Injury and Heart Disease – Legal Aspects*

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Diagnosticians follow the same pattern in their attempt to make an accurate diagnosis. They must obtain what is described as a good past history and detailed account of the present illness followed by a complete physical examination including all the indicated laboratory procedure. There is little doubt but that the past and personal history of the patient is the most important part of the entire procedure. However, personally I feel that medical students should be taught by the legal profession to differentiate between hearsay and evidence, to question, and to cross-examine. The data accumulated in this manner will lead to a consideration by the physician of one, and occasionally two, probable diagnoses and a number of possibilities. He realizes also that about eighty per cent of the time the probable diagnosis will be correct. However, his experience tells him that some of his most brilliant diagnoses were based on possibilities. He also realizes that disregarding a possibility may cost the patient his life.

Inaccurate terminology used by the physician in the case or in the hospital records, and on the death certificate, which is often written on the spur of the moment without adequate thought, has occasionally jeopardized the legal rights of the patient. Little does he realize that the expert witness must accept these terms in their true meaning and as a fact. This situation could be improved if not corrected by:

1. Considering the causes of death given on a death certificate as an opinion.

2. Placing the questions on the death certificate when an autopsy has not been obtained as, "Probable Cause of Death," "Possible Cause of Death," and a place to state cause unknown.

3. Make it mandatory to perform an autopsy when the cause of death is stated as unknown.

It is impossible to cover this entire subject in one article, therefore penetrating wounds to the heart will not be considered since their cause, pathology, diagnosis, treatment, and legal implications are self-evident.

A few years ago even the possibility of cardiac trauma from nonpenetrating injury to the chest was ignored by the medical profession, and I must confess that it was very much easier to convince the laity and the legal profession of not only the possibility

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but the probability of this result. Contusion of the abdominal organs and contusion and concussion of the brain following nonpenetrating injury to the abdomen and skull were known and accepted, but very little consideration was given to contusion of the heart following external violence. This reluctancy on the part of the medical profession to accept even the possibility of myocardial contusion from nonpenetrating injury to the chest is exemplified by the following statements in the November 4, 1923 edition of "The American Medical Journal." A physician from Lincoln, Nebraska wrote the following query:

> A man, age fifty-seven, was struck over the precordium by a golf ball. He was in a state of shock for fifteen minutes and gradually recovered to the extent that he was able to attend to his business for the following two days. On the night of the third day he had an attack of angina pectoris, with electrocardiographic evidence of a cardiac infarction. Prior to the chest injury there had been no symptoms of coronary disease. Is it likely that there was a traumatic myocarditis? Can you refer me to some literature bearing on this subject? The answer was as follows: "Mild infections may predispose to a coronary thrombosis, as mild infection of the upper respiratory tract, mild cystitis, or some minor ailment, and it is conceivable that the slight accident might have had some bearing on the coronary thrombosis. It is much more likely, however, that it was simply a coincidence. Traumatic myocarditis is one of those indefinite terms that is best not used at all, and there is no literature of scientific value on the subject."

My analysis of this incident differs from that quoted. In spite of the inference that the injury was a slight one, the force with which a golf ball can strike is more than slight because in this case it produced shock. In my opinion it is obvious that this man received a myocardial contusion, but this is not the most important error in the query and answer. The doctor who asked the question unfortunately used the term, "traumatic myocarditis" and "electrocardiographic evidence of cardiac infarction." Myocarditis means an infection of the myocardium or heart muscle caused by trauma, and the answer given was in keeping with the meaning of the term. Also the electrocardiographic evidence would be the same for cardiac infarction or cardiac contusion.

Literature of scientific value had been published on the topic of myocardial contusion prior to this date, but it had not been as universally accepted as it is today. However, in spite of the numerous cases of probable cardiac trauma reported, the stereotyped opinion was that the heart was an organ that almost always escaped inury. It was assumed that the thoracic cage afforded a perfect protection to the heart, but no such immunity to injury was extended to the liver, spleen, kidney, brain or other organs of the

body. It is difficult to understand why the medical profession, with its vast experience in trauma, had developed this belief, especially when the anatomical relation of the heart, buttressed against the thoracic vertebrae posteriorly and lying against the sternum anteriorly, is taken into consideration. The oversight was probably due to the fact that most of the traumatic disturbances of the heart which immediately follow trauma are functional and physiological rather than pathological.

