Pathologic Features of Sudden Death in Children, Adolescents, and Young Adults*

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Pathologic investigation of 50 children, adolescents, and young adults who died suddenly and unexpectedly revealed that the most frequently encountered cardiovascular diseases were mitral valve prolapse (12 cases), myocarditis (12 cases), hypertrophic cardiomyopathy (six cases), and anomalous origin of a coronary artery from the aorta (two cases). Five subjects had no evidence of cardiac abnormalities. A striking family history of sudden death was encountered among eight subjects, of whom three had mitral valve prolapse and three had normal hearts. Assessing the circumstances surrounding sudden death, it was found that at the time of collapse, 32 subjects were engaged in regular activity, eight subjects were engaged in active athletics, and ten were found dead in bed. A relationship of the terminal event to emotional stress was reported in three subjects.

It is known that children, adolescents, and young adults may die suddenly, yet there are very few reports regarding this age group. The purpose of this study was to determine the circumstances at the time of sudden death and the types of diseases found at autopsy.

Fifty patients (29 male and 21 female subjects), aged seven to 35 years, who died suddenly were identified from a review of the Cardiovascular Registry of United Hospitals, St. Paul, Minn, for cases accessioned between 1960 and 1983. The cases were derived from numerous sources during this period. In most instances the pathologic material was the gross specimens of heart. In a few cases that we reviewed for this study, only microscopic slides represented the pathologic material. The conduction system was studied in some cases, but this was not done regularly.

**DEFINITION**

The definition of sudden death as used herein is an unexpected natural phenomenon in which loss of all vital functions occurs instantaneously or within six hours of the onset of symptoms or collapse. There were no cases with a past history of successful cardiopulmonary resuscitation following an episode of collapse. To ensure that death was unexpected, we included in the study only those subjects who were not hospitalized at the time of onset of acute symptoms or collapse. No patient who had undergone cardiovascular surgical procedures was included. Children younger than seven years of age were excluded because of the difficulty in determining whether death was unexpected in symptomatic infants with known heart disease and to eliminate the unsolved problem of crib death. Based upon the criteria for inclusion or exclusion of cases in this study, there remained 50 cases of sudden death for review. For each of the 50 cases in this study, use was made of the historic, clinical, and postmortem data from which to designate a cause of death.

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Manuscript received February 20; revision accepted November 7.
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**RESULTS**

The cardiac abnormalities which accounted for sudden death in this series are presented in order of decreasing frequency in the following tabulation:

<table>
<thead>
<tr>
<th>Disease</th>
<th>Cases</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mitral valve prolapse</td>
<td>12</td>
</tr>
<tr>
<td>Myocarditis</td>
<td>12</td>
</tr>
<tr>
<td>Hypertrophic cardiomyopathy</td>
<td>6</td>
</tr>
<tr>
<td>Coronary arterial diseases:</td>
<td></td>
</tr>
<tr>
<td>Congenital</td>
<td>2</td>
</tr>
<tr>
<td>Acquired obstructive:</td>
<td></td>
</tr>
<tr>
<td>Atherosclerotic</td>
<td>1</td>
</tr>
<tr>
<td>Dissecting aneurysm</td>
<td>1</td>
</tr>
<tr>
<td>Myxomatous intimal thickening</td>
<td>1</td>
</tr>
<tr>
<td>Coexisting conditions</td>
<td>3</td>
</tr>
<tr>
<td>Aortic stenosis</td>
<td>2</td>
</tr>
<tr>
<td>Pulmonary hypertension</td>
<td>1</td>
</tr>
<tr>
<td>Degenerative changes in bundle of His</td>
<td>1</td>
</tr>
<tr>
<td>Cardiac tumor</td>
<td>1</td>
</tr>
<tr>
<td>Normal heart</td>
<td>5</td>
</tr>
<tr>
<td>Undetermined</td>
<td>2</td>
</tr>
<tr>
<td>TOTAL</td>
<td>50</td>
</tr>
</tbody>
</table>

**Mitral Valve Prolapse**

There were 12 cases of sudden death attributed to mitral valvular prolapse (included are ten cases previously reported from our laboratory by Chesler and co-workers). There were seven female and five male patients. The ages ranged from 13 to 30 years. The degree of prolapse varied from mild (two cases) to moderate or severe (ten cases). Among the 12 subjects, ten had had auscultatory evidence of mitral valve prolapse. Ventricular premature complexes were documented in six, and one patient had had one previous episode of atrial fibrillation. In only one case was the dysrhythmia treated medically immediately before death. A family history of sudden death was reported in three cases.

