

Pathological findings in coronary arteries associated with sudden death in Austria

Franz Dienstl¹, Robert Gasser¹, Heribert Rauch¹, and Rainer Henn²

Department of Internal Medicine, Department of Cardiology and Intensive Care¹, Institute for Forensic Medicine², University of Innsbruck, Anichstrasse 35, A-6020 Innsbruck, Austria

Summary. 50 witnessed sudden cardiac deaths in the age group between 20–50 years have been studied at autopsy. The most remarkable findings were a high percentage of stenosis and arterioisclerosis of the descending branch of the left coronary artery and a large amount of three vessel disease. It is clear that severe stenosis and sclerosis of the coronary arteries are not essentially related to sudden cardiac death, but a high number of vessels with moderate stenoses and sclerosis has been found.

The severity of vessel disease has been evaluated by a coronary score, which takes the haemodynamic effects of the injured coronary arteries on the myocardium into account. We also noted that patients below 35 years of age who died of sudden cardiac death showed a very low coronary score.

Key words: Sudden death – Coronary disease – Left coronary artery – Autopsy findings

Introduction

A high percentage of the western population dies of sudden cardiac death (SCD). Many pathogenetic principles have been suggested: sudden cardio-vascular collapse may be due to dysrythmia, like ventricular tachycardia with fibrillation, ventricular asystole or severe bradycardia; it may be caused by mechanical blockade like pulmonary thromboembolism, cardiac tamponade, sudden ventricular pump failue (myocardial infarction, critical aortic stenosis) or vasodepressor reflexes. These are the major causes, others have been suggested too [16].

In this study we describe the morphological findings in autopsies on a group of patients between 20 and 50 years. We have looked at these findings mainly from the aspect of coronary disease with the intention to find a morphological substrate for SCD.

Some studies show up which describe coronary artery disease followed by myocardial infarction or fibrosis as the major cause for SCD [18, 21].

Offprint requests to: R. Gasser, Botanikerstrasse 17, A-6020 Innsbruck, Austria

But the role of coronary heart disease and myocardial infarction in connection with sudden death is still "a statistician's nightmare" [15] and the results of these studies differ greatly [18, 21, 14]. We, for instance, have used a coronary score according to Friesinger which takes into account the haemodynamic effects of sclerosis and stenosis of the coronary arteries on the myocardial blood supply.

Material and methods

In the course of five years 4417 autopsies have been performed at the Institute of Forensic Medicine; this has been done in accordance with Austrian law. In this group, 1090 cases (26,7%) were due to SCD. 126 were of the age of 20–50 years. For these a special protocol has been introduced, and the group has been divided into witnessed and unwitnessed SCD's. The protocols of those whose death has been witnessed (50 patients) have been studied and evaluated according to a detailed listing of morphological findings (Table 1, 2). The evaluation was carried out by two independent persons.

Sudden Cardiac Death (SCD). This has been defined as nontraumatic death within one hour after the onset of symptoms being probably of cardiac origin [5]. "Witnessed" has been defined as watched by an observer, regardless whether the patient was medically attended or not [5, 4]. A coronary score has been set up to represent the morphological findings, mainly considering the haemodynamic effects on the blood supply of the myocardium. It includes the localisation and severity of coronary stenosis and arteriosclerosis. Stenosis was estimated from longitudinally opened coronary arteries. All these results have been classified by age, sex and score.

Method of coronary scoring according to friesinger:

A. Considering the stensosis or occlusion:

Stenosis %	Factor	(Fs)
1–39		1
40-59		2
60–79		3
8099		4
100		5

B. Considering the haemodynamic effect:

	Factor (Fi)
Right coronary artery main branch proximal	$3 \times 4 = 12$
Right coronary artery main branch distal	$2 \times 1 = 2$
Right coronary artery ramus interventricularis posterior	$1 \times 2 = 2$
Right coronary artery ramus posterolateralis dexter	$1 \times 2 = 2$
Left coronary artery ramus interventricularis anterior	$3 \times 3 = 9$
Left Coronary artery ramus descendens	$1 \times 2 = 2$
	29

Coronary Score = Factor s × Factor i

 $CS = Fs \times Fi$

Stenosis and Arteriosclerosis were also evaluated in terms such as "slight, moderate and severe" [4]. We correlate "slight" with stenosis up to 50%, "moderate" with 50%-75%, "severe" with over 75% stenosis.

