

Michele J. Catellier,<sup>1</sup> M.D.; Bruce F. Waller,<sup>2</sup> M.D.;  
Michael A. Clark,<sup>3</sup> M.D., Ph.D.; John E. Pless,<sup>3</sup> M.D.;  
Dean A. Hawley,<sup>3</sup> M.D.; and Allen W. Nyhuis,<sup>1</sup> M.S.

## Cardiac Pathology in 470 Consecutive Forensic Autopsies

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**ABSTRACT:** Cardiovascular disease continues to be the single most common generic cause of sudden and unexpected deaths. Atherosclerotic coronary heart disease and acute myocardial infarction are the most prevalent forms of fatal cardiac disease observed at autopsy. Other cardiac lesions are frequently listed as causes of death, but the prevalence of such lesions as incidental findings in the general population is unknown. In this study, 470 consecutive forensic autopsies were evaluated for minor and major anomalies. The most frequently observed major congenital finding was floppy mitral valve (5%). Tunneled coronary arteries, considered minor congenital findings, were seen in 29%. Atherosclerotic coronary heart disease was the most common major acquired finding, observed in 16% of cases. Of the 470 hearts, only 8% were considered normal.

**KEYWORDS:** pathology and biology, cardiovascular system, cardiovascular disease, heart, coronary atherosclerosis, autopsy, congenital heart disease, forensic pathology

Cardiovascular disease is the number one killer in the United States today, claiming nearly as many victims as all other causes of death combined [1]. More than half of these deaths are sudden or unexpected or both [2-4] and thus fall into the realm of the forensic pathologist. The purpose of this study was to evaluate systematically all hearts from a series of consecutive forensic autopsies to determine minor and major cardiac abnormalities. The authors hope that by recognition of the types and numbers of anomalies seen in a typical practice, the forensic pathologist may better differentiate between significant and incidental cardiac findings. In addition, the compilation of these findings may provide a database for the frequency with which they may be found in a group of nonhospitalized decedents.

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<sup>1</sup>Resident physician, and biostatistician, respectively, Department of Pathology, Methodist Hospital of Indianapolis, Inc., Indianapolis, IN.

<sup>2</sup>Professor of pathology and medicine and director, Section of Cardiovascular Pathology, Indiana University School of Medicine, Indianapolis, IN.

<sup>3</sup>Associate professor, Culbertson professor and director, and assistant professor, respectively, Division of Forensic Pathology, Department of Pathology, Indiana University School of Medicine, Indianapolis, IN.

## Methods

### *Site and Population*

This study was based upon the Marion County Coroner's population, derived from an area that includes Indianapolis, Indiana, and surrounding townships. Based upon the 1985 census, the population of Indianapolis and environs within Marion County was estimated at 1.2 million. The total number of Marion County Coroner's cases examined by forensic pathologists in 1987 was 1322; of these, 677 were autopsied. The total number of cases included in this series was 500, which represented a caseload of approximately 9 months. Selected cases, including those that were skeletonized or badly decomposed, were excluded, leaving a total of 470 hearts for this analysis.

Data analyses were performed using SAS software.<sup>4</sup> Numeric comparisons were done using the unpaired *t*-test; the percentages were compared using the binomial test.

### *Evaluation of Hearts*

All the hearts evaluated in this study were initially examined by a forensic pathologist, then reexamined in detail by a cardiac pathologist. All gross findings were recorded. Distinction was not made at the time of cardiac evaluation as to the significance of the lesions. Routine histology was performed on all the hearts and served to confirm the gross diagnoses.

The cardiac observations were divided into the following categories: (1) normal versus abnormal hearts, (2) congenital versus acquired anomalies, and (3) *Major* versus *minor* cardiac findings.

### *Definitions Used in this Study*

*Major* cardiac findings were those that were recognized as responsible for death or as a potential cause of death. *Minor* cardiac findings were those that were not considered as major findings, but were also not normal. Under most circumstances, minor findings may be classified as incidental.

For the purposes of this study, *sudden cardiac death* was defined as that which occurred within six hours of the onset of symptoms, if any. Although the individual may have had a history of cardiac disease, if he or she had been pursuing a life-style "normal" for that person, and the death came unexpectedly, it was considered sudden. The six-hour time span was chosen for this study to exclude myocardial necrosis (that is, acute myocardial infarction) as a cause of death [5,6].

*Severe atherosclerotic coronary heart disease* is defined as that which had produced greater than 75% cross-sectional area luminal narrowing of a major coronary artery vessel on gross examination. *Healed myocardial infarction* is defined as that recognized on gross examination by fibrous scar tissue deposition.

