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# Trauma and Heart Disease

by

*Alan R. Moritz*

WITH LEGAL ANNOTATIONS BY OLIVER SCHROEDER, JR.\*

**D**IFFERENCES of opinion in respect to the part played by trauma or stress in the causation of heart disease or in the causation of the failure of the diseased heart are responsible for an enormous volume of litigation. It is clear from reviewing the evidence presented before workman's compensation boards and trial courts in such cases that many attorneys know too

little about the causal relationships that may or may not exist and that many doctors are insufficiently critical in distinguishing between medical possibility and medical probability. The following discussion

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of the problem is directed at attorneys in the hope that it will help them to a better understanding of this rather complex and controversial group of medicolegal problems.

In the succeeding discussion the following relationships between trauma and heart disease are considered:

Trauma resulting in direct injury to the heart.

Trauma, exertion or emotional disturbance resulting in increase of heart rate or increase in blood pressure, either of which may increase the work of and cause failure of an already diseased heart.

Trauma resulting in shock and slowing of blood flow which may predispose to the occurrence of thrombosis in previously diseased arteries.

Trauma resulting in increased coagulability of the blood which may predispose to the occurrence of thrombosis in previously diseased coronary arteries.

Trauma resulting in immobilization of an extremity or in confinement to bed which may predispose to pulmonary thrombo-embolism.

Trauma resulting in fracture or in damage to fatty tissue which may result in pulmonary fat embolism.

Trauma complicated by infection which may localize in the heart, thereby resulting in heart disease.

Trauma caused by unexpected collapse resulting from heart disease.

Trauma preceding heart disease or heart failure and not causally related to either.

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*Trauma as the Cause of Heart Disease*

The only circumstance in which mechanical violence (trauma) can be said to be the direct cause of cardiac injury or disease is when the heart has been wounded by an object that has penetrated the body or has been bruised or crushed by a violent impact against the body.

If the heart or the pericardium<sup>1</sup> is perforated or lacerated by a sharp object, or by a free missile, it may fail immediately from the shock of the impact or may fail gradually because of the ensuing hemorrhage or infection. In such instances there is rarely any doubt as to the causal relationship between the injury and the ensuing heart failure. If such an injury is survived by as long as a week without there being clear medical evidence of hemorrhage or infection within the pericardial sac, it is unlikely that any subsequently recognized cardiac disturbance could be reasonably attributed to the injury.

If a violent impact against the chest is sustained, the heart may be bruised or crushed. In persons over middle age the ribs are sufficiently rigid that it would be most unlikely for the heart to be bruised unless the bony cage of the chest were fractured. In young individuals, it is possible for a blunt impact against the chest to damage the heart without breaking any of the bones. If the heart has sustained a crushing or bruising injury, there is not likely to be any symptom-free period between the injury and unmistakable evidence of cardiac damage. Unless the injury has caused immediate heart failure or immediate and continuous impairment of cardiac function, it can be assumed that no direct damage to the heart was sustained.

It is frequently claimed that disability or death from coronary thrombosis is causally related to a blunt injury of the chest which has bruised one of the coronary arteries on the surface of the heart.<sup>2</sup> It is possible for a coronary artery to be damaged by a blunt injury if the injury has been such as to cause a broken bone to press against the surface of the heart. It is conceivable, although not probable, that such injury might lead to the clotting of blood within the damaged vessel. If this happens, it could be said that the injury has caused coronary thrombosis, and if the damaged vessel were a large one, heart failure would probably result. If such a relationship is to be established, it would be necessary for the signs and symptoms of coronary thrombosis to appear almost immediately after the injury. Even in this event, the cause-and-effect relationship would remain a matter of speculation unless established by postmortem examination.

<sup>1</sup>The pericardium is the fibrous membrane which covers the heart.

<sup>2</sup>The two arteries that supply the heart muscle with blood are the coronary arteries. The formation of a blood clot within a coronary artery, thus interfering with the flow of blood through it, is called coronary thrombosis.