Joseph Urbach[1] in 1922 described the situation when he wrote:

> Those experienced in the examination of affliction of the heart which are the result of trauma know the difficulties with which the expert must cope. These difficulties lie on the one hand in the unreliable and often incomplete factual material which the examiner has at his disposal and on the other hand in the disparity of the opinion of the different authors who have handled the question. While some are accustomed to give wide margins to traumatic reactions upon the heart, others are of a different opinion and say that the influence of trauma upon the heart is not valid in any respect.

Nevertheless, as Barber[2] stated, "Many doctors can quote one or two cases in which they were convinced that trauma produced disease of the heart or that it so aggravated the condition of a heart already diseased that the whole life history was changed." However, the frequency of traffic accidents has stimulated clinical and experimental investigations of this problem and cardiac trauma is gradually being accepted as a definite entity. Even though the heart is well protected by the bony thoracic cage, there is abundant evidence that cardiac injury does occur by either direct or indirect violence many times without external evidence of injury and often from what appears to be only slight trauma.

The consideration of this subject is confused by the relationship of strain to trauma. Also, injury may be penetrating to the chest and result in a nonpenetrating injury to the heart by a fragment of bone. A broad view of the subject must take into consideration the effect of electrical shock, extreme pressures due to explosion, foreign bodies, oxygen deficiencies due to bad air, carbon monoxide intoxication, poisonous and allergic air-borne substances, radiation and toxic substances, as well as shock, anger, and emotional excitement upon certain types of heart disease.

Warburg[3] called attention to possibly the first case of myocardial contusion. It was described by Oluff Broch of Copenhagen in 1676 as follows:

> An eight year old boy, Christianus Sudus, fell between a table and a footstool bruising his chest very badly. During the next few months he grew pale and complained of pain in his abdomen, his decline was rapid and death occurred

> in approximately four months. At autopsy the right auricle was almost six times the size of the left but pale and full of clots, was yellowish and nearly decayed.

He believed that the injury to the right auricle was the result of the blow that had been received. The question as to whether these findings are of traumatic origin is, of course, open to doubt, first because of the length of time between the injury and death and, secondly because of the possibility of the lesions being the result of some infectious process.

The more generally accepted first reported case of cardiac trauma following nonpenetrating chest injury is that of the Dutch surgeon, Stephen Blanchard,[4] in 1688. A peasant forty-five years of age had a load of hay fall on him and following this complained of a prickling sensation and pain in his chest, with a feeling of pressure and tension. At autopsy the pericardium was found full of pus and the auricles were partly decayed and very soft, this extended also into the left ventricle. In this case the time element was more consistent with cause and effect and although infection was evidently present in the pericardium, still it is generally accepted that a purulent pericarditis frequently follows cardiac trauma. The first unquestionable case was that of Mark Akenside[5] who reported that while he was court physician in 1764, a boy fourteen years of age received a blow from his chief in which a plate penetrated between two ribs. He had a cough, hemoptysis, and six months and two days later he died. The post mortem revealed a necrosis the size of a half crown on the left side of the heart involving nearly the whole wall with a totally adherent pericardium.

The possibility of a shot entering into the thorax but not penetrating the pericardium of the heart and causing a myocardial contusion must also be considered. Antonius Borellus,[6] a Norwegian surgeon, reported such a case in 1682 which caused a lesion of the cardiac muscle and hemopericardium. William Harvey[7] in his works translated by Sydenham in 1847, is believed probably to be the first to describe a rupture of the heart following a non-penetrating injury to the chest. Warburg[3] called attention to the fact that for two hundred years, from 1676 until 1875, there were only twenty-seven cases reported in the literature, some of which, were doubtful. Then in 1868 Fischer[8] published his impressive work and collected seventy-six cases of lesions of the heart due to non-penetrating injury; sixty-nine of these cases dealt with traumatic cardiac rupture, and only seven cases with myocardial contusion. In 1900 the number of cases reported greatly increased, which indicated that a few authors were now paying special attention to this condition but still the condition was considered a rare lesion by both clinicians and pathologists.

