Prehospital sudden death from ischaemic heart disease *A postmortem study*

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SUMMARY A detailed postmortem study of the myocardium and coronary arteries was performed on 114 men and 37 women who died suddenly outside hospital. Data on the disease history and certain risk factors were collected through an 'Ischaemic Heart Disease Register'.

Men and women did not differ with respect to history of previous symptoms of ischaemic heart disease or necropsy findings. A history of hypertension and the previous use of digitalis or diuretics tended to be more frequent in women than in men but current smoking was more common in men (69%) than in women (43%). Of the subjects in whom necropsy revealed an old myocardial infarction, 86 per cent had a history of symptomatic ischaemic heart disease but a previous myocardial infarction was known in only 42 per cent of them. A definite recent myocardial infarction was found in 26 per cent and an early infarction in 51 per cent. About half of the patients with a definite recent or an early myocardial infarction, and half of those with no recent myocardial infarct, died at once. Fourteen per cent of the subjects died after symptoms lasting 2 hours or more.

Triple vessel disease in the coronary arteries was found in 56 per cent of cases, double vessel disease in 31 per cent, and single vessel disease in 11 per cent. Aortic valvular stenosis or a history of hypertension was present in 3 cases with no obvious coronary disease. Suddenness of death did not correlate with severity of coronary obstruction. Arterial lesions were more severe in patients with an old myocardial infarction than in those without this. The more severe the coronary artery disease, the more likely a definite recent myocardial infarction was to be present. The mildest disease was found in patients with no detectable recent myocardial infarction.

No significant differences were found between patients with symptomatic and those with silent but definite myocardial infarction. Heavy smoking was most common in patients with an early recent myocardial infarction. Patients dying without warning and with no recent infarction (those with a primary arrhythmia) had severe coronary artery disease less frequently than those dying with a recent infarction. None of the former had been heavy smokers. They had, however, large scarred hearts, and a high incidence of previous hypertension, of ischaemic heart disease, and of treatment with digitalis or diuretics.

Sudden death is a major problem in ischaemic heart disease. Its aetiology is difficult to study, for without clinical data the diagnosis has to be based mainly on information at necropsy. The establishment of Ischaemic Heart Disease Registers according to the guidelines suggested by the World Health Organization has permitted the identification of all deaths

from ischaemic heart disease within a defined community (Romo, 1973). Reliable collection of data concerning the fatal attack and the previous illnesses of a patient is also feasible (Siltanen, 1972).

The diagnosis of death from ischaemic heart disease is easy if an acute myocardial infarction has developed to the stage of a necrotic lesion. Difficulties arise when death occurs within a few hours of the onset of the attack, since uniform criteria have not been accepted for the pathological diagnosis of acute myocardial infarction. In order to resolve this problem a co-operative study by the WHO has been carried out. The preliminary results

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(World Health Organization, 1973), in which various methods of study previously proposed (World Health Organization, 1970) were tested and evaluated, indicated outlines for the pathological diagnosis of sudden death from myocardial infarction.

The present study was sponsored by the Ischaemic Heart Disease Register of Helsinki. Data concerning sudden death have been previously published in a report on all registered (Romo, 1973) and all fatal cases of ischaemic heart disease (Rissanen *et al.*, 1975). This paper deals with the subgroup of sudden deaths in which a postmortem study was performed using the methods proposed by the WHO, focusing the main attention on the presence of recent myocardial infarction and the prevalence of different coronary lesions or their combinations in relation to the course of the fatal attack.

Subjects and methods

Altogether, 365 deaths from ischaemic heart disease, outside hospital, were registered during a period of 12 months in Helsinki. Elderly subjects (aged 65 years or over) were studied for a period of 3 months only. A medico-legal necropsy using a special technique of examination of the heart was performed in 213 registered cases and those in this study consisted of 151 in whom the fatal attack was witnessed, the circumstances of the attack well documented, and in whom death occurred within 24 hours of the onset of the attack. There were 114 men aged between 31 and 83 years, and 37 women aged between 44 and 90 years.