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Normal or Diseased Heart



Penetrating or blunt  
injury of chest



May bruise or  
wound heart.



If ensuing signs or symptoms of heart failure are due to direct damage to heart, they should be apparent within seconds or minutes or at the most within a few hours.

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*Trauma or Stress as the Cause of Failure of an Already Diseased Heart*

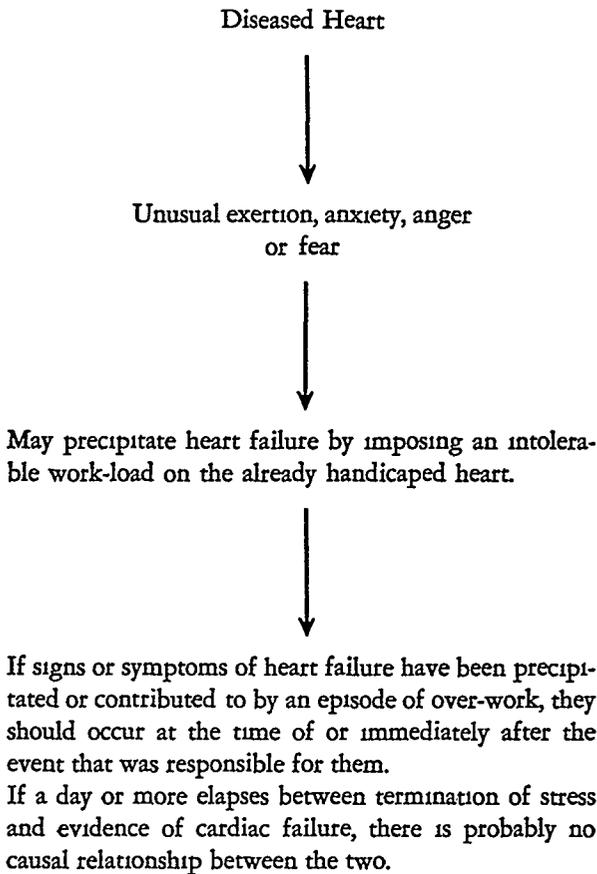
Injuries to any part of the body may, and frequently do, result in extra work for the heart. Such a sudden increase in work may cause a diseased heart to fail. It is not necessary that a wound be produced in order to cause extra work load for the heart. An episode of violent exertion, fear, pain, anger or anxiety may greatly increase the work of the heart independently of trauma. This may come about by either one or a combination of two mechanisms. Exertion, injury or emotional disturbance may cause the heart to beat more rapidly than usual. Frequently the pulse rate is doubled following such an episode of stress. If the same amount of blood is discharged from the heart with each beat and if the rate is doubled, it is obvious that the heart will be performing twice as much work as it normally does. Another factor responsible for additional work for the heart as a consequence of injury, exertion or emotional disturbance is the rise in blood pressure which often accompanies such an event.<sup>8</sup> The cause of the increased blood pres-

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<sup>8</sup> In the case of *Wagner v. Lewis*, 13 N.J. Misc. 807, 181 Atl. 394 (1935), a 31 year old laborer, apparently strong and in good health, collapsed and died while sawing

sure is a generalized reflex spasm of small blood vessels which narrows the passage way through them, thus increasing the resistance to blood flow. This factor alone may double the work load of the heart. The combined effects of these two common reactions to injury or excitement may double, treble or even quadruple the burden of the heart.

Such an increase in work is usually of brief duration and does not damage a normal heart. It does not cause coronary thrombosis, persistently high blood pressure, persistently rapid pulse, nor does it cause or precipitate disease of a normal heart. It may, however, precipitate the failure of an already diseased heart.



timber. Only after the essential post mortem examination was it revealed that he had a diseased and weakened heart. It was found that his previously diseased heart failed because of the increased blood pressure that resulted from his physical exertion.