The mode of injury has varied over the years with the various mechanical advancements. For example, Fischer[8] in 1868 reported traumatic heart disease was caused most frequently by being run over or crushed by wagon wheels; crushed by machinery; falling from a height; struck by flying and falling objects; gored by a bull; kicked in the chest by humans and animals; trampling; and being held or pinned against a wall. In modern times this type of injury is most frequently caused by automobile accidents in which the chest strikes either the steering wheel or the back of a seat, but there are still a great number of these injuries produced by being crushed under vehicles and falling debris. Leinoff[9] in 1940 undertook to determine the frequency of traumatic heart disease following fatal automobile accidents by examining the protocols of fifty non-selected consecutive cases through the courtesy of Dr. Gonzales, chief medical examiner of New York City. Eight cases, or sixteen per cent, showed definite macroscopic findings of cardiac damage.

It is obvious that all this clinical and historical background required experimental evidence pertaining to traumatic heart disease. In 1908 Kulbs[10] performed experiments on a series of twenty-three dogs which were struck from one to three times in the left breast with a club and after hours or days the animals died and the thoracic organs were damaged. Conspicuous hemorrhage of the skin or thoracic wall was very seldom found and the most regular finding was hemorrhage into the heart valve. In all of the animals there was injury to the pericardium and subpericardial hemorrhages. Microscopic examination showed fresh hemorhage in the heart muscle which invaded the inner spaces of the muscle fibers and often crowded them aside. The muscle fibers lying below the spots of hemorrhage were swollen with granular or lumpy protoplasm and had lost their striation, which indicated beginning heart muscle destruction.

Bright and Beck[11] in a series of twenty-five experiments opened the thorax and exposed the hearts of dogs to direct injury applied to the heart muscle. A number of blows by a metal instrument were struck to the right and left ventricular wall. These blows caused the myocardium to become swollen from hemorrhage. In twenty of the twenty-five experiments the contusions were well tolerated, however, hemorrhagic effusions into the pericardial cavity occurred frequently. The healing process was usually adhesions developing between the heart and pericardium over the area of contusion. Our experimental production of cardiac trauma was with nineteen anesthetized[12] dogs that were struck blows to the external chest. The blows were of varying intensity ranging from that produced by a hard rubber mallet to a ten pound sledge hammer.

The animals were grouped into series according to the degree of external force applied. The autopsy findings in these cases revealed that the most frequent location of injury was to the pericardium and the repair was by thickening and fibrosis. The production of tears and ruptures with hemopericardium, subendocardial hemorrhage, tears with various degrees of myocardial contusion and hemorrhage, resulted chiefly from the severe blows. However, these findings were frequently present when only mild external violence had been delivered. The subpericardial hemorrhage varied from small petechial-like spots to large areas of bruising and discoloration with myocardial hemorrhage and contusion extending deep into the muscle wall from points on the pericardium. The frequency of myocardial lesions such as hemorrhage and contusion corresponded with the findings of Kulbs[10], who used the same type of blunt force, however there was some difference in the location of the lesions since we produced more lesions in the auricles.

It was interesting to note that two experimental animals died immediately from fatal arrhythmias and no cardiac damage could be found at autopsy. Sudden death from external chest injury without demonstrable cardiac pathology has been observed in humans by Nelaton[13] and Reidinger[14] and they were of the opinion that it was due to an extreme lowering of the blood pressure produced by irritation of the vagus nerve. This is not very likely because the experiments showed some fatal arrhythmia or cardiac standstill.

The mode and mechanism of injury is of considerable legal and medical interest. Cardiac injury frequently results from being crushed between objects and since one object acts as an abutment against which the body is compressed, the impact need not always be severe. When both objects are moving and strike the chest simultaneously the compression is from both front and back, or from both sides. However, blows directed against the left side of the chest produce the most injuries. Squeezing accidents such as being crushed between bumpers, doors, elevator shafts, and machinery, or by falling objects, etc., are similar. In fact, the only difference is in the relative movement of one or more of these objects. The injuries caused by machinery, such as being caught in a wheel or belt, result from repeated assaults until the victim is freed. After excluding falls from high places, Urbach[1] listed the following sequence of frequency: blows from the fist or kicks, blows by a heavy object or wagon tongue, and butting by animals.