DATA COLLECTION

Information about the fatal attack and previous illnesses was obtained from hospital records and from relatives. The principles of data collection have been described in detail in an earlier paper (Romo, 1973). Previous angina pectoris, myocardial infarction, dyspnoea on exertion, and hypertension were recorded using definitions by the World Health Organization (1976). Premonitory symptoms in the 28 days preceding death were noted. Medical care before death was assessed from the drugs taken by the patients, particularly digitalis, diuretics, and antihypertensives.

The patients were allocated into 4 categories according to smoking habits: (1) heavy smokers, that is those smoking 25 or more cigarettes a day; (2) other cigarette smokers; (3) ex-smokers, that is those who had given up smoking at least three months before death; and (4) non-smokers.

NECROPSY DATA

Medico-legal necropsies were performed at the Department of Forensic Medicine, University of Helsinki. The principles outlined by the World Health Organization (1970, 1973) were applied to the examination of the hearts. All necropsies were carried out by the same person (V.R.).

A double contrast modification of the postmortem coronary angiogram was developed to show changes in the coronary arteries (Rissanen, 1970), and in addition, they were opened longitudinally and examined carefully. The degree of coronary stenosis was estimated by these procedures. The right coronary artery (RC), left anterior descending coronary artery (LAD), and left circumflex coronary artery (LC) were each graded according to the maximum obstruction, using three scores: Score 0 = no stenosis or less than 50 per cent stenosis; Score 1 = a 50 per cent stenosis or more but no occlusion; Score 2 = occlusion.

The scores of the three vessels were added to give a stenosis score for the whole coronary arterial tree ranging from 0 to 6. A stenosis in the main trunk of the left coronary artery was classified as double vessel disease.

After angiography the heart was sliced from the apex to the base. The slices were stained by a macroscopic histochemical method (Nachlas and Shnitka, 1963) to show the infarcted areas, from which myocardial blocks for histological study were taken. When macroscopic examination was negative, blocks were taken from the anterior and posterior walls of the left ventricle and from the interventricular septum. Haematoxylin-eosin and phosphotungstic acid haematoxylin were used to stain sections. The principles of the WHO co-operative study (World Health Organization, 1973) were applied in the histological diagnosis of a recent infarction. The two stages of a recent myocardial infarction were defined as follows:

—*early myocardial infarction*: Macroscopic discoloration, pallor, or softening, and/or at least two of the following microscopic changes of the myocardium: (1) cytoplasmic hypereosinophilia, coagulation, clumping, alteration of striation (contraction bands), (2) loss of nuclei in the myofibres, (3) margination of polymorphonuclear leucocytes, (4) leucocytosis, or (5) oedema.

—*definite myocardial infarction*: Macroscopic necrosis, softening, or discoloration of the myocardium and microscopic infiltration of polymorphonuclear leucocytes in the area of necrosis with or without macrophages.

The recognition of a recent myocardial infarction was ultimately based on findings of the histological examination. A scar of 0.5 cm or more in diameter

Table 1 Duration of fatal attack and history of previous angina pectoris, myocardial infarction, dyspnoea, and premonitory symptoms: distribution according to age and sex

Age	No. of cases	Suddenness of death			Angina pectoris			Infarction			Dyspnoea			Premonitory symptoms		
		Inst.	< 2 h	> 2 h	Data available	No.	%	Data available	No.	%	Data available	No.	%	Data available	No.	%
Men																
30-44	9	5	4	—	8	1	12.5	8	1	12.5	7	6	86.0	8	6	75.0
45-54	30	12	11	7	28	17	61.0	28	8	29.0	24	14	58.0	26	18	69.0
55-64	56	32	17	7	51	36	71.0	51	18	35.0	52	37	71.0	52	31	60.0
65 or over	19	11	8	—	14	7	50.0	16	4	25.0	14	10	71.0	12	4	33.0
Total	114	60	40	14	101	61	61.0	103	31	30.0	97	67	69.0	98	59	60.0
Women																
40-54	8	7	—	1	6	3	50.0	7	1	14.0	6	5	83.0	6	4	67.0
55-64	19	10	6	3	16	12	75.0	18	6	33.0	16	12	75.0	16	9	56.0
65 or over	10	1	6	3	9	2	22.0	10	1	10.0	8	4	50.0	8	3	37.5
Total	37	18	12	7	31	17	55.0	35	8	23.0	30	21	70.0	30	16	53.0

was regarded as an old healed infarction (Guzman *et al.*, 1968).

