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Sudden Unexpected Death from Cardiac Concussion (Commotio Cordis) with Unusual Legal Complications

The clinical concept of cardiac concussion (commotio cordis) has been known for many centuries; however, it has received little attention since it was reviewed in the 1940s. Blunt force injuries to the chest wall causing damage to the heart are observed in injuries involving vehicular accidents, sports, and all activities entailing impact or crushing. Occasionally, little or no evidence of pathologic change is observed grossly or microscopically. The clinical course may depend on the degree of involvement of the conductive system of the heart, with death usually a result of ventricular fibrillation. Good clinical documentation of the events before death and a complete pathologic evaluation are necessary if the cause of death is to be determined. This paper reports a case of blunt force trauma to the anterior chest wall resulting in sudden death, unexpected legal complications arising from improper embalming of the body, and the pathologist's need to consider legal problems arising from his activities.

Case Report

A 15-year-old boy was playing softball when he was struck in the mid-sternal region by a fly ball. Within moments after the impact, he suffered a cardiopulmonary arrest. Cardiopulmonary resuscitative measures were immediately started, but spontaneous respiration was not established for 1½ h. A palpable pulse returned only after almost 3 h of cardiac massage. Epinephrine, calcium gluconate, bicarbonate solution, dopamine, and Levophed® bitartrate (levarterenol bitartrate) were administrated at the scene, and the boy was air-evacuated to the Arizona Health Sciences Center (AHSC). During the resuscitative measures, he was "cardioverted" 19 times with a 300-W·s dose. During the 4½ h of hospitalization, he was given lidocaine and dopamine. His blood pressure stayed low, and he demonstrated paradoxical pulses. Cerebral edema that developed shortly after admission was treated with intravenous Decadron® (dexamethasone sodium phosphate). Seven hours after the initial trauma, he developed ventricular fibrillation that failed to respond to further resuscitative measures.

The autopsy findings indicated the body was that of a well-developed, well-nourished, muscular Papago Indian boy. He had several small abrasions on the face, left arm, and right knee. His anterior chest wall showed no injury, though numerous small scars appeared on all parts of the body; moreover, there was no evidence of internal injury. The heart was enlarged, weighing 270 g, and the endocardium was smooth and glisten-

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ing but appeared thicker than normal with a gray-white opacity. The valves appeared normal. However, a small area of contusion, 0.4 cm in diameter, was noted on the epicardial surface in the septal region of the left ventricle. The lungs weighed about 750 g each, exhibiting prominent edema and congestion. Examination of the brain showed tonsillar herniation with flattened sulci. Several small foci of myocardial hemorrhage and chronic inflammation were observed microscopically. Fibrous connective tissue thickened the endocardial area, and subendocardial fibrosis was observed (Fig. 1), as were small foci of interstitial fibrosis. Smooth muscle hyperplasia of the bronchial walls, thickening of the respiratory epithelial basement membranes, and infiltration of eosinophils and mast cells in the bronchial walls revealed a previously undiagnosed asthmatic condition (Fig. 2). Death was attributed to the nonpenetrating, blunt cardiac trauma with an underlying cardiomyopathy and asthma.

Discussion

Every sudden and unexpected death has an actual or potential medicolegal aspect of great interest. Such deaths may result directly from trauma or from a disease, perhaps unsuspected, that contributes to the traumatic death. Cardiovascular diseases result in more such deaths than diseases of any other organ system [*1-3*]. In addition, determining whether trauma or disease was a sufficient cause of death may not be easy, for the questions asked must involve cause and effect relationships between trauma and the disease.

Schlomka and Hinrichs [*4*] used the phrase "cardiac concussion" (commotio cordis) in their clinical and experimental observations beginning in 1932. A decade later, the phenomenon was rigorously defined by Hediger [*5*], but little heed was paid to it until Golikov and Borisenko [*6*] reviewed the observations of the many cases of blunt force cardiac trauma at the Sklifosovski Institute in Moscow. As a result of these observations, they made a clear clinical distinction between cardiac concussion and myocardial contusion (Table 1).