Table 1. Has been set up in accordance with the protocol we established for each patient

Stenosis – Sclerosis – Atheromatosis:	male	female	%
Soft vessels	4	2	12%
Soft vessels + 1 slight stenosis	2	4	8%
Soft vessels + 3 slight stenoses	1 .	0	2%
Soft vessels + 1 slight sclerosis	0	0	0%
Soft vessels + 1 slight sclerosis + 1 stenosis in the same vessel	0	1	2%
Soft vessels + 1 slight slcerosis + 1 stenosis in another vessel	2	0	4%
Slight sclerosis in more than 1 vessel		1	6%
Slight sclerosis in more than 1 vessel + 1 stenosis inside 1 of these vessels	3	1	8%
Slight sclerosis in more than 1 vessel + 2 stenoses in soft vessels	0	1	2%
Slight to moderate scleroses in at least 3 vessels + 1 stenosis in a sclerotic vessel	1	0	2%
Moderate sclerosis in at least 3 vessels		0	8%
Moderate sclerosis in at least 3 vessels + at least 1 stenosis in a sclerotic vessel	13	1	28%
Moderate sclerosis in at lest 3 vessels + at least 1 stenosis in a sclerotic vessel + at least 1 stenosis in a soft vessel	1	0	2%
Moderate to severe sclerosis in at least 2 vessels	1	0	2%
Moderate to severe sclerosis in at least 2 vessels + at least 1 stenosis in one of the sclerotic vessels	0	0	0%
Moderate to severe sclerosis in at least 2 vessels + at least 1 stenosis in one of the sclerotic vessels + other stenoses	3	0	6%
Severe sclerosis in at least 3 vessels		0	2%
Severe sclerosis in at least 2 vessels + stenoses in the sclerotic vessels	2	0	4%
Severe sclerosis in at least 1 vessel + 75% stenosis in this vessel		0	0%
Severe sclerosis in at least 1 vessel + 75% stenosis in this vessel + other stenoses	1	0	2%
	41	9	100%

Table 2. Has been set up in accordance with the protocol we established for each patient

	Sclerosis Sclerosis with thrombotic Occlusions		Stenosis	
RCA proximal	1	0	1	
RCA ramus circumflexus	2	1	1	
RCA ramus interventricularis posterior	1	1	1	
LCA proximal + main branch	2	0	1	
LCA ramus descendens	16	2	4	
LCA ramus circumflexus	1	1	1	

RCA = Right coronary artery; LCA = Left coronary artery

Results

The defined group of 50 patients (see material and methods) has been divided into males and females. The study shows a major group of males dying of SCD compared with females (see Fig. 1). The rather low number of females in the age group of 36–40 is remarkable (14%).

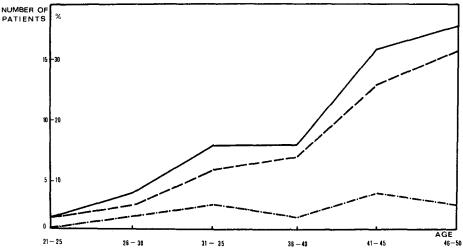
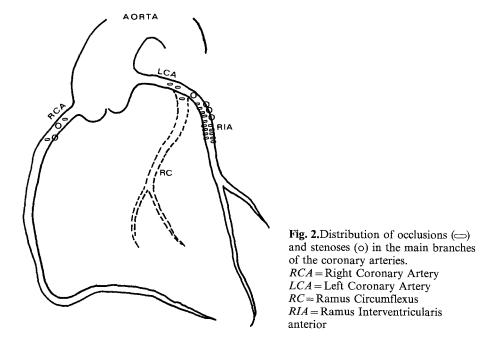
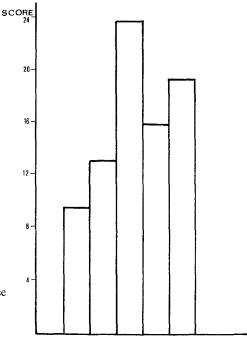


Fig. 1. Distribution of Sudden Cardiac Deaths according to age and sex (--= total; ---= female; --= male)



Eleven patients had very soft vessels, four of them showed one slight stenosis in spite of the severe outcome of the disease. Sixteen patients had moderate sclerosis and slight stenosis in one to three vessels (details see Table 1). Sixteen patients had moderate sclerosis in at least three vessels, and fourteen of them one isolated severe stenosis. Four patients had a severe stenosis in two vessels with severe arteriosclerosis. Finally three pa-

AGE



21-25 26-30 31-35 36-40 41-45 46-50

Fig. 3. Coronary score in comparison with the age groups. There is no coronary disease to be found in the age group of 21–25. The highest score shows up in the age group of 36–40

tients suffered from very severe sclerosis of all vessels and also several stenoses (Table 1).