*Dilated cardiomyopathy* was established in hearts that demonstrated dilation of all four cardiac chambers unassociated with major valvular, coronary artery, or pericardial disease [7,8]. In most cases, the etiology of the dilated cardiomyopathy was classified as idiopathic. *Hypertrophic cardiomyopathy* was diagnosed by asymmetric septal hypertrophy (a ratio of ventricular septum to free wall thickness of at least 1.3 to 1.0), increased heart weight, normal or small left ventricular cavity size, and histologic confirmation of myocardial fiber disorganization [9].

The *neoplastic* lesions seen in this study were all benign papillomas. These lesions,

<sup>4</sup>SAS Institute, Inc., Carey, NC.

also called papillary fibromas or papillary fibroelastomas, grossly resemble a sea anemone with many finger-like projections of connective tissue overlying a short pedicle [10,11].

The hearts were identified as demonstrating *excess adiposity* by subjective evaluation. Normal surface fat was graded 0; Grade 4 demonstrated 100% coverage of the right ventricle with subepicardial fat.

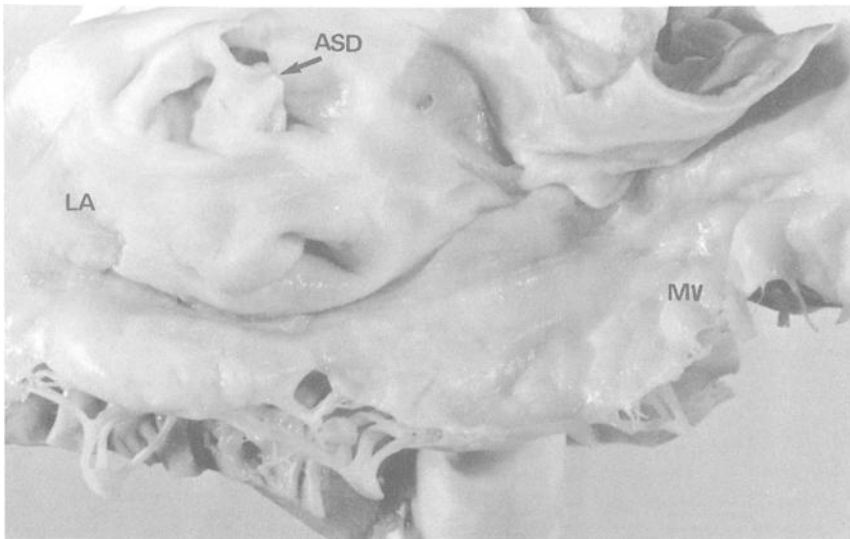
A mitral valve was considered *floppy* (mitral valve prolapse) on gross examination if it demonstrated thickening of the valve leaflets with an increased leaflet surface area and dilated annulus (Figs. 1 and 2). Manifestations included elongation and thinning of chordae tendineae, interchordal hooding or redundancy of the valve leaflet or leaflets, and involvement of either the anterior leaflet, the posterior leaflet, or both [12–15]. Further evidence in support of this diagnosis included the presence of ventricular endocardial friction lesions (Fig. 2). These were not necessary for the diagnosis, but were supportive evidence of valvular dysfunction [14]. In some cases, the valves demonstrated partial criteria for this diagnosis, for example, marked hooding of a portion of the posterior leaflet only. Such a valve was then classified as a “possible floppy valve.”

A coronary artery ostium was considered to demonstrate a *high take-off position* if its ostium rose 5 mm or more above the sinotubular junction (Fig. 3). A *conal* coronary artery was defined as an accessory or third vessel arising in the right sinus of Valsalva and supplying the conal area (that is, the outflow tract of the right ventricle) (Fig. 3) [16]. Intramural segments of the major branches of the coronary arteries, that is, those surrounded by bridges of cardiac muscle at least 1 mm in thickness for part or all of their course, were designated *tunneled coronary arteries* [17–22] (Fig. 4).

## Results

### Demographics

*Gender*—The study population consisted of 310 males, 153 females, and 7 cases in which the sex was not recorded. (Following routine cardiac evaluation at autopsy, 7 hearts were presented to the cardiac pathologist unlabeled by the morgue assistant.