After blood clots had been removed from the chambers, the heart was weighed. The presence of a fresh coronary thrombus was estimated visually from angiograms and/or the longitudinally opened coronary arteries but without histology.

Results

HISTORY OF FATAL ATTACK

According to the interviewers of the Ischaemic Heart Disease Register, 53 per cent of men and 49 per cent of women of this series died instantaneously at the witnessed fatal attack without showing prior symptoms (Table 1). Only 21 patients (14%) had symptoms for longer than 2 hours. In the remaining 52 (34%) death occurred within this period but even in the majority of these the duration of the episode was only a few minutes.

Eighty patients (53%) died at home, 32 (21%) on the way to hospital or at the outpatient department, and the remaining 39 elsewhere.

DISEASE HISTORY AND NECROPSY DATA

Previous symptoms (angina pectoris, dyspnoea, other premonitory symptoms) or a history of a previous infarction did not show any relation to age or sex (Table 1). The necropsy disclosed no clear influence of age or sex in the incidence of recent or old myocardial infarction, coronary occlusion or thrombosis, the range of the stenosis score, or the heart weight (Table 2). A history of hypertension and previous use of digitalis or diuretics was slightly more common in women than men (Table 3). This, however, was not statistically significant. Of the men, 69 per cent, and of the women 43 per cent had been current smokers (Table 3).

In all age groups the necropsy finding of old myocardial infarction (Table 2) was much more

Table 2 Incidence of recent and old myocardial infarction, coronary occlusion, and thrombosis, and median (M) and range (R) values of stenosis score and heart weight, according to age and sex

Age	No. of cases	Recent infarction				Old infarction		Coronary occlusion		Coronary thrombosis		Stenosis score R(M)R	Heart weight R(M)R
		Definite No.	Initial No.	Total No.	%	No.	%	No.	%	No.	%		
Men													
30-44	9	1	6	7	78	5	56	7	78	6	67	1(3)6	410(440)630
45-54	30	12	15	27	90	17	57	24	80	17	57	1(4)6	350(470)880
55-64	56	11	30	41	73	40	71	45	80	23	41	0(4)6	340(500)820
65 or over	19	5	7	12	63	13	68	14	74	9	47	2(4)6	380(470)880
Total	114	29	58	87	76	75	66	90	79	55	48	0(4)6	340(490)880
Women													
40-54	8	2	4	6	75	2	25	2	25	2	25	0(1)5	310(455)610
55-64	19	6	8	14	74	12	63	14	74	5	26	1(4)5	320(450)620
65 or over	10	4	5	9	90	3	30	9	90	5	50	3(4)5	430(465)830
Total	37	12	17	29	78	17	46	25	68	12	32	0(4)5	310(455)830

Table 3 *History of hypertension, use of digitalis, and/or diuretics, and history of smoking habits in whole series*

	Men (total 114)			Women (total 37)		
	Data avail- able	No. of cases	%	Data avail- able	No. of cases	%
History of hypertension	92	28	30	29	14	48
use of digitalis or diuretics	90	32	36	28	14	50
Smoking habits	87			26		
heavy smokers		28	32		2	8
other smokers		30	35		9	35
ex-smokers		15	17		3	12
non-smokers		14	16		12	46

frequent than could be expected on the basis of the Ischaemic Heart Disease Register interviews (Table 1). A previous episode of clinical myocardial infarction was known about in only 38 of the 91 patients (42%) with a myocardial scar found at necropsy. Of the 53 patients with a scar but with no history of previous infarction, 40 had had angina pectoris or dyspnoea on exertion. The remaining 14 per cent of the subjects with an old infarction (12 men, 1 woman) had no history of clinical heart disease. Two of them had had hypertension, and 7 had been heavy smokers.

FATAL ATTACK IN RELATION TO A RECENT ISCHAEMIC MYOCARDIAL LESION

Necropsy showed a recent myocardial infarction in 77 per cent of all cases in the series. A definite

myocardial infarction was present in 26 per cent and an early myocardial infarction in 51 per cent. A macroscopic recent myocardial lesion, if present, was nearly always limited to the subendocardial zone of the left ventricle. Anomalous crossband formations were characteristic of altered myofibres on microscopy in cases of early myocardial infarction.