Aside from the transmission of force to the heart by a blow to the chest, it may be injured by being compressed from various angles. The most frequent form is from front to back, perpendicular to the axis of the body. Experiments have shown that in the youth-

ful thorax the sternum can be depressed to the extent that it touches the spine. It is occasionally possible for pressure in a transverse or oblique direction to injure the heart. Leupold[15] believes that the lungs act as an elastic air cushion absorbing at least a portion of the blow. In severe trauma this protection does not suffice because the heart has been completely torn from the great vessels in this manner. These lesions are most frequently caused by squeezing the body from the feet toward the thorax. The severe compression produces cardiac rupture and the tearing lesions in the auricles is a result of a violent displacement of the heart. This latter mechanism occurs in falling from high places and in one such accident the heart was found entirely free in the left pleural cavity.

When a fracture of the bony cage occurs fragments of bone may, without penetration, cause injury to the pericardium and heart. However, if a fragment of a rib penetrates the pericardium the injury should be classified as a penetrating wound. It is also possible to cause myocardial contusion by a foreign body which penetrates the chest without penetration of the heart or pericardium. In this type of injury the projectile, while it has sufficent force to penetrate the thoracic wall, does not penetrate the pericardium but is sufficient to produce myocardial contusion. Damage to the heart and disturbances of function can result from a displacement due to the continuous pressure of crushed-in and deformed ribs or sternum. Also when hemorrhage occurs in the posterior mediastinal space, adhesions later may not only compress the heart but also constrict the great vessels.

There are certain facts that help us to understand the mechanism of nonpenetrating cardiac injury. The heart, attached by the great vessels, hangs free in the chest which permits it to be violently thrown against the thoracic wall or displaced and torn from these atachments. Although a hollow viscus, the heart becomes a solid mass during the pressure of the systole upon the contained blood. Besides the demonstrated possibility of compressing the sternum against the spine, contusion of the heart is facilitated by the fixation of the thorax during inspiration which usually occurs immediately before an accident.

A brief review of the pathology will give a good insight into the symptomatology, physical signs, electrocardiographic changes, treatment and prognosis. Myocardial contusions are characterized by a large number of blood cells in the interspaces between the muscle fibers which often appear to crowd them aside. The hemorrhages are usually subendocardial or subpericardial, but what appears to be a hemorrhage under the pericardium or endocardium is often the apex of a large triangular area of myocardial contusion.

There is fragmentation and tearing of the muscle fibers which may involve the papillary muscles. Of great interest and importance are the pathologic changes in the neighborhood of the old hemorrhages. A wall of polymorphonuclear leukocytes, intermingled with granules, surrounds the area and the muscle fibers have partly lost their transverse striation and are swollen with a lumpy or granular protoplasm. These lesions are usually produced by severe injury to the external chest wall, with or without external evidence. The areas of hemorrhage and contusion are usually situated in the muscle and serial sections are necessary to reveal the pathology. It has also been observed that gross lesions which are not obvious in the fresh specimen will show up well after fixing the specimen.

We have reported previously a series of patients who had lived for various periods of time following myocardial contusion. In those who had lived more than six months but not more than eighteen months myocardial fibrosis had occurred in ninety per cent. There was, however, still evidence of hemorrhage in one per cent. In those who died within a short period of time there was myocardial hemorrhage, fragmentation and edema in each instance. After a few weeks the hemorrhage and associated displacement of the muscle fibers began to be replaced by fibrous tissue. In the older cases there appeared muscle fiber destruction from anemic necrosis with thinning of the myocardial wall in the area involved.[16]

There is a very close similarity between the lesion of myocardial contusion and infarction as the result of coronary artery occlusion. In a recent myocardial lesion the only evidence which would invariably serve to identify myocardial infarction, when there is a question of traumatic etiology, is in finding of a recent occlusion of a coronary artery. The older lesions are also extremely difficult to distinguish since scars of myocardial contusion and infarction are frequently identical.