It might be well at this point to review the characteristics of spontaneous heart disease. There are three important forms of heart disease. One of the three involves the coronary arteries and is sometimes called arteriosclerotic heart disease. This term means that the coronary arteries which supply the heart muscle with blood are the seat of chronic degenerative changes which have caused thickening of their lining, thereby interfering with the flow of blood through them. In this condition the coronary arteries are not capable of dilating when the work of the heart is such as to require additional blood supply, so that the heart has a reduced reserve for meeting emergencies. Often the coronary arteriosclerosis is so severe that the heart does not receive enough blood to do its ordinary work. In the latter event, the heart may fail even though no additional burden is placed upon it. The spontaneous failure of a heart whose coronary arteries are diseased may be gradual or may be sudden and unexpected.

Sometimes this kind of heart disease (arteriosclerotic heart disease) is referred to as angina pectoris.<sup>4</sup> Angina pectoris denotes pain in the chest. Many people who have coronary arteriosclerosis are subject to periodic attacks of crushing or sharp pain in the chest which sometimes extends into the left shoulder or down the left arm. Instead of being in the chest, the pain may be in the upper abdomen, in which event it may be confused with the discomfort of indigestion. It should be remembered, first, that pain in the chest is often caused by diseases other than heart disease, and, second, that many people who actually die of coronary heart disease never complain in life of angina pectoris.

Still another name given to this form of heart disease is coronary thrombosis. The use of this term implies that the physician believes that the coronary arteries were not only narrowed by chronic disease but that their patency was still further reduced or obliterated by the formation of a blood clot at the site of severe arterial disease. Frequently it is the sudden plugging of the chronically diseased artery by such a blood clot or thrombus that causes the pain and the attack of heart failure that first calls attention to the disease. Neither trauma nor exertion cause coronary thrombosis al-

<sup>4</sup>Some courts have erroneously credited angina pectoris with causing death while in reality it is merely the pain symptom of the serious heart disorder called "arteriosclerosis." See *MacDonald v. Metropolitan Street Ry.*, 219 Mo. 468, 118 S.W. 78 (1909); *Haviland v. Industrial Comm'n*, 135 Ohio St. 545, 21 N.E.2d 658 (1939). Breaking coal with a two-pound hammer for an hour and a half, a task more arduous than normal duties, has been held to be sufficient to cause the severe pain and unconsciousness of an angina pectoris attack. *Hoon v. State Ins. Fund*, 149 Pa. Super. 236, 27 A. 2d 776 (1942) (but the experience was held not to be an "accident" within the Workmen's Compensation statutes). In the *Haviland* case it was stated that some indication of pain or suffering during the angina pectoris attack is a probable expectation: in its absence it is questionable if the individual has suffered such an attack. Cf. *Bittner v. Supervisors of Saltlick*, 119 Pa. Super. 436, 179 Atl. 902 (1935) (muscular strain bringing on attack).

though either may cause the failure of a heart already handicapped by the presence of coronary arteriosclerosis or coronary thrombosis.<sup>5</sup>

It should not be inferred that coronary thrombosis is necessarily fatal. It may not even be associated with angina pectoris. Many people survive repeated episodes of coronary thrombosis and live to die of some totally unrelated condition. If, as a result of coronary thrombosis, a part of the heart muscle is so deprived of blood supply that it dies, the condition may be referred to as myocardial infarction. An infarct is local death of tissue due to inadequate blood supply, and myocardium is the name of heart muscle. A large myocardial infarct usually results in serious impairment in cardiac function. A small myocardial infarct may be survived with little or no recognizable impairment in health. Often, myocardial infarcts are first recognized as the result of electrocardiographic examination. The normally beating heart discharges a characteristic amount of electricity, which can be measured by placing electrodes on the surface of the body and recording the electrical flow by means of a galvanometer. Certain characteristic alterations in the electrical discharge of the heart may occur if any part of the heart muscle is not functioning properly. The presence of an infarct may be inferred from such electrical disturbances.