The clinical suddenness of death did not show any clear relation to the stage of a recent ischaemic myocardial lesion. In about half the patients with a definite or an early infarction, as well as of those with no recent ischaemic lesion found at necropsy, the fatal attack ended abruptly (Table 4). Symptoms lasting over two hours were, however, more frequent in patients with a definite myocardial infarction.

DEGREE OF OBSTRUCTIVE CORONARY ARTERY DISEASE

Numerous combinations of obstructive changes in the three coronary arteries were found. The 9 most

Table 4 *Suddenness of death of persons with definite or initial myocardial infarction found at necropsy, and of those with no recent myocardial lesion*

Infarction	No. of cases	Instantaneous < 2 hours death		< 2 hours		> 2 hours	
		No.	%	No.	%	No.	%
Definite*	40	20	50	8	20	12	30
Initial	76	40	53	30	39	6	8
No recent MI	35	18	51	14	40	3	9

*Rupture in 5 cases (4 instantaneous deaths, 1 with symptoms over 2 hours).

Table 5 *The most common patterns of coronary stenosis in the total series, and in cases with and without an old infarction. RC = right coronary artery, LAD = left anterior descending coronary artery, LC = left circumflex coronary artery*

Total series (151 cases)				With an old infarction (91 cases)				No old infarction (60 cases)			
No. of cases	RC	LAD	LC	No. of cases	RC	LAD	LC	No. of cases	RC	LAD	LC
21	+	-	-	16	+	-	-	6	-	+	-
15	-	+	-	12	+	+	+	5	+	-	-
12	+	+	+	9	-	+	-	5	-	-	+
10	-	-	-	8	+	+	-	5	-	+	-
9	+	-	○	6	-	+	+	5	+	-	○
8	+	+	-	6	-	-	+	5	-	-	○
8	-	+	+	4	+	-	+	5	-	○	○
8	-	+	○	4	+	+	○	4	-	-	○
8	-	-	○	4	○	+	○	4	○	-	○
				4	+	-	○				
≤6	18 different combinations			<3	8 different combinations			<2	12 different combinations		

+ = an occlusion,
- = a 50% stenosis,
○ = no severe stenosis.

Table 6 Percentages of triple, double, or single vessel disease or no coronary stenosis in present series and in 4 clinical series of patients with ischaemic heart disease studied by coronary arteriography

Series	Triple %	Double %	Single %	No disease %
Present series				
Total	56	31	11	2
With old myocardial infarction	69	28	2	1
With no old myocardial infarction	37	37	23	3
Clinical series				
Webster <i>et al.</i> (1974)	38	38	24	
Burggraf and Parker (1975)	31	41	28	
Fuster <i>et al.</i> (1975)				
Angina pectoris	31	37	24	
Subendocardial myocardial infarction	42	40	13	
Transmural myocardial infarction	38	30	27	
Hekali (1976)	31	41	28	

common patterns of stenosis constituted two-thirds of all cases (Table 5). A severe stenosis in the main left coronary artery was found in 7 patients. Only 3 had no coronary stenosis of any severity. There were distinct differences in the pattern of disease between patients with an old infarction and those without one. In the former, triple vessel disease was more common and single vessel disease less common (Table 6). Coronary occlusion was found in 87 per cent of patients with a myocardial scar at necropsy and in 58 per cent of those with no scar. An occlusion in all three coronaries was found in 12 cases (8%), each patient with an old myocardial infarct.

The obstructive changes in the coronary arteries were no different in patients who died suddenly than in those who died after a period of symptoms, whether under or over two hours (Fig. 1). The three patients with no major stenosis all died without warning.

Fig. 1 Stenosis scores in sudden deaths and in cases with a fatal attack of under or over 2 hours in duration. The figures above the columns indicate the number of cases.

Fig. 2 Stenosis scores in cases with a definite or an early myocardial infarction and in cases of sudden death with no recent myocardial lesion.

Whether or not an old infarction had occurred coronary artery disease was most severe in patients in whom a definite recent myocardial infarction was found at necropsy, and least severe in those with no recent myocardial lesion (Fig. 2).