**Myocarditis**

There were 12 cases with myocarditis, of which 11
were presumed to be viral and one sarcoid. Among the 11 cases considered to be of viral origin, eight were chronic, and three were acute.

Chronic myocarditis was characterized by multifocal scars without any major localized scar. Leukocytic infiltration was either absent or minimal, the leukocytes usually being lymphocytes and macrophages.

Acute myocarditis was characterized by the presence of necrotic myocardial fibers and varying degrees of leukocytic infiltration, including polymorphonuclear neutrophil. Among the cases of acute myocarditis was a nine-year-old boy with mumps who died suddenly on the third day of illness.

**Hypertrophic Cardiomyopathy**

Six subjects had hypertrophic cardiomyopathy. Included are four female and two male patients whose ages ranged from 11 to 21 years. Four subjects had asymmetric septal hypertrophy with a ratio of septal-to-left ventricular free wall thickness of up to 2.2. Microscopic examination in these areas revealed myocardial disarray and interstitial fibrosis. There were two subjects who had hypertrophic cardiomyopathy without asymmetric hypertrophy, although disarray of myocardial fibers was present in one of these cases. In the latter two the cardiac weights were 420 g in a 14-year-old girl and 535 g in a 21-year-old man.

**Coronary Arterial Disease**

**Anomalous Coronary Arterial Origin.** There were two subjects in whom the entire coronary arterial system arose from the aorta but with anomalous position of origin of one of the coronary arteries. The first case was that of a presumably healthy 15-year-old girl who died suddenly while playing basketball. Postmortem examination revealed that the right and left main coronary arteries each arose from the right aortic sinus. The ostium of the left main coronary artery was located in the right aortic sinus anterior to the normally positioned ostium of the normal right coronary artery. The left coronary artery left the aorta at an acute angle, passing sharply leftward between the aorta and the pulmonary trunk, reaching the base of the anterior interventricular sulcus, where it branched into the anterior descending and circumflex arteries. The coronary arteries were devoid of atherosclerosis.

The second case was that of a 20-year-old man in whom both the left and right coronary arteries arose from the left aortic sinus. As the right coronary artery left the aorta, it exhibited an acute angle with the aorta and then followed a normal distribution. The acute angle resulted in the coronary artery being intimately related to the wall of the aorta with an intramural route of the proximal portion of the anomalous coronary artery. There was a common media between the aorta and the anomalous coronary artery with no intervening adventitia. Neither atherosclerosis nor other abnormalities were noted.

**Dissecting Aneurysm of a Coronary Artery.** This case (previously reported from our laboratory), a 32-year-old woman three weeks postpartum, suffered from acute primary dissecting aneurysm of the anterior descending coronary artery, causing occlusion of the vessel. Cystic medial necrosis of the left anterior descending artery was found. The myocardium showed foci of acute myocardial infarction in the healing stage.

**Myxomatous Intimal Thickening.** In one case, myxomatous changes in coronary arterial intima were associated with sudden death. The patient was a 21-year-old man. Myxomatous change was found in small intramyocardial arteries. Fibrous mucoid connective tissue was deposited in the artery of the atrioventricular node, causing marked luminal narrowing. Minimal changes of this type were present in the anterior descending artery.

**Atherosclerosis.** A 34-year-old diabetic man showed major atheromatous narrowing of the circumflex artery by an atheroma. The left anterior descending and posterior descending coronary arteries showed grade-2 atherosclerosis. The inferolateral wall of the left ventricle showed a purple discoloration suggesting early acute infarction. Histologically, early infarction of the myocardium could not be excluded.

**Coexisting Conditions**

Three patients each exhibited two conditions, either of which may have been responsible for sudden death. These are summarized subsequently:

**Myocardial Dysplasia and Mitral Valve Prolapse.** A 23-year-old man had major dysplasia of the right ventricle and minor dysplasia of the left ventricle and a minor degree of mitral valve prolapse. He presented with an episode of pain in the chest and palpitations one hour before death.

**Myocardial Dysplasia and Coronary Atherosclerosis.** A 31-year-old man had severe scattered focal atrophy of the right ventricular muscle with marked dilatation of the right ventricle. He also had grade-3 coronary atherosclerosis of the left main coronary, of the proximal part of the left anterior descending coronary, and of the mid-right coronary arteries.