There are several facts about coronary heart disease that are important in relation to trauma. One is that although any unusual stress may cause an arteriosclerotic heart to fail, heart failure due to coronary disease usually occurs spontaneously and independently of any known episode of external stress. If the failure of a chronically diseased heart is to be attributed to an episode of stress, it should occur immediately thereafter. If hours or days elapse between the stress designated and the subsequent attack of heart failure, the relationship between the two is probably nothing more than a fortuitous sequential coincidence.<sup>6</sup> Persons with advanced coronary ar-

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<sup>5</sup> In *U.S. Fidelity & Guaranty Co. v. Industrial Comm'n*, 96 Colo. 571, 45 P.2d 895 (1935), a plant guard who was involved in a strenuous argument and ensuing struggle became an "industrial death" when the over-exertion affected his heart, which had already been weakened by coronary arteriosclerosis. In *Merritt v. Dept. of Labor and Industry*, 251 P.2d 158 (Wash. 1952), a 68 year old man was required by the nature of his employment to sit at a bench and handle levers with 40 lb. pressure. He died of pulmonary embolism within twenty minutes after leaving his bench at the end of a work day. The continuous movement of the upper part of his body as he reached about three feet to operate the levers was held to be sufficient work activity to cause the loosening of a thrombus which had previously formed in a chamber of his previously diseased heart.

<sup>6</sup> A period of two months between a body strain resulting from a barrel falling toward a man and the developing of a shortness of breath signalling a heart ailment was held to be too long a time to sustain the probability that the accident aggravated a pre-existing heart difficulty. *Powell v. Am. Employers Ins. Co.*, 14 So.2d 333 (La. Ct. App. 1943). It has been held that two days between the time of collapsing from over-exertion (carrying a 35-40 lb. gasoline can for 240 feet) and death from acute dilation of the heart would not establish a probable causal relationship between over-

teriosclerosis and with serious impairment of cardiac reserve frequently appear to be in good health, frequently go about their work in a normal fashion, frequently perform hard physical labor without complaining and frequently collapse and die unexpectedly while reading the evening paper or while lying comfortably at rest in bed.

Another important form of chronic heart disease is commonly referred to as hypertensive heart disease. This means that for months or years the heart has worked against the handicap of high blood pressure; that as a result of this overwork it has dilated and enlarged and that its capacity to discharge extra work has been diminished. An important difference between the hypertensive heart disease and arteriosclerotic heart disease is that rapid circulatory failure rarely occurs in the former,<sup>7</sup> either spontaneously or as a result of external stress. Heart failure in persons with uncomplicated hypertensive heart disease is usually a slowly progressive deterioration and the course of the disease is rarely significantly affected by physical exertion or emotional disturbance. Frequently hypertensive heart disease is complicated by coronary arteriosclerosis, and in that event the heart has the same propensity to fail suddenly under stress as does the heart of uncomplicated coronary arteriosclerosis.

A third common form of heart disease is valvular heart disease. This means that one or more of the valves that normally make the heart an efficient pump have become deformed as a result of chronic disease. The diseases most commonly responsible for such valvular abnormalities in adults are rheumatic fever and syphilis, neither of which are caused or predisposed to by trauma or stress.

Usually, although not invariably, the person whose cardiac reserve has been significantly impaired by reason of valvular heart disease has been long since recognized to be suffering from heart disease. Ordinarily persons suffering from valvular heart disease have learned to protect themselves against the kinds of stresses that are likely to precipitate heart failure. Their inability or failure to do so, however, may result in the sudden and unexpected failure following stress of a previously competent heart with resulting premature invalidism or death.<sup>8</sup>

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exertion and death. *Speaks v. Hoage*, 78 F.2d 208 (D.C. 1935). But *cf. Commercial Casualty Ins. Co. v. Hoage*, 75 F.2d 677 (D.C. 1935), where a causal relationship was sustained between the over-exertion and the death resulting from the rupture of the aortic valve over a year later.