Of the 32 patients, 29 (91%) with the highest stenosis scores of 5 and 6 had a history of previous myocardial infarction, angina pectoris, or dyspnoea. In 30 of them a myocardial scar was found at necropsy. Low scores of 0 and 1, that is no coronary stenosis or a 50 per cent stenosis in one vessel, were found in 10 per cent of the cases; these coronary patterns were rare in subjects with a recent infarction (5%) but commoner in those without one (23%) ($P < 0.01$). The low score values were also less frequent in subjects with an old infarction (3%) than in those without one (20%) ($P < 0.001$). They were commoner in women (22%) than in men (7%) ($P < 0.01$). The highest frequency of scores 0 and 1 was found in patients with neither recent nor old myocardial lesions (44%).

Table 7 *History and necropsy findings in cases of 'late', 'early', 'silent' acute myocardial infarction and 'primary arrhythmia' (see text for definitions)*

	Late AMI			Early AMI			Silent AMI			Primary arrhythmia		
No. of cases, total	12			30			20			18		
Mean age (y)	58.5			58.8			62.7			56.3		
Age range	46-90			41-84			44-86			40-65		
Men/Women	8/4			23/7			14/6			14/4		
	Data available	No. of cases	%	Data available	No. of cases	%	Data available	No. of cases	%	Data available	No. of cases	%
History of												
Angina pectoris	11	9	82	26	15	58	18	10	56	17	14	82
Myocardial infarction	10	3	30	27	6	22	18	6	33	17	8	47
Dyspnoea	11	8	73	24	16	67	17	14	82	16	13	81
Prodromal symptoms	10	8	80	24	12	50	17	10	59	18	11	61
Use of digitalis or diuretics	8	3	38	26	5	19	16	7	44	17	13	77
Hypertension	11	4	36	27	7	26	17	6	35	18	9	50
Necropsy findings of												
Old myocardial infarction	12	8	67	30	19	63	20	12	60	18	13	72
Stenosis score ≥ 4	12	10	83	30	17	57	20	17	85	18	5	28
Cardiac hypertrophy*	12	8	67	30	9	30	20	14	70	18	13	72
History of myocardial infarction/myoc. scar												
	3/6			6/18			6/11			8/13		

* ≥ 500 g in men, ≥ 450 g in women.**ACUTE MYOCARDIAL INFARCTION—PRIMARY ARRHYTHMIA**

The correlation between clinical symptoms during the fatal attack and ischaemic myocardial lesions seen after death was poor. Accordingly the following subgroups were studied: (1) 'late' deaths from acute myocardial infarction: patients whose fatal attack lasted longer than 2 hours and in whom the ischaemic myocardial lesion had developed to the stage of a definite infarction; (2) 'silent' deaths from acute myocardial infarction: patients who died suddenly but in whom a definite myocardial infarction was found at necropsy; (3) 'early' deaths from acute myocardial infarction: patients who had symptoms for less than 2 hours and in whom

necropsy disclosed an early ischaemic myocardial lesion; (4) deaths from 'primary arrhythmia': patients who died suddenly with no recent myocardial lesion at necropsy.

No significant differences were found in the incidence of symptomatic ischaemic heart disease, hypertension, or the use of drugs in the 'late', 'silent', and 'early' death groups (Table 7), but subjects in the early death group were less likely to have big hearts (≥ 500 g in men, ≥ 450 g in women) than those in the other two ($P < 0.01$) and severe coronary obstruction was also less common ($P < 0.05$). This group showed the highest incidence of current smokers, and of heavy smokers (Table 8).

There were certain differences between the

Table 8 *History of smoking habits in cases of 'late', 'early', and 'silent' acute myocardial infarction and 'primary arrhythmia' (see text for definitions)*

	Late AMI 9		Early AMI 19		Silent AMI 15		Primary arrhythmias 15	
Data available	No. of cases	%	No. of cases	%	No. of cases	%	No. of cases	%
Current smokers	6	67†	17	90*	10	67†	4	27
Heavy smokers	4		12		4		0	
Other smokers	2		5		6		4	
Ex-smokers	1	11	1	5	2	13	5	33
Non-smokers	2	22	1	5	3	20	6	40