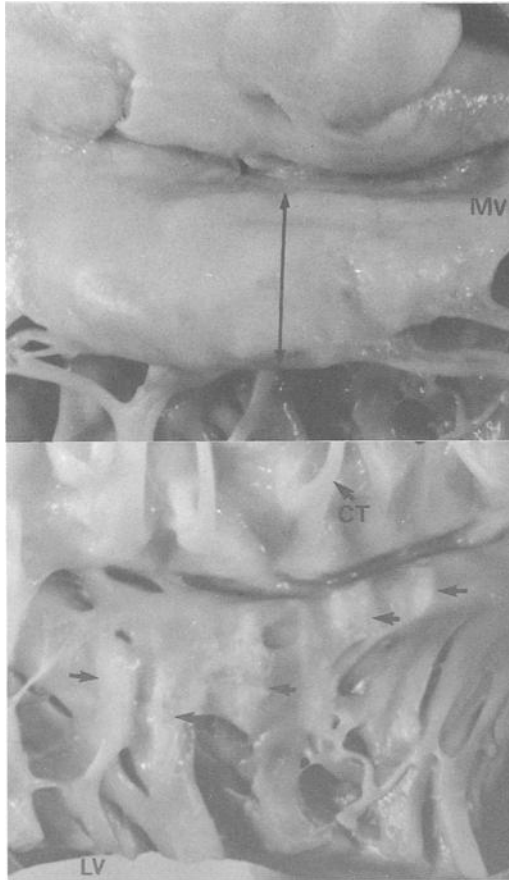


FIG. 2—Close-up of the floppy mitral valve (MV) shown in Fig. 1, and demonstrating leaflet “hooding” (long arrow, upper) and left ventricular (LV) endocardial friction lesions (small arrows, lower) underlying the chordae tendineae (CT).

Demographics could not be correlated with those specimens.) A significantly higher percentage of males was represented (67% of the study group, in comparison with the 51% of the total Marion County decedent population for a comparable time period [23]) ( $P < 0.0001$ ) (Fig. 5). There were no significant differences in the average ages between males and females.

*Age*—The age distribution of the decedents was also skewed, as is depicted in Fig. 6. Much younger individuals were represented in the coroner’s population than in the decedent population of Marion County as a whole, with the mode of the former at 30 to 39 years and the mode of the latter at 70 to 79 years. The ages of newborn to 99 years were represented in the study population, with a mean age of 38.7 years.

*Causes of Death*

The general causes of death for the 470 cases included in this study are depicted in Fig. 7. Fifteen percent of the deaths (71 cases) were diagnosed as cardiac in nature. Fifty-seven percent were included in the broad category of “traumatic,” which encompassed

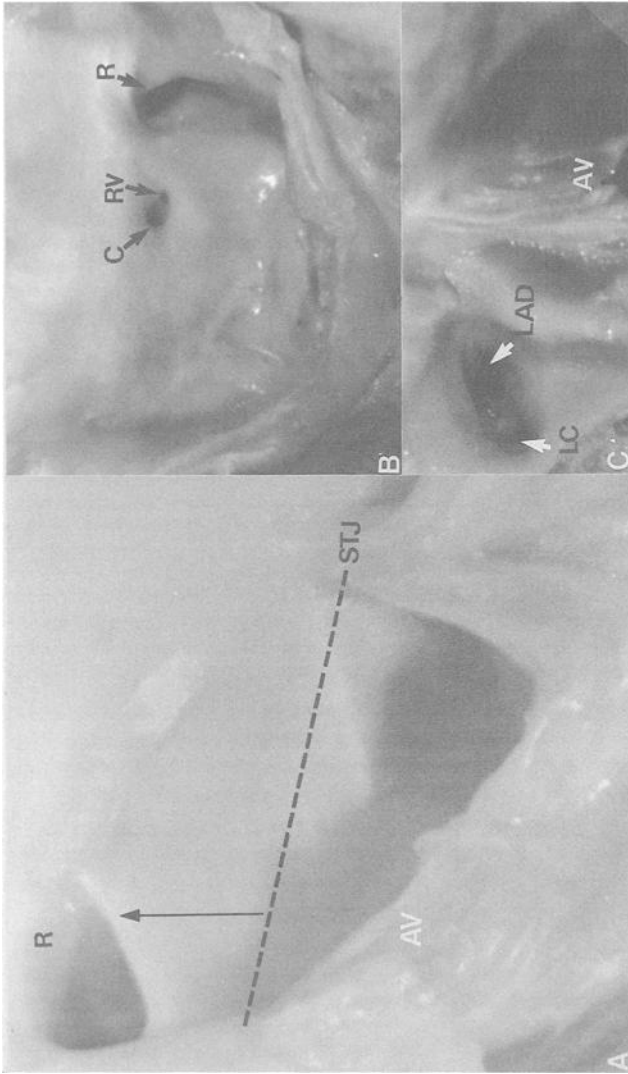


FIG. 3—Coronary artery anomalies: (a) the ostium of the right coronary artery (R) located above the level of the sinotubular junction (STJ) ("high take-off position"); (b) a conal right (C) artery and a smaller right ventricular branch (RV) artery adjacent to the ostium of the main right coronary artery (R); (c) the left circumflex (LC) and left anterior descending (LAD) coronary arteries arising as separate coronary ostia in the left sinus of Valsalva. The left main coronary artery is absent. AV = aortic valve.