Endocardial lesions vary from a small subendocardial petechial spot to large areas of bruising and discoloration. The larger hemorrhagic areas have the appearance of hematomas, and occasionally the underlying myocardium is necrotic. These lesions were found in nineteen per cent of all the severe and in fifty per cent of the moderate and moderately severe cases. Numerous small tears were found, some also involving the myocardium, and the healing was by scar tissue.

The anatomic structure and relationship of the pericardium predisposes it to nonpenetrating injuries. The observed traumatic injuries were the same, whether produced experimentally in animals or found in humans at necropsy. In those receiving severe to moderate chest trauma, subpericardial hemorrhage occurred

in ninety-eight per cent. This was associated with pericardial hemorrhage and quite frequently small pericardial tears.

The subpericardial hemorrhages are usually anterior and near the large arteries, and they also vary in size from small petechiae to large bruised areas. The lesions revealed at necropsy in the patients who lived for at least six months showed areas of subpericardial fibrosis. These uncomplicated lesions of the visceral pericardium have a tendency to heal in a very short period of time. There is, however, always the possibility that a hemopericardium may result from oozing or hemorrhage of a bruised area or tear. This discussion is limited chiefly, however, to myocardial contusion and therefore hemorrhages, large and small tears, herniation through the pericardial sac, and adhesive pericarditis will not be considered.

The pathologic lesions of traumatic heart disease explain why the symptoms and physical signs are similar to those produced by other causes. A correct appraisal must be made of the cardiac condition of the patient before, at the time of, and immediately after the trauma. Nevertheless, at the present time more cases of traumatic heart disease are overlooked than are diagnosed incorrectly. Traumatic pericarditis produces various types of murmurs. These may be loud or soft, and occasionally they are to-and-fro. When the pericardium is torn, the heart sounds frequently become very peculiar, such as a clicking or a loud or soft murmur with a sharp interruption or a noise similar to blowing on an empty bottle. These murmurs frequently disappear with a few cardiac contractions or on the change of body position, to reoccur later. A friction rub may replace or be associated with the murmur at any time and the sudden disappearance of the murmur or sound is a signal to be on the lookout for pericardial effusion or hemorrhage with beginning tamponade.

The pain when due only to a pericardial lesion, is typical of that seen in pericarditis and may become more severe with inspiration. There is also dyspnea and occasionally orthopnea. The pain however persists for a much longer time than that which is caused by the local pericarditis of a myocardial infarction.

The most constant symptom of myocardial contusion is immediate excruciating pain located in the precordial or substernal area and radiating usually to the left shoulder and down the arm, although it may be referred to both shoulders and up the sides of the neck. It is identical with that seen in coronary insufficiency, and in myocardial infarction. After a period of a few weeks the pain may be brought on by exertion as in angina pectoris. Occasionally, because of other severe injuries producing either unconsciousness or severe pain, this cardiac pain is not the chief complaint. There

have been cases in which the pain started after a short latent period, but this is not the rule. It was observed in the necropsy cases that those persons who failed to have pain as a symptom had only small areas of myocardial contusion or traumatic pericarditis. Close questioning of the patient will frequently differentiate the cardiac pain from that caused by fracture and injury to the bony cage.

Approximately seventy-five percent of all cases of traumatic heart disease have symptoms immediately, but there may be a delay up to one month. The most common symptoms to develop following a latent period are signs of myocardial weakness, beginning congestive heart failure, and nocturnal paroxysmal dyspnea. Tachycardia and palpitation are common in practically all cases of myocardial contusion. Arrhythmia is frequently seen, and these irregularities present their own characteristic signs and symptoms.

As you can readily see, the differential diagnosis of myocardial contusion and coronary artery occlusion with myocardial infarction is extremely difficult and occasionally impossible. This, however, is expected when the underlying pathologic lesions are considered. Caution should always be used in making the diagnosis of myocardial contusion or traumatic heart disease, and the possibility should always be considered that there may have been a coincidental coronary artery occlusion with myocardial infarction following an accident. On the other hand, there have been entirely too many cases of chest injury with these typical signs and symptoms that have been diagnosed as coronary artery occlusion. It must also be remembered that myocardial contusion can occur in a heart that is already damaged by some other type of disease.