*Significant difference between early AMI and primary arrhythmia; $P < 0.001$.†Significant difference between late + silent AMI and primary arrhythmia; $P < 0.05$

subjects dying from acute myocardial infarction and those in whom death probably resulted from a 'primary arrhythmia', that is those who died suddenly and had no acute ischaemic myocardial lesion (Table 7). Previous hypertension was more common in the arrhythmia group and digitalis or diuretics had been given more often (77%) compared with 30 per cent ($P < 0.001$). Cardiac hypertrophy was also significantly more frequent ($P < 0.05$). Severe obstructive coronary arterial disease, however, was significantly less in this group than in those with late or silent infarction ($P < 0.001$). Smoking was not a characteristic feature of subjects who died from a 'primary arrhythmia' (Table 6).

Within the 'primary arrhythmia' group only 7 subjects (5%) had no definite evidence of ischaemic heart disease, that is no suggestive history, no myocardial lesion, and no coronary occlusion or thrombosis at necropsy. Two of them had aortic stenosis, and 2 a history of hypertension with pronounced cardiac hypertrophy. In the absence of ischaemic heart disease or a condition causing pronounced cardiac hypertrophy, therefore, an arrhythmia appears to be a rare cause of sudden death.

Discussion

The present series of sudden deaths is selected, including only those necropsies in which a particular technique was used, and excluding any case in which the fatal attack was unwitnessed. The latter clearly excluded many cases who died quickly and may have influenced the frequency of detectable acute infarction at necropsy. Despite this, some general conclusions on the characteristics of sudden death can be drawn, for example with regard to the distribution of coronary lesions, but the small number of cases in some comparisons must make interpretation tentative. The diagnosis of death from ischaemic heart disease is based mainly on the demonstration of a recent ischaemic lesion in the myocardium. It has been suggested that such lesions should be divided into two types: (1) myocardial infarction; a localised and discrete area of muscle necrosis in the myocardium visible to the naked eye and several centimetres in diameter and (2) diffuse subendocardial necrosis; circumferential myocardial necrosis involving the inner zone of the whole left ventricle (Davies *et al.*, 1976). In the present study the classification of recent ischaemic myocardial lesions was based on the microscopical changes in the myocardium. While many would accept as an acute myocardial infarction only those lesions with foci of polymorphonuclear leucocytes within the area of the necrotic myofibres or more advanced microscopical changes (Scott and Briggs, 1972;

Baroldi *et al.*, 1974), that is, criteria comparable with ours of a definite infarction, the histological changes taken as indicators of an early infarction by us, though not specific, were suggestive of very recent ischaemic myocardial injury (World Health Organization, 1973).

In relation to the acute ischaemic event, about half the deaths occurred during the earliest stages of myocardial injury and a quarter when frank infarction had taken place while the remainder resulted from a primary arrhythmia often before any infarction at all. Though most sudden deaths seemed to occur at a very early phase of the acute myocardial infarction, it was obvious that in many cases the acute infarction had either been silent, misinterpreted, or ignored. When death occurred at the stage of frank infarction, coronary artery disease tended to be severe, and vice versa. It is possible that myocardial adaptation to chronic coronary obstruction influenced the course of the ischaemic attack, the patients with severe coronary lesions being those who had avoided fatal arrhythmias during the earliest stage of myocardial injury by the development of collaterals. According to Baroldi (1975) the presence of 'contraction bands' in a myofibre means that it has died in a hypercontracted state, and the finding of this in cases of early myocardial infarction supports the concept of a possible role of catecholamines or sympathetic overstimulation in the pathogenesis of sudden death (Baroldi, 1975).

In the study of the IHD Register of Helsinki a history of hypertension was most common in those dying suddenly and lowest in the survivors of acute myocardial infarction (Romo, 1973), and other studies have shown that hypertension in patients with ischaemic heart disease increases the risk of death, particularly sudden death (Beck and Hochrein, 1974; Burggraf and Parker, 1975; Kornitzer *et al.*, 1975). In the pooled Helsinki register findings a history of hypertension was nearly twice as frequent in women with acute ischaemic heart disease as in men (World Health Organization, 1976). A similar sex difference was found in the present study though without the same level of significance because of the size of the series. Population studies in Finland have also shown a higher incidence of hypertension in women than in men in middle age (Aromaa, 1974). In the present study hypertensives were found more frequently than in the Finnish population (Aromaa, 1974), and may explain a number of the sudden deaths without definite pathological evidence of ischaemic heart disease. Patients dying suddenly without complaining of symptoms indicative of an ischaemic attack, and in whom no ischaemic lesion was found in the myo-