**Aortitis and Hypoplastic Right Coronary Artery.** In a 21-year-old man, findings at autopsy included a narrow right coronary ostium with a diminutive right coronary artery and healed inferior left ventricular infarction. Microscopically, there was extensive aortitis with a tendency in some areas for granuloma formation, with giant cells in varying numbers. Some of the media showed infarct-like necrosis, and there
was heavy infiltration with lymphocytes and plasma cells in both the outer media and adventitia. The intima showed fibrous thickening and was capped by unorganized thrombus. Neither coronary atherosclerosis nor emboli were identified.

The presence of healed inferior myocardial infarction raises the question as to whether the hypoplastic right coronary artery underlay not only the old myocardial lesion, but was also a cause of a fatal ventricular arrhythmia. An alternative explanation both for the old myocardial infarction and for the sudden death was embolism of thrombotic material attached to the intimal surface of the diseased aorta.

Aortic Stenosis

Two subjects had congenital unicuspid unicommissural aortic stenosis. One was a 19-year-old man who was known to have aortic stenosis but was asymptomatic and received no medication. The circumstances of his witnessed death revealed that he had emotional stress at that time. At autopsy the heart weighed 690 g. In addition to the congenital aortic valvular unicommissural stenosis, the lining of the outflow tract of the left ventricle showed regurgitant lesions from associated aortic insufficiency. The posteromedial papillary muscle of the mitral valve was infarcted.

The second subject was a 26-year-old man with no known disease or symptoms. The heart weighed 375 g. The aortic valve was congenitally malformed, represented by a unicommissural valve with dysplastic thickening and prolapse of the right anterior portion of the valve. The single commissure was in the position of the left-posterior commissure, while the right-posterior and right-left commissures were represented by low, focally calcified raphes. The posteromedial papillary muscle of the mitral valve was scarred.

Pulmonary Hypertension

A 20-year-old woman died suddenly 62 hours after delivery of a premature infant. At autopsy, there was evidence of chronic pulmonary hypertension based on thrombogenic pulmonary hypertensive vascular disease associated with secondary features of chronic pulmonary hypertension in the form of right ventricular hypertrophy. There was no evidence of amniotic embolism.

Cardiac Tumor

A nine-year-old girl had mesothelioma of the atrioventricular node. No prior symptoms had been reported. The neoplasm abutted upon the central fibrous body and extended from the lower portion of the atrial septum to within 0.1 cm of the tricuspid anulus.

The tumor infiltrated the atrial musculature but did not infiltrate the atrioventricular bundle. There were subendocardial, band-like myocardial calcifications of both ventricles and of the right atrium. Superficial medial calcification of the pulmonary trunk and focal calcification of the pulmonary valve were present.

Degenerative Conduction System

The patient was a 15-year-old boy. Both ventricles showed slight to moderate endocardial fibroelastosis. Microscopically, within the atrioventricular bundle, increased amounts of adipose tissue were present. There were also multifocal areas of fibrosis of the atrioventricular bundle, and the left bundle showed focal areas of replacement of conduction tissue by collagen.

Normal Hearts

One female and four male subjects in this series had no evidence of disease of any organ. One case is that of an 18-year-old woman who collapsed and died upon being awakened by a ringing telephone. There was a strong family history of sudden death, in that the mother and maternal uncle had died suddenly at 23- and 19 years of age, respectively, and their autopsies revealed no anatomic cause of death.

Undetermined

Included in this category were two subjects whose only findings were minor cardiac abnormalities which could not positively account for sudden death. In one, a 14-year-old boy, autopsy showed minimal right ventricular hypertrophy. Since the lungs were not available, the possibility of underlying primary pulmonary hypertension cannot be excluded. If an obstructive lesion had occurred in the lungs with the increased cardiac output during exercise, right ventricular failure might possibly have occurred. It should be noted that in this case, there was a strong family history of Friedreich's ataxia.

The other patient was a 20-year-old man whose findings included mild hypertrophy of the heart, which weighed 440 g. Histologically, there was moderately severe intimal thickening of some of the medium-sized intramyocardial arteries. There was patchy eccentric elastosis of the intima, and the lumen appeared to be reduced to approximately 50 percent; however, the majority of the intramyocardial arteries did not show these alterations.