<sup>7</sup> In *Hoage v. Liberty Mut. Ins. Co.*, 78 F.2d 874 (D.C. 1935), the immediate collapse and death of a 30 year old laborer who used a 20 lb. sledge hammer to break concrete for an hour was held not to have been caused by over-exertion on his hypertrophied heart. The autopsy revealed that the very thin ulcerated areas of the aorta were not perforated as they would have been had the death been due to over-exertion. Expert testimony adduced that death was caused by the stopping of the heart muscle's beating because of flabby muscles and leaking valves.

<sup>8</sup> *Bernstein Furniture Co. v. Kelly*, 115 N.J.L. 500, 180 Atl. 832 (1935) (furniture

It is frequently difficult to assess the true significance of a particular stress that is alleged to have precipitated the incapacitating or fatal failure of a previously diseased heart. Thus it may be claimed that the janitor sustained an incapacitating or fatal attack of coronary insufficiency because of the unusual exertion of sweeping an unusually dirty floor, or of using a worn out broom with a loose handle. It may be claimed that the watchman had his attack of heart failure because of the unusual stress of walking up the stairs which he was compelled to do because the elevator was out of order.<sup>9</sup>

When the particular stress stipulated as the cause of heart failure so closely approximates the stresses of ordinary living, as in the previously cited cases of the janitor and the watchman, the importance of that particular stress to the exclusion of other equally stressful events to which the person was probably subjected, reduces the causal significance of the event from probable to possible. In such circumstances the importance of the stipulated stress may well be questioned because it probably was no more severe than digesting the last meal eaten or sneezing or coughing. Since most people who develop heart failure because of chronic heart disease do so because of a spontaneous change in the status of their disease rather than because of some unusual external stress, the causal importance of external stress cannot be considered a medical probability unless it possesses quantitative or qualitative attributes that set it apart from the stresses of ordinary everyday existence.<sup>10</sup>

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deliveryman, whose heart was diseased with chronic syphilitic aortitis, collapsed and died after carrying furniture four floors on a hot day); *Travelers Ins. Co. v. Johnson*, 84 S.W.2d 354 (Tex. Civ. App. 1935) (strenuous activity in hot weather by a husky, 30 year old man caused an increase in his blood pressure and rupture of the wall of his aorta which had already been weakened by syphilis).

<sup>9</sup> A trolley bus operator, driving through heavy fog at a slow speed while negotiating many turns, collapsed at the wheel and died of coronary thrombosis. The nervous strain, anxiety and excitement alone could not produce death; however, he had prior symptoms of circulatory failure—dizziness, shortness of breath and pain in the heart area—which indicated a pre-existing heart deficiency. When coupled with this deficiency it was contended that nervous strain did cause death by imposing an intolerable burden on the already diseased heart. Although the jury in the trial court found that the strain and excitement caused the death, the Ohio Supreme Court denied compensation because *as a matter of law* there was insufficient evidence to sustain the proximate causal relationship between decedent's death and his employment. Two judges dissented. *McNees v. Cincinnati Street Ry.*, 152 Ohio St. 269, 89 N.E.2d 138 (1949). In *Hoage v. Royall Indem. Co.*, 90 F.2d 387 (D.C. 1937), a 48 year old claimant with a pre-existing condition of arteriosclerosis and a nine months' medical history of headaches, insomnia, heartburn and one heart attack was awarded compensation mainly because of expert testimony that nervous strain he had undergone produced a spasm in a susceptible coronary artery, thereby disturbing the vasomotor system and causing angina pectoris.