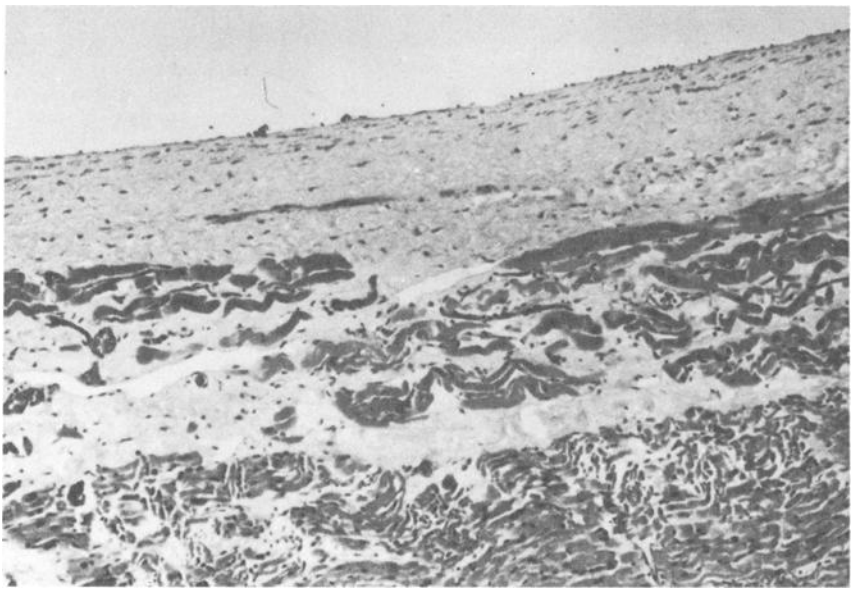

FIG. 1—*Endocardial and subendocardial fibrosis (hematoxylin and eosin stain).*

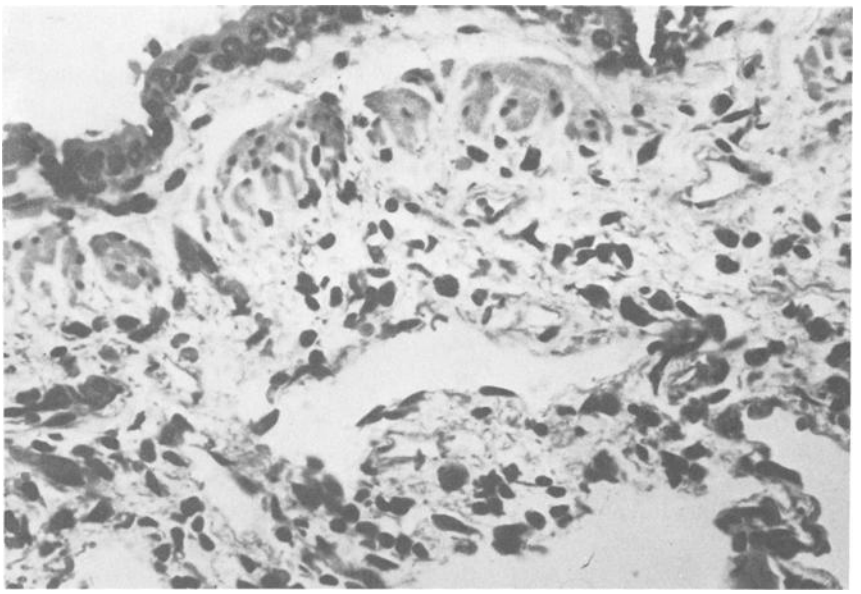

FIG. 2—*Histologic changes consistent with asthma. Smooth muscle hyperplasia and thickened respiratory epithelial basement membranes of bronchioles (hematoxylin and eosin stain).*

TABLE 1—*Differentiation between cardiac concussion and cardiac contusion.*[a]

Data	Concussion	Contusion
Site of trauma	precordial only	chest, anywhere
Direction of blow	sternum to vertebrae	of no significance
Force	sharp; not necessarily violent	generally violent
Thoracic cage intact	essential	not essential
Onset	immediate	gradual
Course	transitory	lasting
Loss of consciousness	as a rule	not characteristic
Blood pressure	frequently drops	normal; rarely up
Disturbances of rhythm and conduction	characteristic; immediate	absent or delayed
Changes in S-T segment and T wave	generally absent	always present

[a] Modified from Golikov and Borisenko [6] by Lindsey [7].