Athletic Activity and Sudden Death

In this study, eight subjects (16 percent) died suddenly while engaged in sports. There were five male and three female subjects, and the ages ranged from 11...
Table 1—Cardiac Findings in Eight Patients Who Died Suddenly during Athletic Activity

<table>
<thead>
<tr>
<th>Condition</th>
<th>No. of Cases</th>
<th>Age (yr); Sex</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mitral valvular prolapse</td>
<td>2</td>
<td>F, 27; M, 30</td>
</tr>
<tr>
<td>Chronic myocarditis</td>
<td>2</td>
<td>M, 5; M, 30</td>
</tr>
<tr>
<td>Undetermined</td>
<td>2</td>
<td>M, 14; M, 20†</td>
</tr>
<tr>
<td>Anomalous origin of left coronary artery from right aortic sinus</td>
<td>1</td>
<td>F, 15</td>
</tr>
<tr>
<td>Idiopathic hypertrophic subaortic stenosis</td>
<td>1</td>
<td>F, 11</td>
</tr>
<tr>
<td>Total</td>
<td>8</td>
<td></td>
</tr>
</tbody>
</table>

*Right ventricular hypertrophy.
†Thickening of intramyocardial arteries.

to 30 years. The cardiac findings pertaining to these cases are summarized in Table 1. Of the various conditions found, the two that appeared twice were mitral valve prolapse and chronic myocarditis. Idiopathic hypertrophic subaortic stenosis and anomalous origin of a coronary artery were each observed once.

Familial Occurrence of Sudden Death

Among the 50 subjects, eight had a family history of sudden death. Among these eight subjects the cardiac findings accounting for sudden death were as follows: mitral valve prolapse, three cases; and idiopathic hypertrophic subaortic stenosis and right ventricular hypertrophy, one case each. In each of three cases, postmortem examination revealed that the heart was normal.

Among relatives of these eight subjects, a total of 17 relatives had died, and four living relatives exhibited evidence of heart disease. The postmortem findings of the eight subjects and of their 17 relatives, as well as clinical data on the living relatives, are presented in Table 2.

Features of Fatal Event

The location of the terminal event was at home in 28 cases (56 percent), and in 22 cases (44 percent) the event occurred in a public place. At the time of collapse, 32 subjects (64 percent) were engaged in regular activity (walking; standing; studying). Eight subjects (16 percent) were engaged in active athletics, and ten others were found dead in bed. A relationship of the terminal event to emotional stress (anger or fear) was reported in three subjects. Death occurred instantaneously in 44 subjects. The interval between the onset of cardiac arrest and cessation of resuscitative measures was estimated to be ten minutes in one case, one hour in three cases, two hours in one case, and three hours in another case. The collapse was witnessed in 40 subjects, of which 27 received cardiopulmonary resuscitation.

Discussion

We conducted this study on sudden death of children, adolescents, and young adults based on clearly defined criteria in order to determine the causes and circumstances of death in this particular age group. Published data of sudden death in children and young adults are sparse. An international study on this issue included 254 subjects, and there was wide range in the degree to which death could be identified as unexpected. Only 5 percent of the subjects in that study had no previously known heart disease.

The majority of subjects in our study on sudden death have been ruled to have died of one or another form of cardiovascular disease. This study cannot be taken as a comprehensive one on sudden death in the young, since the cases were referred to our laboratory either because a cardiovascular condition was identified, or it could not be excluded by the referring pathologist.

In 47 of the 50 subjects in this study, a structural cardiovascular abnormality was identified at autopsy, and in 45 subjects, it was considered accountable for sudden death. The most frequently encountered diseases were mitral valve prolapse and myocarditis.

Table 2—Familial Occurrence of Sudden Death and Cardiac Abnormalities in Relatives