The major component of the stenoses and scleroses has been found in the descending branch of the left coronary artery (40% of all patients), a minor component in the right coronary artery (8%) and only one has been found in the circumflex branch of the left coronary artery (Table 2, Fig. 2). The highest coronary score (CS = 23.7 ± 1.8) has been found in the age group 36–40, there is a coronary score also at the ages of 41–45 (CS = 17.8 ± 3.1) and in the group of 46–50 (CS = 19.2 ± 3.4). Most of the younger patients did not suffer from severe or moderate arterioscleroses, some of them showed no coronary artery disease [10, 11].

Another finding was that the major percentage of the patients had a coronary score between 10–29 (60%), very few showed a higher score like 30–39 (8%) or over 40 (4%) (Fig. 4). This shows that even moderate under perfusion caused by coronary artery disease may be followed by SCD [19, 20, 3, 13, 14].

Finally we found a high incidence of three vessel disease (58%), which supports similar findings by others [20].

Discussion

Within this group of fifty men and women who died suddenly there was a different extent of arteriosclerosis and thrombotic occlusions. The most

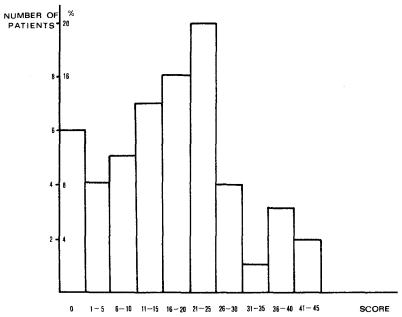


Fig. 4. Distribution of score related to age: Only a few patients show a very high score. The major amount of patients show a score below 25

remarkable location of a great number of occlusions and stenoses was found in the descending branch of the left coronary artery (Table 2). These facts support the conclusion that disease of the decending branch of the left coronary artery can be assumed to be a predisposition for SCD, which may suggest a more invasive management in treatment of the disease.

However we found that sudden death is not related to a high degree of coronary artery sclerosis or stenosis - on the contrary, many cases especially younger patients who died suddenly showed a moderate or slight stenosis or arteriosclerosis - whereas higher degrees of coronary artery disease are rarely found in sudden death patients. Other factors might be stated to be more important, especially in younger patients, such as hormonal changes, electrolyte abnormalities, coronary spasms, viral coronary disease [12], mental factors, vegetative dysregulations and others. An interesting disease which correlates with these findings and which also demonstrates the variety of reasons for SCD is the Pok - Kuri disease, which is found in East Asia: young patients below 40 spontaniously die of sudden cardiac failure without any morphological findings in coronary arteries. Coronary spasms are suggested as the cause of a lack of myoglobin in their myocytes [9]. Baroldi also has found less coronary artery disease in cases of sudden death [3]. He states that greatly enlarged collateral vessels are responsible for these findings [2, 1,]. One might also suggest that extensive fibrosis could cause ectopics which could lead to ventricular fibrillation in some cases. James supposes that collagen fibres may conduct mechanical vibrations to the sinus node and so cause ectopic beats there [19, 10, 11, 17, 18]. Schneider supports this theory by finding a changed quotient between myocytes and collagen fibres in patients dying suddenly [19]. This might be due to continuous underperfusion over a long period in certain areas of the myocardium. It is possible to extend James's hypothesis to the myocytes of the left ventricle, which may be stimulated by mechanical movements of the collagen fibres. There is no doubt that metabolic changes in the underperfused areas of the myocardium – eventually caused by short spasms – are of major importance. They may lead to slower conduction of the propagated excitation and enhance re-entry tachycardia and ventricular fibrillation.