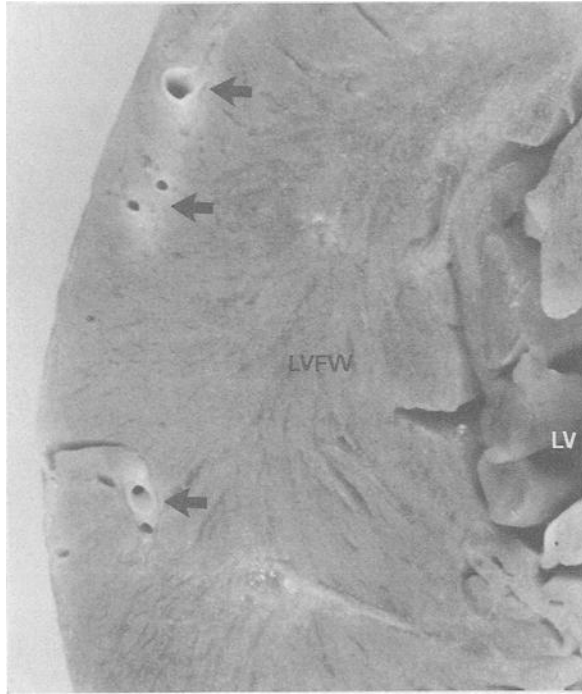


FIG. 4—Several intramural or "tunneled" branches of the left circumflex coronary artery are shown within the left ventricular free wall (LVFW). LV = left ventricular cavity.

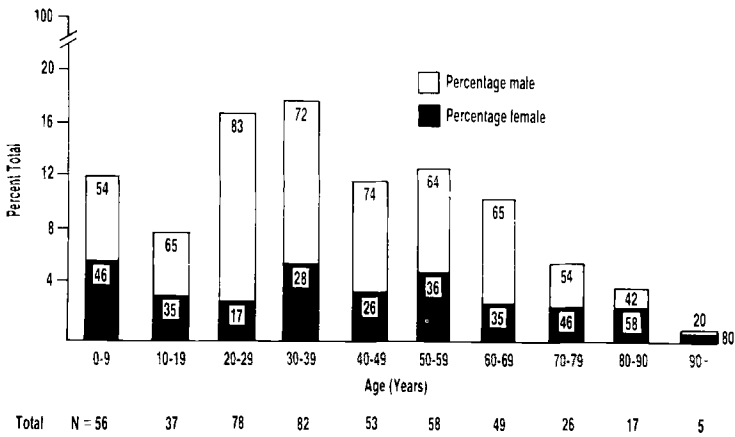


FIG. 5—Age and sex distribution for 470 necropsy patients.

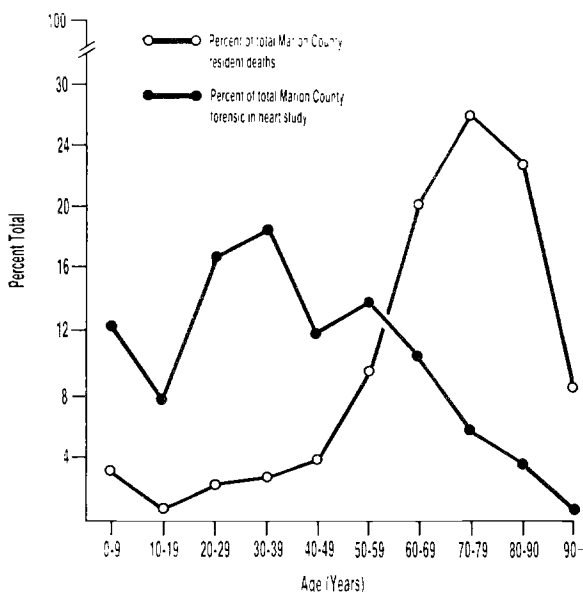


FIG. 6—Graph showing the age distribution of the study patients in comparison with that of patients in Marion County, IN.

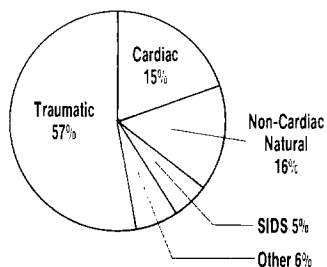


FIG. 7—Breakdown of various causes of death.

homicides, suicides, and accidents. The remaining causes were noncardiac natural deaths (16%), sudden infant death syndrome (5%), and other (6%).

*Cardiac Versus Noncardiac Deaths*—Cardiac deaths were attributed as due to either acquired or congenital conditions. Acquired and congenital conditions were subclassified under coronary, myocardial, and valvular etiologies (Tables 1 and 2). Of the total cardiac deaths, 55% were classified as sudden and unexpected (Fig. 8).