<table>
<thead>
<tr>
<th>Age (yr); Sex</th>
<th>Cardiac Findings</th>
<th>Data Concerning Relatives</th>
</tr>
</thead>
<tbody>
<tr>
<td>14, F</td>
<td>Mitral valve prolapse</td>
<td>Mother, brother (11 yr), and sister (12 yr) died suddenly; all had normal hearts</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Living sisters (18, 22, and 24 yr); all have mitral valve prolapse</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Living cousin; has asymmetric septal hypertrophy</td>
</tr>
<tr>
<td>19, M</td>
<td>Mitral valve prolapse</td>
<td>Mother’s sibling died suddenly (14 yr); had features of Marfan’s syndrome</td>
</tr>
<tr>
<td>21, M</td>
<td>Mitral valve prolapse</td>
<td>Sister (17 yr) died suddenly</td>
</tr>
<tr>
<td>18, F</td>
<td>Normal heart</td>
<td>Mother (23 yr) and maternal uncle (19 yr) died suddenly; normal hearts</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Brother (19 yr) and paternal uncle (young) died suddenly</td>
</tr>
<tr>
<td>22, M</td>
<td>Normal heart</td>
<td>3 siblings (18, 29, and 27 yr) died suddenly; normal hearts</td>
</tr>
<tr>
<td>15, F</td>
<td>Idiopathic hypertrophic</td>
<td>Mother (46 yr) died suddenly; idiopathic hypertrophic subaortic stenosis</td>
</tr>
<tr>
<td></td>
<td>subaortic stenosis</td>
<td></td>
</tr>
<tr>
<td>14, M</td>
<td>Undetermined; right</td>
<td>4 of mother’s siblings died suddenly; all had Friedreich’s ataxia</td>
</tr>
<tr>
<td></td>
<td>ventricular hypertrophy</td>
<td></td>
</tr>
<tr>
<td></td>
<td>present</td>
<td></td>
</tr>
</tbody>
</table>
Although it has been concluded that mitral valve prolapse is "benign,"

sudden death had been documented.2 Nevertheless, it must be exceedingly rare, considering the common occurrence of mitral valve prolapse in the population.

In a previous report from our laboratory1 on the clinicopathologic features of sudden death associated with the myxomatous mitral valve, it had been suggested that friction by chordae upon the left ventricular wall might be a basis for ventricular ectopy. Coronary embolism from thrombotic lesions occurring in the angle between the posterior leaflet and the left atrial wall may be another basis for the fatal ventricular dysrhythmia.

Of special interest is the finding in our series of two subjects with mitral valve prolapse who died suddenly while engaged in athletic activity. Pocock and Barlow6 studied 12 patients with mitral valve prolapse while subjected to strenuous exercise. Under this condition, these investigators observed multifocal ectopic beats and other ventricular tachyarrhythmias and warned that sudden death may complicate exercise. Thus, they recommended that a postexercise electrocardiogram be done in all patients with a late systolic murmur or nonejection click.

A familial incidence of mitral valve prolapse has been recognized,4 and sudden death has been reported in relatives of involved subjects.7,8 This is further substantiated by the findings in this study of three subjects having mitral valve prolapse and a family history of sudden death.

Myocarditis was encountered in 24 percent (12) of our subjects, while it is encountered in 1 to 5 percent of unselected autopsies.9 We encountered eight subjects with chronic myocarditis accountable for sudden death. The mechanism of sudden death in chronic myocarditis may be due to the fibrosis and scar formation in the myocardium. Thus, the damaged tissue is involved in the creation of recurrent arrhythmia and conduction abnormalities. Acute myocarditis reveals variable clinical features. Sudden death rarely appears as the initial manifestation of illness.

Most cases of sudden death associated with anomalous origin of a coronary artery from the aorta reported in the literature were those in which the left coronary artery arose from the right aortic sinus10 and coursed anterior and to the left between the aorta and the pulmonary trunk. In our files, there was one subject with this anomaly. Roberts et al11 reported three patients who died suddenly in whom the right coronary artery arose from the left aortic sinus. Our fatal case with the right coronary artery arising from the left aortic sinus is, as far as we are aware, the sixth reported case of sudden death due to this anomaly. There is controversy as to the mechanism by which the anomalously arising coronary artery causes sudden death.

Some authors suggest a distal kinking of the anomalous artery or compression, especially while exercising, of the anomalous artery as it passes between the aorta and the pulmonary trunk.12,13 Cheitlin and co-workers12 suggest that the acute angle at which the anomalous artery leaves the aorta results in a flap-like mechanism at the coronary ostium. Expansion of the aorta during exercise results in this flap obstructing the ostium. During exercise, when the diastolic filling time of the coronary arteries is shortened, the important role of this obstruction becomes crucial.

Sudden and unexpected death is a recognized complication in symptomatic and asymptomatic children and adults with hypertrophic cardiomyopathy. In our series, six subjects with hypertrophic cardiomyopathy died suddenly, one of whom was engaged in physical exertion at the time of death.

Maron and co-workers14 reported on 26 cases with hypertrophic cardiomyopathy in which death was the first definitive manifestation. These investigators concluded that sudden death accountable to this disease is common in children and young adults and is often related to physical exertion. The mechanism of death in patients with hypertrophic cardiomyopathy has been attributed to ventricular dysrhythmia.15 It is known that sudden death is more prevalent in families with hypertrophic cardiomyopathy than in nonaffected families. A history of sudden death in a closely related relative may be a poor prognostic sign. This was the feature in a 15-year-old girl in our series who died suddenly with evidence for idiopathic hypertrophic subaortic stenosis and whose mother later died suddenly at 46 years of age of the same disease.