cardium, probably died of an arrhythmia. About 10 per cent of the cases fell into this category. These patients were not heavy smokers nor did they have severe coronary arterial disease. A history of hypertension, of the clinical symptoms of ischaemic heart disease, and of treatment with digitalis or diuretics was common. Their hearts tended to be enlarged and scarred and this, in the absence of severe coronary lesions, suggests that ischaemic heart disease in these subjects was of a different character from that in the patients who died with a detectable infarction. The use of digitalis and diuretics may simply reflect poor myocardial function. Alternatively, hypokalaemia following diuretic therapy may have caused arrhythmias such as ventricular ectopic beats or ventricular tachycardia in ischaemic subjects (Dyckner *et al.*, 1975).

Another factor increasing mortality in patients with ischaemic heart disease is smoking (Rose, 1973; Wilhelmsson *et al.*, 1975), and heavy smoking seems to be related to sudden death (Romo, 1973). Current smokers were overrepresented in the present series compared with the general Finnish population (Rimpelä *et al.*, 1975), and heavy smokers were common in the group in which death occurred a short time after the onset of infarction. On the other hand, heavy smoking was not characteristic of patients who died suddenly without infarction. Smoking, therefore, exerts a harmful influence via ischaemia rather than a tendency to arrhythmia, but may, however, favour the development of arrhythmias at the early stage of an ischaemic myocardial injury. It has been shown that the stimulation threshold for provocation of ventricular fibrillation decreases in laboratory animals inhaling tobacco smoke (Bellet *et al.*, 1970).

Severe obstructive coronary artery disease is characteristic of prehospital sudden deaths as well as of deaths from acute myocardial infarction in hospital (Rissanen *et al.*, 1975). The patterns of coronary stenosis varied considerably in the present series, with triple vessel disease with the major occlusive site in either the right or left anterior descending coronary artery being common. Patients with and without an old myocardial infarction also differed, the occlusive disease being more severe in the former than in the latter. A similar difference has also been found in quantitative studies of coronary atherosclerosis (Rissanen, 1975). The prognosis of single vessel disease has been shown to be better than that of double or triple vessel disease (Webster *et al.*, 1974; Burggraf and Parker, 1975) but in our patients without myocardial scarring coronary artery involvement was similar to that reported in many clinical series studied by coronary arteriography (Table 6). In patients with an old

myocardial infarction, however, severe triple vessel disease was commoner than in clinical series. Clearly these patients were a self-selected group at a final stage. An important question remains; did a previous attack of infarction enhance the development of coronary artery disease in these patients? An infarction, even if the patient survives, causes deterioration both by myocardial damage and by the thrombotic obstruction of the artery supplying the infarcted area (Fuster *et al.*, 1975; Davies *et al.*, 1976). The concept of mural thrombi as the major factor in plaque growth and production of stenosis is now gaining general acceptance (Roberts, 1973). Alternatively, previous infarctions are more likely to have occurred in patients with the severest coronary artery disease.

No particular pattern of coronary lesion was characteristic of any of the clinical types of sudden death but a high incidence of severe involvement of the right coronary artery seems to be a common finding (Rissanen *et al.*, 1975). An important function of clinical studies is to show which coronary lesions carry a risk legitimately correctable by surgery: a severe stenosis in the left main coronary artery is one generally accepted (Burggraf and Parker, 1975; Julian, 1975). This region was significantly obstructed in only 5 per cent of our patients, a similar incidence to that reported by Fuster *et al.* (1975) in patients with myocardial infarction (5%) or with angina (8%).

Attention has recently focused on angina pectoris and myocardial infarction in patients with no coronary stenosis. Several unusual conditions have been listed as a cause of such infarction (Cheitlin *et al.*, 1975). Long-term observations in this group have shown a favourable prognosis (Bemiller *et al.*, 1973). Aortic valvular stenosis, and hypertension with cardiac hypertrophy were found in the 3 cases of sudden death without coronary artery disease in the present series.

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