In the boy's case, a minute area of epicardial hemorrhage was noted in the septal region of the left ventricle. The lesion was not associated with a major conduction tract and could have resulted from the cardiopulmonary resuscitative attempts, and thus it could not be regarded as evidence of a true cardiac contusion. Golikov and Borisenko [6] classified cardiac concussion as the least severe of a series of consequences of blunt cardiac injury, a series including cardiac concussion, myocardial contusion, traumatic cardiodystrophy, myocardial infarction, and rupture (most severe). This listing indicates that cardiac concussion is a normally transient and benign condition and is consistent with the findings observed at the AHSC that levels of phosphokinase MB isoenzymes rose transiently, without other evidence of cardiac injury, in a significant proportion of drivers

involved in auto accidents [7]. The cardiac isoenzymes were studied after the boy's death in this case and, although levels were elevated, no conclusions could be drawn because of the repeated cardioversions.

The experimental observations of Liedtke et al [8], however, suggest that cardiac concussion can be fatal. The typical course of cardiac rhythm after a graded blow to the heart is complete heart block with ventricular arrest reverting via ventricular tachycardia to sinus rhythm. In some of their cases, as in this one, the ventricular tachycardia progresses to ventricular fibrillation and death. Viano and Artiman [9], from experimental data based on the analysis of electrocardiograms of twelve anesthetized pigs who were struck with blunt force on the chest, were recently able to demonstrate that all the animals developed some degree of conducting system dysfunction, intraventricular conduction defects, or ventricular fibrillation. The findings, again as in this case, did not correspond to the more common biomechanical "indicators" of the Abbreviated Injury Scale [10].

The energy transmitted to the boy's precordium by the impact of the softball cannot, of course, be determined in retrospect, but an upper limit can be set. A professional ball player's line drive to shortstop delivers a force of almost 120 J. Such a force should produce no demonstrable morphologic damage [11].

McAllister at the Armed Forces Institute of Pathology[2] reviewed the case and concurred with the pathologic findings of an idiopathic fibrotic cardiomyopathy manifested by subendocardial fibrosis, myocarditis, and focal interstitial fibrosis. The cause of the disease entity is obscure but may be due to intrauterine anoxia, stress reaction, genetic metabolic defects, inflammatory disease, or abnormal differentiation of the bulbus cordis. In this case, it was impossible to determine the actual cause of the fibrotic process, but it is thought that trauma to the heart and an asthmatic condition contributed to the death. Though neither by itself may have been sufficient to cause death, the combination perhaps was.

The case of the "fatal fly ball," as it was later called, did not cease upon completion of the postmortem examination and release of the body to the funeral home. The boy's family complained to the Pima County Attorney's Office, which initiated a series of legal actions. The complaint stated that the body was inadequately or improperly embalmed and that it was in a state of advanced decomposition which required further preparation by another funeral home. The matter was referred to the federal authorities because it involved a complaint from an Indian reservation. The Federal Bureau of Investigation determined that no federal law was violated. Later, a letter, allegedly soliciting business in violation of state law, was sent by the funeral director to the tribal chairman. As a result, the Attorney General of the State of Arizona began an investigation of the entire case. He requested the State Board of Funeral Directors and Embalmers to investigate the claims that state health codes had been violated by the use of improper embalming techniques and that business had been illegally solicited. The family also filed a tort action for damages against the funeral home.

Four of the five members of the Board of Funeral Directors and Embalmers heard 14 h of testimony in which charges of improper embalming techniques and attempted fraud were recounted. Two of the authors appeared at the hearing. The senior author was examined for 2½ h and explained how the case was managed from the time of death to the release of the autopsied body, how the autopsy was performed, how the body appeared upon release, and how the postmortem interval might have affected decomposition. Proper management and autopsy techniques prevented any countercharges by the defendant. Ultimately, the license of the mortuary was suspended, and the embalmer's license was permanently revoked.

[2] Personal communication, 1977.

Summary

Sudden and unexpected death may result from cardiac concussion following blunt force trauma to the thorax. Undiagnosed pathologic disease must be carefully evaluated as a possible contributory element. Legal complications may arise from any autopsy. It is recommended that a photograph be taken upon completion of the autopsy. This photograph and adequate records can be used to refute any charges against the pathologist or assistants for the poor condition of a body after its release.

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