In myocardial dysplasia the right ventricular musculature is partially absent and is replaced by fatty and fibrous tissue. Isolated cases have been reported in which left ventricle is also involved.16 The first subject in our series presented myocardial dysplasia of the right ventricle and dysplasia of the left ventricle as well. The presence of mitral valve prolapse, as in this patient, in association with myocardial dysplasia had been reported previously by Marcus and co-workers,16 who reported 24 cases with right ventricular dysplasia, of which three had mitral valve prolapse.

Patients who have a major deficiency of the right ventricular myocardium may survive to adulthood but may present clinical symptoms such as congestive cardiac failure or recurrent ventricular arrhythmia. When ventricular tachycardia is the principal manifestation, it has been termed by Fontaine and co-workers17 as arrhythmogenic right ventricular dysplasia. According to previous studies the prognosis appears to be rather good with regard to the arrhythmias.18 One case of repeated attacks of ventricular fibrillation has been described by Olsson and co-workers,18 and a case of identical adult twins who both
had episodes of ventricular dysrhythmia was described by Hoback and associates. As far as we are aware, our two cases are the first reported cases in which sudden death was the first manifestation.

Two subjects in our series, one 19 and the other 26 years of age, presented with congenital unicuspid aortic stenosis. The rather frequent association of aortic stenosis with sudden death has been reported previously by many authors.9-11

The three small primary tumors of the heart that are associated with sudden death are Purkinje cell tumors, fibroma of the central body, and mesotheloma. A review of the literature reveals that patients with mesotheloma of the atrioventricular node have a wide range in age at the time of death, from an eight-month-old fetus to 86 years of age. The majority of the subjects were female. Pathologic examination shows that these tumors have usually been less than 20 mm in the largest dimension and often have not been grossly visible. Microscopically, the findings are those of cystic or tubular spaces lined by single or multiple layers of cuboidal or columnar epithelium, together with areas of hemorrhage and chronic inflammatory infiltrate, which consist mostly of lymphocytes and foec of calcification. Usually, various degrees of heart block are present.12,13 In our case the absence of the typical signs such as syncopal episodes, conduction disturbances, or arrhythmia precluded any chance of detection of the disease prior to death.

Five subjects (10 percent) in our series had apparently normal hearts. Reichenbach and associates14 were unable to demonstrate structural heart disease in 8 percent of their postmortem examinations of adult victims of sudden death. Some of the cases with normal hearts may be accounted for by metabolic and electrolytic abnormalities, exercise-induced arrhythmias, and, possibly, neurally mediated arrhythmia.15 In 1957, Jervell and Lange-Nielsen16 described a heritable syndrome consisting of syncope and sudden death in patients with deaf mutism and a prolonged Q-T interval.

Wellens and co-workers17 studied a patient who suffered from repeated attacks of ventricular fibrillation when auditory stimuli were applied during sleep and concluded that their patient represented another example of the Q-T prolongation syndrome. We encountered an 18-year-old woman who collapsed and died upon arousal from sleep by a ringing telephone. This case may represent a possible relation between auditory stimuli and the formation of ventricular fibrillation. It should be noted that in this case, there was a striking family history of sudden death. The mother of that subject died suddenly at the age of 23 years, and the maternal uncle died suddenly at 19 years of age. Both had normal hearts at autopsy. One may postulate that this family represents a fatal familial disease of auditory arrhythmic features.

Impaired nutrition to the atrioventricular node by obstruction of this artery can lead to degeneration and fibrosis of the atrioventricular junctional conduction tissue. This process, which was encountered in one subject in our series, had been observed in association with sudden death.28

The question of whether psychologic stresses predispose the heart to ventricular fibrillation and thus to sudden death was searched in investigations conducted by Lown and associates29 and by DeSilva and Lown.30 Their results indicate that the nervous system's activity plays a role in the genesis of arrhythmia. Increases in the input of sympathetic activity to the heart, whether induced by neural or neurohormonal action, predisposes the heart to ventricular fibrillation. In contrast to another study,3 in which the association between the terminal event and emotional stress could not be identified, we encountered three subjects who were reported to be under psychologic stress at the time of sudden death.

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