atherosclerotic heart disease.

Location of myocardial lesions. The only difference observed between the sudden and non-sudden CHD deaths was the topographical location of myocardial lesions. The sudden CHD deaths had a higher frequency of lesions which involved the posterior septum, the subendocardium, and the apex. The non-sudden CHD deaths more frequently involved all of the left ventricle except the septum, were transmural and involved the base and center of the heart. This difference in location may be one of the factors responsible for determining whether a CHD death is sudden or non-sudden, since part of the conductive system is located in the septum. This difference in pattern of location of myocardial lesions between the sudden CHD deaths and non-sudden CHD deaths is based on a small number of cases and merits careful consideration in the definitive study.

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