<sup>10</sup> In *Raley v. Camden*, 72 S.E.2d 572 (S.C. 1952), a 47 year old man, bending and stooping as he marked lines on a street and lifting posts weighing 35-40 lbs., became dizzy and faint. His heart was burdened with a chronic auricular fibrillation (skipping heart) which was aggravated by his manual labor. This caused his first

*Post-traumatic Shock and Coronary Thrombosis*

A type of circulatory failure called secondary shock may develop immediately after any one of a large variety of injuries in which an excessive amount of blood has been lost or in which extensive destruction of tissue has occurred. Secondary shock may also develop as a result of a surgical operation undertaken to repair an injury, or as a result of a transfusion given to replace the blood lost incident to an injury. Secondary shock is characterized by low blood pressure, cold skin, severe prostration and a diminished blood volume. The condition is ordinarily of short duration, and usually the disturbed state of circulation either improves with rapid return to normal or deteriorates to a fatal termination within hours or at the most a few days after its onset. If the victim of secondary shock has arteriosclerotic heart disease, coronary thrombosis may occur as a result of the circulatory dis-

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Diseased Heart  
(Coronary arteriosclerosis)



Severe injury to any part of body, usually with excessive loss of blood.



Secondary shock with prostration, low blood pressure and slowing of blood flow.



May predispose to thrombosis of diseased coronary artery with death or immediately recognized evidence of increased cardiac disability.

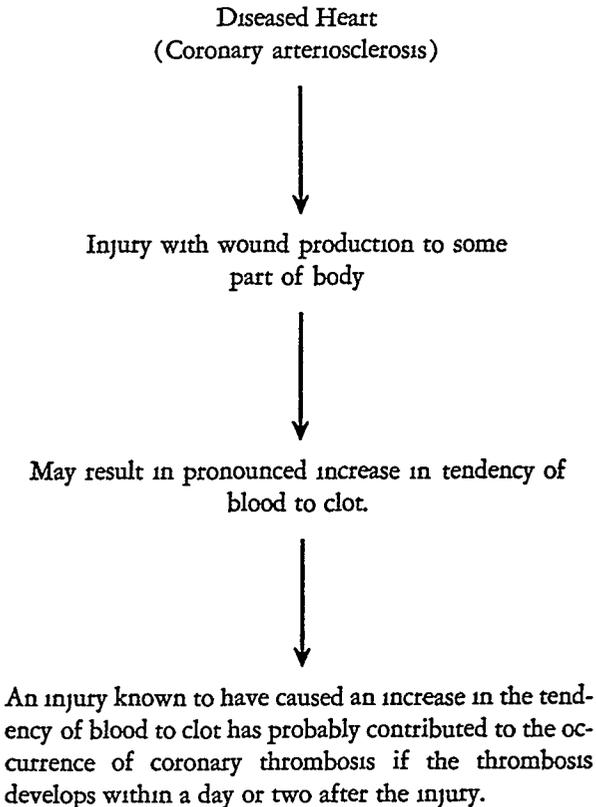
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heart attack and he received compensation as an industrially disabled person. Cf. *Robertson v. Industrial Comm'n*, 109 Utah 25, 163 P.2d 331 (1945).

turbances incident to the shock. In such an event there may be satisfactory recovery from the injury itself and from the shock which followed it, and yet the individual may be left with a more or less permanently damaged heart because of coronary thrombosis which developed during and because of secondary shock.

*Post-traumatic Increase in the Coagulability of the Blood  
and Coronary Thrombosis*

It has been observed that injuries which result in wound production are sometimes followed by a brief period in which the circulating blood has a greater than normal tendency to clot within the vessels. This increased tendency to clot is not dependent upon the occurrence of shock. If the injured person who develops increased coagulability of the blood is suffering from arteriosclerotic heart disease, coronary thrombosis may develop during that brief period after injury in which the coagulability of the blood is



disturbed. If this occurs, the injured person may be left with a more or less permanently damaged heart, even though he makes an uneventful recovery from the direct effects of the injury. To establish the probability that coronary thrombosis occurred from this cause it would be necessary to have laboratory evidence that the coagulability of the blood had been increased.

*Post-traumatic Immobilization as a Cause of Phlebothrombosis<sup>11</sup>  
and Pulmonary Thrombo-embolism<sup>12</sup>*

Any injury which requires the immobilization of an extremity by a

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Normal or Diseased Heart



Injury requiring confinement to bed or splinting of a lower extremity.