Those cases demonstrating cardiac causes of death are depicted in Table 3. The age distribution of cardiac deaths is skewed to the right, representing a larger percentage of the unexpected deaths of middle-aged and elderly individuals. Table 4 depicts the distribution of cardiac deaths in relation to gender. The majority of cases of atherosclerotic coronary heart disease (ACHD)—that is, acute myocardial infarctions and atherosclerosis of the coronary arteries—were seen in male victims in this study at 85 and 77%, respectively. This was not statistically significant, but reflects the male predominance in this population. However, more females than males died of cardiomyopathy ( $P < 0.01$ ).

TABLE 1—Major and minor congenital cardiac findings.

Cardiac Findings	Total No.	Percent
MAJOR CONGENITAL		
Complex <sup>a</sup>	4	0.8
Floppy mitral valve <sup>b</sup>	22	5.0
Bicuspid aortic valve	3	0.6
Atrial septal defect	1	0.2
Ventricular septal defect	1	0.2
MINOR CONGENITAL		
Tunneled coronary artery	138	29.0
Conal right coronary artery	120	26.0
Patent foramen ovale	86	18.0
High take-off coronary ostium	39	8.0
Absent left main coronary artery	2	0.4
Bicuspid pulmonic valve	1	0.2
Quadricuspid pulmonic valve	1	0.2

<sup>a</sup>Tetralogy of Fallot; hypoplastic left ventricle and aortic atresia; endocardial fibroelastosis (4 lesions in 3 cases).

<sup>b</sup>Includes 19 true floppy mitral valves and 3 possible floppy valves.

TABLE 2—Major and minor acquired cardiac findings.

Cardiac Findings	Total No.	Percent
MAJOR ACQUIRED		
Atherosclerotic coronary heart disease		
Severe occlusive disease <sup>a</sup>	77	16.0
Acute myocardial infarction	15	3.0
Healed myocardial infarction	30	6.0
Left ventricular aneurysm	1	0.2
Cardiac trauma	36	8.0
Pericardial lesions	7	1.5
Cardiomyopathy <sup>b</sup>	6	1.2
Myocarditis	4	0.8
Mitral stenosis (rheumatic)	3	0.6
Aortic stenosis (rheumatic)	1	0.2
MINOR ACQUIRED		
Cardiac adiposity	101	22.0
Left ventricular hypertrophy	61	13.0
Right ventricular dilation	31	7.0
Right ventricular hypertrophy	21	4.7
Mitral valve annular calcium	18	4.0
Aortic stenosis (degenerative)	7	1.5
Neoplasm <sup>c</sup>	2	0.4

<sup>a</sup>Coronary atherosclerosis involving greater than 75% of the cross-sectional area of a vessel or occlusive thrombus.

<sup>b</sup>Dilated (4 cases at 0.8%); hypertrophic (2 cases at 0.4%).

<sup>c</sup>Both were papillomas (see text).



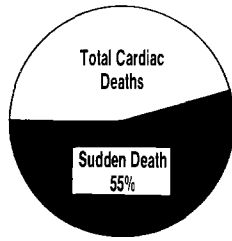


FIG. 8—Percentage of sudden deaths occurring among patients with cardiac death.

TABLE 3—Cardiac causes of death by age.

Age	AMI. No. (%)	ACHD. No. (%)	ARR. No. (%)	CM. No. (%)	MITIS. No. (%)	Rheumatic. No. (%)	FMV. No. (%)	CHD. No. (%)
0 to 9 years	0	0	0	0	0	0	0	3 (100)
10 to 19 years	0	0	0	1 (11)	0	0	0	0
20 to 29 years	1 (5)	0	0	0	0	0	1 (33)	0
30 to 39 years	3 (15)	4 (13)	1 (33)	0	1 (100)	0	1 (33)	0
40 to 49 years	3 (15)	6 (19)	1 (33)	1 (11)	0	0	1 (33)	0
50 to 59 years	7 (35)	5 (16)	0	3 (33)	0	0	0	0
60 to 69 years	4 (20)	10 (32)	0	4 (44)	0	0	0	0
70 to 79 years	2 (10)	4 (13)	0	0	0	1 (100)	0	0
80 to 90 years	0	2 (6)	1 (33)	0	0	0	0	0
90+ years	0	0	0	0	0	0	0	0
Total (71)	20	31	3	9	1	1	3	3
Sudden death	· · ·	25	2	7	0	1	2	0

TABLE 4—Cardiac deaths—sex distribution.

Cardiac Findings	Total No.	Female. %	Male. %
Acute myocardial infarction	20	15	85
Coronary heart disease	31	23	77
Congenital heart disease	3	33	67
Cardiomyopathy	9	78	22
Myocarditis	1	0	100
Floppy mitral valve	3	67	33
Rheumatic heart disease	1	0	100
(Arrhythmia)	3	0	100

### Major and Minor Cardiovascular Findings

*Major*—The most prevalent *acquired major* finding was ACHD (16%). Manifestations of ACHD included acute and healed myocardial infarction (9%) and ventricular aneurysm (0.2%). Other major acquired findings included cardiac trauma (8%) and cardiomyopathy (1%).