Hinkle describes arrhythmia as the primary cause of SCD in middle aged American men [7, 8, 6]. The biochemical and electrophysiological mechanisms for stimulation of ectopic beats are not entirely clear. This study supports evidence for a relationship between SCD and stenosis or arteriosclerosis of the descending branch of the left coronary artery. It also shows that SCD is not necessainly correlated with severe coronary artery disease but, in contrast slight or moderate stenosis or sclerosis has been found in association with this mode of death.

Acknowledgement. We thank Dr. Susanne Benedikter, Isabella Nasal and Maria Großgasteiger for their volunteer help.

References

- 1. Baroldi G (1978) Coronary stenosis: ischemic or non ischemic factors? Am Heart J 96:139
- Baroldi G, Scomazzoni G (1967) Coronary Circulation in the normal and Pathological Heart, Washington D.C., U.S. Government Printing Office, American Registry of Pathology, Armed Forces Institute of Pathology
- 3. Baroldi G, Mariani F (1979) Sudden coronary death. A postmortem study in 208 selected cases compared to 97 "control" subjects. Am Heart J 98:20
- 4. Biörk G, Wikland B (1972) "Sudden Death" What Are We Talking About? Circulation, 45:256
- 5. Goldstein S (1982) The necessity of a uniform definition of sudden coronary death: Witnessed death within 1 hour of the onset of acute symptoms, Am Heart J 103:156
- Hinkle L (1977) Pathogenesis of an unexpected sudden death: Role of early cycle VPCs. Am J Cardiol 39:873
- 7. Hinkle L, Witney L, Lehmann E, Dunn J, Benjamin B, King R, Plakun A, Flehinger B (1968) Occupation, education and coronary heart disease. Science 161:238
- 8. Hinkle L (1979) The Antecendents of Sudden Death. Prospective Studies (Report on contract Nr. NHL 70-02069, prepared for the Cardiac Diseases Branch, Division of Heart and Vascular Diseases, National Heart, Lung and Blood Institute; Bethesda MD 20014). Part 1: Methods and Criteria. Copies available from National Technical Information Service, 5285 Port Royal Road, Springfield VA 22151
- Ishiyama I, Kamiya M, Rose M, Komuro E, Takatsu A (1982) Fulminant deletion of myoglobin from myocardial fibres in state of acute cardiac failure inducing sudden cardiac arrest. Lancet 12:1468
- 10. Jmes T (1961) Anatomy of the human sinus node. Anat Rec 141:109
- 11. James T (1972) Mysterious sudden death. Chest. 62:454
- 12. James T, Imamura K (1981) Virus-like particles associated with intracardiac ganglionitis in 2 cases of sudden unexpected death. Jpn Heart J 22:447
- 13. Kaltenbach M (1980) Röntgenanatomie und Nomenklatur, Quantifizierung und Dokumentation koronarangiographischer Befunde. In: Vom Belastungs-EKG zur Koronarangiogra-

- phie. Kaltenbach M, Roskamm H (eds) Springer, Berlin, Heidelberg, New York, chap 3.2
- 14. Lie J, Titus J (1975) Pathology of the Myocardium and the Conduction System in Sudden Coronary Death, Circulation 51 and 52 (Suppl III):41
- 15. Lovegrove T, Thompson P (1978) The role of acute myocardial infarction in sudden cardiac death a statistician's nightmare, Am Heart J 96:711
- 16. Lown B (1980) Cardiovascular collaps and sudden death, in Heart Disease, Braunwald E (ed). Philadelphia, Saunders chap 22
- 17. Perper J, Kuller L, Cooper M (1975) Arteriosclerosis of Coronary Arteries in Sudden, Unexpected Death. Circulation 51 and 52 (Suppl. III) 27
- 18. Roberts WC, Jones AA (1979) Quantitation of Coronary Arterial Narrowing at Necropsy in Sudden Coronary Death, Am J Cardiol 44:39
- 19. Schneider J (1981) Der plötzliche Herztod als Folge einer Reizleitungsstörung, Schweiz Med Wochenschr 111:366
- Steinbrunn W, Kappenberger L, Lichtlen P (1981) Die Häufigkeit des plötzlichen Herztodes bei der Koronaren Herzkrankheit. Schweiz Med Wochenschr 111:1697
- 21. Weaver WD, Lorch G, Alvarez HA, Cobb L (1976) Angiographic findings and prognostic indicators in patients resuscitated from sudden cardiac death. Circulation 54:896

Accepted June 10, 1985