Such immobilization may result in spontaneous clotting of blood in veins of legs.



A clot thus formed may break loose after days or weeks and be carried back to the heart and thence to the lungs where it will obstruct the flow of blood through the large pulmonary vessels.



Such pulmonary thrombo-embolism may cause rapid and fatal heart failure.

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Normal or Diseased Heart



Fracture of long bone or wound in  
fatty tissue



Fat droplets from the injured tissue may enter the blood stream in large numbers at the time of or at anytime within two weeks after the injury. Once in the blood stream they are carried back to the heart and thence to the lungs where they may obstruct the small pulmonary vessels.



Such pulmonary thrombo-embolism may cause rapid and fatal heart failure.

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cast or splint, or which requires a period of bed rest, may result in spontaneous clotting of blood either in the veins of the injured region or in the veins of the legs, regardless of where the injury may be located. Continued use of muscles is an important factor in maintaining the flow of blood through them. This is particularly true of the muscles of the legs. When muscles are inactive over a period of time, there is a tendency for blood to clot spontaneously in the veins which drain them. If the thrombus stays in the veins where it was formed, little or no harm is done. However, if such a thrombus loses its attachment to the wall of the vein where it formed, it is likely to be carried by the blood up to the heart, where it tends to obstruct

<sup>21</sup> Phlebothrombosis is the formation of a blood clot or thrombus in a living vein.

<sup>22</sup> Pulmonary thrombo-embolism is the obstruction of blood flow through the lungs by one or more thrombi which formed elsewhere and were carried to the lungs by way of the blood stream.

the flow of blood from the heart to the lungs. Such an obstruction constitutes pulmonary thrombo-embolism and may result in unexpected death from heart failure.<sup>13</sup>

Several facts concerning pulmonary thrombo-embolism should be borne in mind. One is that the post-traumatic development of phlebothrombosis may be asymptomatic, and its presence may be unrecognized either by the patient or by his physician. Another is that days or even weeks may elapse between the occurrence of the injury and the detachment of the thrombus with ensuing death from pulmonary embolism.

Bed rest and immobilization are not prerequisite to the development of post-traumatic phlebothrombosis. Thus, thrombosis may occur in an extremity that has been severely bruised or sprained, even though that extremity was not subsequently immobilized by cast or splint.

Another complication of trauma which may cause fatal failure of a previously normal heart is pulmonary fat embolism. If a long bone is broken or if fatty tissue is severely injured, droplets of fat may gain entrance into the blood. In the course of hours or days an amount of fat may become lodged in the tiny blood vessels of the lung sufficient to plug them.

#### *Post-traumatic Infection and Valvular Heart Disease*

If infection is incurred because of an injury, or if an injury disturbs an already present but previously dormant infection, bacteria may gain access to the blood stream, may lodge and grow on the heart valve and produce an acute type of valvular heart disease. A previously normal heart is so resistant to infection that it is likely to escape damage unless there is overwhelming invasion of the blood stream by bacteria. In such an event the sequence of injury, infection, overwhelming invasion of the blood stream by bacteria and the development of valvular heart disease is usually obvious and not debatable.

The causal sequence may be less obvious in the case of a person whose heart has already been damaged by valvular heart disease. Many people with early or relatively mild forms of valvular heart disease are not seriously handicapped by their disease and in the normal course of events would probably live many years without serious impairment of health. Bacteria capable of producing disease may gain access to the blood stream of such a person as the result of an injury. Even though the number of bacteria reaching the circulating blood by this means may be insufficient to produce recognizable blood stream infection, they may nevertheless lodge and grow

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<sup>13</sup> A person sustaining a compound fracture of his left leg and a partial fracture of his right ankle after a 15-20 foot fall from a scaffolding had to remain in his hospital bed. He died a month later, while still in bed, because of an embolism. His death was held to be causally related to the trauma. *Kelly v. Wills*, 116 App. Div. 758, 102 N.Y. Supp. 223 (1907).

on the previously diseased valves. If this occurs, the heart disease is likely to progress rapidly in severity and unless treated early and effectively may result in premature and permanent cardiac disability or death.