Floppy mitral valve represents the most common *congenital major* anomaly, seen in 22 cases (5%) (19 cases were unequivocally floppy; 3 were possibly floppy). One percent of the cases (4 individuals) had congenitally bicuspid aortic valves.

*Minor*—The most common *acquired minor* findings were excess surface adiposity (25%) and left ventricular hypertrophy (13%).

Of the *congenital minor* findings, tunneled coronary arteries (29%) and conal right coronary arteries (26%) were the most common. High take-off ostial position was identified in 8% of the hearts. Nearly 20% of the cases demonstrated “probe” patency of the foramen ovale.

### Discussion

The major goal of this study was to focus on cardiovascular findings in a group of deaths examined by forensic pathologists. To our knowledge, this is the first study to evaluate and tabulate sequentially all gross cardiac findings. Previous studies have evaluated a series of cases for specific factors (for example, cause of death [24,25], sudden death [25–29], and aging changes [30]); for specific diagnoses, such as floppy mitral valve [12,14,15,31]; or for the characteristics of a certain subpopulation (for example, children [3,32,33] and athletes [9,34]).

The case load in this series significantly overrepresented males, when compared with the decedent population of Marion County, Indiana, as a whole (Fig. 6). This skewed gender distribution has been seen in other forensic autopsy studies [24,35,36] and may result in the underrepresentation in this study of any pathologic conditions that are more prevalent in females.

It is of interest that cardiac deaths accounted for only 15% (71 cases) of the study population. This contrasts with the 32% of the total Marion County deaths ascribed to cardiovascular causes [23] in 1987 and with the estimated 48% with cardiovascular causes out of the total deaths in the United States (1985) [1]. In addition to the excess “traumatic” deaths in our subpopulation, many of the forensic cases “signed out” without autopsies were diagnosed as cardiovascular based on the history and external physical examination. (In 1988, of 597 Marion County coroner’s cases “signed out,” 225 [38% of sign-outs] were considered cardiovascular deaths.) Thus, the total forensic population contains a greater percentage of deaths attributed to cardiac causes than those autopsied. Conversely, it is known that a certain percentage of causes of death that are listed as cardiovascular on the death certificate in any population would be disproven at autopsy [24]. Thus, without a 100% autopsy rate, an accurate assessment of the incidence of any disease process is difficult.

### Expected Findings

*Coronary Artery Disease*—Major findings in this series that were *expected* in light of current medical knowledge and literature review included the high frequency of ACHD and the higher frequency of men (than women) who manifest this illness [1,25,28].

*Floppy Mitral Valve*—A 4 to 5% incidence of floppy mitral valve in our population is in keeping with that described for the population as a whole [12–15,31,37]. Thirteen of

the cases were males (nine females), which possibly reflects the higher incidence of males in our study population.

The incidence of conal right coronary arteries, as well as of high take-off of coronary arteries, at 26 and 8%, respectively, is similar to that published in previous reports [16,20]. An incidence of 1% (three cases) with bicuspid aortic valves is consistent with previously reported studies [38].

*Rheumatic Heart Disease*—One cardiac death secondary to rheumatic heart disease was seen in our population (Table 1). Only one rheumatic aortic, and three rheumatic mitral valves were recognized. These findings reflect the reported decrease in incidence of this entity currently in the United States [39].

### *Unexpected Findings*

*Myocarditis*—Several studies have reported an incidence of myocarditis ranging from approximately 7 to 12%, particularly in pediatric cases [3,33,35,40]. The frequency of this diagnosis in our study was only 0.8%. Factors which might explain this difference in frequency of myocarditis include the number of cardiac sections evaluated microscopically and the definition of myocarditis employed. Each case of myocarditis in our series occurred in individuals over 30 years of age.

*Tunneled Coronary Arteries*—The existence of the “bridged” or “tunneled” coronary artery has been acknowledged in the literature for approximately 200 years [22]; the first discussion implicating this entity as a possible mechanism in disease appeared in 1950 [22]. Since then, the incidence of this anatomic variation has been reported in between 5.4 and 85.7% of cases [20–22]. The early work of Geiringer [22] implicated these vessels as exerting a protective influence against the formation of atherosclerosis, but this hypothesis has since been debated [21]. Tunneled coronary arteries have been suggested as the etiology of symptoms such as ventricular arrhythmias, chest pain, and myocardial infarction [19], as well as being implicated in atherogenesis [20] and sudden death [17,18]. Our results confirm the high frequency (30%) of this entity in our study population, but mitigate against the likelihood of it being relevant to myocardial injury or cardiac death.

Hypertrophic cardiomyopathy has been implicated as a common cause of sudden or unexpected death, particularly in young individuals [3,9,40]. In our study group, only two cases of hypertrophic cardiomyopathy were seen. This constitutes only 0.05% of sudden deaths in our series.

It is of interest that only 8% of the hearts examined in this study were identified as grossly “normal” when standard anatomic criteria were employed.

### **Summary**

In an attempt to identify all cardiac findings, whether insignificant or directly related to the cause and manner of death, all hearts in a forensic pathology service were systematically examined for a total of 470 cases. The purpose was to tabulate the major and minor cardiovascular findings in this population, which generally reflects nonhospitalized and previously “well” individuals. Ninety-two of the hearts studied had at least one abnormality. Major findings included floppy mitral valves and atherosclerotic coronary heart disease. Minor findings, of interest due to their frequency in this population, included the tunneled coronary arteries and cardiac adiposity. Thus, a forensic service contains a vast array of cardiovascular conditions.

### *Acknowledgments*

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**References**

- [1] 1988 *Heart Facts*. American Heart Association, Dallas, TX, 1988.
- [2] Gillum, R. F., "Sudden Coronary Death in the United States 1980-1985," *Circulation*, Vol. 79, No. 4, April 1989, pp. 756-765.
- [3] Rajs, J., "Cardiovascular Abnormalities in Children: A 10-Year Forensic Pathology Study," *Journal of Forensic Sciences*, Vol. 30, No. 4, Oct. 1985, pp. 1157-1178.
- [4] Kannel, W. B. and Schatzkin, A., "Sudden Death: Lessons from Subsets in Population Studies," *Journal of the American College of Cardiology*, Vol. 5, No. 6, June 1985, pp. 141B-149B.
- [5] Waller, B., "Pathology of Acute Myocardial Infarction: Definition, Location, Effects of Reperfusion, Complications and Sequelae," *Cardiology Clinics*, Vol. 6, No. 1, Feb. 1988, pp. 1-28.
- [6] Virmani, R. and Roberts, W. C., "Sudden Cardiac Death," *Human Pathology*, Vol. 18, No. 5, May 1987, pp. 485-492.
- [7] Edwards, W. D., "Cardiomyopathies," *Human Pathology*, Vol. 18, No. 6, June 1987, pp. 625-635.
- [8] Silver, M. and Silver, M., "Cardiomyopathies," *Cardiovascular Pathology*, M. Silver, Ed., Churchill Livingstone, New York, 1983, Chapter 11, pp. 489-516.
- [9] Maron, B. J., Roberts, W. C., McAllister, H. A., Rosing, D. R., and Epstein, S. E., "Sudden Death in Young Athletes," *Circulation*, Vol. 62, No. 2, Aug. 1980, pp. 218-229.
- [10] McAllister, H. A., Hall, R. J., and Cooley, D. A., "Surgical Pathology of Tumors and Cysts of the Heart and Pericardium," *Contemporary Issues in Surgical Pathology*, Vol. 12, 1988, pp. 343-366.
- [11] Prichard, R. W., "Tumors of the Heart," *Archives of Pathology*, Vol. 51, 1951, pp. 98-128.
- [12] Virmani, R., Atkinson, J. B., Forman, M. B., and Robinowitz, M., "Mitral Valve Prolapse," *Human Pathology*, Vol. 18, No. 6, June 1987, pp. 596-602.
- [13] Jeresaty, R., "Mitral Valve Prolapse: An Update," *Journal of the American Medical Association*, Vol. 254, No. 6, 9 Aug. 1985, pp. 793-795.
- [14] Lucas, R. and Edwards, J., "The Floppy Mitral Valve," *Current Problems in Cardiology*, Vol. 7, No. 1, July 1982, pp. 5-48.
- [15] Davies, M. J., Moore, B. P., and Brainbridge, M. V., "The Floppy Mitral Valve," *British Heart Journal*, Vol. 40, 1978, pp. 468-481.
- [16] Blake, H. A., Manion, W. C., Mattingly, T. W., and Baroldi, G., "Coronary Artery Anomalies," *Circulation*, Vol. 30, Dec. 1964, pp. 927-940.
- [17] Cheitlin, M. D., "Editorial: The Intramural Coronary Artery: Another Cause for Sudden Death with Exercise?" *Circulation*, Vol. 62, No. 2, Aug. 1980, pp. 238-239.
- [18] Morales, A. R., Romanelli, R., and Boucek, R. J., "The Mural Left Anterior Descending Coronary Artery, Strenuous Exercise and Sudden Death," *Circulation*, Vol. 62, No. 2, Aug. 1980, pp. 230-237.
- [19] Faruqui, A. M., Maloy, W. C., Felner, J. M., Schlant, R. C., Logan, W. D., and Symbas, P., "Symptomatic Myocardial Bridging of the Coronary Artery," *American Journal of Cardiology*, Vol. 41, June 1978, pp. 1305-1310.
- [20] Polacek, P., "Relation of Myocardial Bridges and Loops on the Coronary Arteries to Coronary Occlusions," *American Heart Journal*, Vol. 61, No. 1, Jan. 1961, pp. 44-52.
- [21] Edwards, J. C., Burnside, C., Swarm, R. L., and Lansing, A., "Arteriosclerosis in the Intramural and Extramural Portions of Coronary Arteries in the Human Heart," *Circulation*, Vol. 13, Feb. 1956, pp. 235-241.
- [22] Geiringer, E., "The Mural Coronary," *American Heart Journal*, Vol. 41, 1951, pp. 359-368.
- [23] Health Department Data, Marion County, IN, 1987.
- [24] Asnaes, E. and Paaske, F., "Uncertainty of Determining Mode of Death in Medicolegal Material Without Autopsy—A Systematic Autopsy Study," *Forensic Science International*, Vol. 15, 1980, pp. 3-17.
- [25] Luke, J. and Helpert, M., "Sudden Unexpected Death from Natural Causes in Young Adults," *Archives of Pathology*, Vol. 85, Jan. 1968, pp. 10-17.
- [26] Penttala, A. and Ahonen, A., "The Epidemiology of Autopsies in Cardiovascular Deaths of Middle-Aged Men in Finland in 1973," *Forensic Science International*, Vol. 13, 1979, pp. 239-251.
- [27] Roberts, W. C. and Jones, A., "Quantitation of Coronary Arterial Narrowing at Necropsy in Sudden Coronary Death," *The American Journal of Cardiology*, Vol. 44, July 1979, pp. 39-45.
- [28] Baroldi, G., Falzi, G., and Mariani, F., "Sudden Coronary Death: A Postmortem Study in 208 Selected Cases Compared to 97 'Control' Subjects," *American Heart Journal*, Vol. 98, No. 1, July 1979, pp. 20-31.
- [29] Marek, Z., "Morphological Changes in the Myocardium as Substrate of 'Functional Assymetry' in Sudden Death," *Forensic Science*, Vol. 1, 1972, pp. 427-436.