A form of injury particularly likely to result in blood stream infection and rapidly progressive valvular heart disease is the disturbance by trauma of a chronically infected tooth. The extraction of infected teeth from a person suffering from quiescent or chronic valvular heart disease is so likely to result in dangerous infection of the heart valves that the procedure should never be undertaken without concomitant treatment with antibiotic agents in order to destroy whatever bacteria may gain access to the blood stream.

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Normal or Diseased Heart



An injury which introduces disease-producing bacteria into the tissues or an injury which disturbs an already existing locus of active or quiescent infection.



Bacteria may gain access to the blood stream and may localize on the heart valves.



Valvular heart disease may result.

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#### *Heart Disease as a Predisposing Cause of Traumatic Injury*

One of the most important relationships between trauma and heart disease is a reverse sequence of events, namely, injury caused by heart disease rather than heart disease caused or precipitated by injury. A person with any form of chronic heart disease, and particularly with arteriosclerotic heart disease, is likely to collapse spontaneously and unexpectedly from sudden

heart failure. If the attack of heart failure occurs while the individual is standing, he is likely to fall. If he is driving a car he is likely to lose control of it. The resulting injury may or may not be severe. Frequently a post-mortem examination is required in order to determine whether death was due to heart disease or to the injury which was incurred as the result of the heart disease. If, following such an event, the victim is recognized to be suffering from heart disease, it may be difficult to determine whether the injury precipitated a fatal attack or heart failure precipitated the injury. When such a problem arises, it is important to examine with care the evidence bearing on the circumstances in which the injury was sustained. If it appears that the misadventure occurred because of sudden physical collapse, it is fair to conclude that the injury was the result and not the cause of heart failure. If it appears that the misadventure was not preceded or precipitated by circulatory collapse, it may be inferred that the heart failure was precipitated by the injury.

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#### Heart Disease

The presence of heart disease may or may not have been previously recognized.



Disease caused sudden and unexpected collapse in circumstances such that an injury was sustained.



If injury can be recognized to have occurred before death and if its effects can be recognized to be incompatible with life, *death can be said to have been caused by the injury.*



If the heart disease found at autopsy constitutes an adequate explanation of death and if the injury either (1) occurred after death or (2) was of insufficient severity to have caused death independently of the heart disease, *death can be said to have been caused by heart disease.*

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*Fortuitous Coincidence of Trauma and Heart Disease*

The last and most important relationship to be considered is that of fortuitous coincidence. A large proportion of the male population becomes disabled and dies because of arteriosclerotic heart disease. It is in-

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Normal or Diseased Heart



Injury or episode of stress.



Signs or symptoms of heart disease developing days, weeks or months later.



Proof of a causal relationship between the designated episode and ensuing heart condition requires evidence either that the heart *was* immediately and permanently disturbed by the event in question or that certain complications of the injury did in fact occur which in turn damaged or interfered with the function of the heart.



According to the laws of chance, most if not all adults who develop heart disease will have previously sustained some injury or will have previously been subjected to some episode of stress. It should not be assumed that what follows was necessarily caused by that which preceded.

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evitable that the disability or death of many of these persons will be preceded by some remembered incident of injury or stress. The ability of most persons to remember such events is tremendously enhanced by the prospect of double indemnity or workmen's compensation benefits if a cause and effect relationship between trauma and heart disease can be established. The overwhelming majority of such claims for compensation that have been brought to my attention represent nothing more than a fortuitous coincidence in which *post hoc ergo propter hoc* is cited as proof of eligibility for compensation. Unfortunately from the standpoint of justice, judges, lawyers and juries are frequently unduly impressed by the sequence in which events have occurred.