- [30] Pomerance, A., "Ageing Changes in Human Heart Valves," *British Heart Journal*, Vol. 29, 1967, pp. 222-231.
- [31] Scala-Barnett, D. and Donoghue, E., "Sudden Death in Mitral Valve Prolapse," *Journal of Forensic Sciences*, Vol. 33, No. 1, Jan. 1988, pp. 84-91.
- [32] Akman, D., Berenson, G. S., Blonde, C. V., Webber, L. S., and Stopa, A. R., "Heart Disease in a Total Population of Children: The Bogalusa Heart Study," *Southern Medical Journal*, Vol. 75, No. 10, Oct. 1982, pp. 1177-1181.
- [33] Noren, G. R., Staley, N. A., Bandt, C. M., and Kaplan, E. L., "Occurrence of Myocarditis in Sudden Death in Children," *Journal of Forensic Sciences*, Vol. 22, No. 1, Jan. 1977, pp. 188-196.
- [34] Virmani, R. and Robinowitz, M., "Cardiac Pathology and Sports Medicine," *Human Pathology*, Vol. 18, No. 5, May 1987, pp. 493-501.
- [35] Topaz, O. and Edwards, J., "Pathologic Features of Sudden Death in Children, Adolescents and Young Adults," *Chest*, Vol. 87, No. 4, April 1985, pp. 476-482.
- [36] Rokkanen, P. and Slatis, P., "Causes of Death After Severe Trauma," *Annales Chirurgiae et Gynaecologiae Fenniae*, Vol. 56, 1967, pp. 313-318.
- [37] Perloff, J. K. and Child J. S., "Clinical and Epidemeologic Issues in Mitral Valve Prolapse: Overview and Perspective," *American Heart Journal*, Vol. 113, No. 5, May 1987, pp. 1324-1332.
- [38] Schoen, F. J. and Sutton, M. S., "Contemporary Issues in the Pathology of Valvular Heart Disease," *Human Pathology*, Vol. 18, No. 6, June 1987, pp. 568-576.
- [39] Gordis, L., "The Virtual Disappearance of Rheumatic Fever in the United States: Lessons in the Rise and Fall of Disease," *Circulation*, Vol. 72, No. 6, Dec. 1985, pp. 1155-1162.
- [40] Schwartz, C. J. and Walsh, W. J., "The Pathologic Basis of Sudden Death," *Progress in Cardiovascular Disease*, Vol. 13, No. 5, March 1971, pp. 465-481.

Address requests for reprints or other information to  
Michael A. Clark, Ph.D., M.D.  
Room 157, Medical Science Building  
635 Barnhill Drive  
Indianapolis, IN 46202-5120