The type of cardiac arrhythmia is influenced by the location of the myocardial injury. Premature contractions are by far the most frequent type. Auricular fibrillation, flutter, sino-auricular block, nodal rhythm, auriculo-ventricular block, and idioventricular rhythm have all been observed. These disturbances of rhythm have their own characteristic signs, symptoms and electrocardiographic changes. Auricular paroxysmal tachycardia appears less frequently, but ventricular paroxysmal tachycardia, fibrillation and idioventricular rhythm are the frequent causes of death in the fatal cases. Almost every type of arrhythmia has been produced experimentally in dogs. The electrocardiographic changes in the RST components and T waves due to myocardial contusion and pericardial trauma are similar to those seen in myocardial infarction and pericarditis. These changes may be of the characteristic T-I or T-III type with the corresponding changes in the unipolar chest and arm leads, but frequently are not reciprocal in leads I and III. There are changes indicative of myocardial abnormality and

the unipolar leads are useful in locating the site of the lesion and identifying traumatic pericarditis. The electrocardiographic changes in both myocardial contusion and traumatic pericarditis return to normal in a much shorter time than those produced by disease. The alteration of the electrocardiogram frequently does not occur until twenty-four to forty-eight hours after the injury, and therefore a series of records should be taken. Contusion of the lung is quite frequently associated with traumatic heart disease and one must therefore be on the lookout for the development of pulmonary edema. It is most frequently the result of the pulmonary injury rather than heart failure.

Traumatic valvular rupture occurs in both normal and diseased valves; but great care should be taken to differentiate the latter from a spontaneous rupture. Tears of cusps which do not heal result in fragmentation of the free edge and rupture of the chordae tendineae. The healing occurs with the formation of scar tissue, especially when the tear is along the base or commissure of the leaflet. The healing of hemorrhages and hematomas results in contraction and incompetency of a valve after a latent period and when healing occurs in the fragmented parts of the valve cusps it tends to smooth off the rough edges. The fragments may grow together or become attached to the wall of the heart but complete healing with obliteration of the opening is not accomplished. The thickened cusps produce some stenosis with insufficiency. Reubold[17] believed that traumatic stenosis is produced by the extravasation of blood which organizes and mechanically distorts the cusps. Since it is known that insufficiency of a valve may occur from dilatation of the valvular ring without abnormality of the cusps, it is reasonable to assume that valvular insufficiency can occur from the dilatation and stretching of the contused myocardium in the immediate vicinity of the valve.

Statistical experimental studies of Urbach,[1] Barie,[18] and Howard[19] showed that most valve ruptures occur on the left side of the heart and that the aortic valve is most frequently involved. Men are naturally more predisposed because their activities and occupations are more dangerous. Falling from heights or being buried under an avalanche of debris most frequently cause rupture of the aortic valve by greatly increasing the diastolic pressure of the aortic column of blood against the closed cusps. This concept is supported by the finding that usually the tears are only partial, because as soon as the rupture occurs the pressure is relieved. The passing of a cardiac catheter through a valve against the blood flow will injure or rupture the cusps, but passage with the blood stream seems to have no effect on the valve or endocardium.

The anatomy of the mitral valve, with its large, broad, thick

cusps supported by the chordae tendineae, is a prominent factor in protection from rupture. The chordae tendineae and papillary muscles rather than the cusps are more frequently ruptured. These lesions are explained by the overbending of the cusps during compression of the heart. This probably also explains the jagged tearing when the chordae tendineae hold fast. Small incomplete tears and hemorrhage most frequently are on the auriclar side. These lesions are also found in the tricuspid and pulmonary valves, but rupture is rare and when it does occur it is invariably associated with other severe cardiac injuries.

There have been numerous cases reported of infectious endocarditis following external chest injuries but in the majority of the cases they developed gradually and after a considerable lapse of time. Rosenbach,[20] Orth,[21] Whyscokowitch,[22] and Weichselbaum[23] were the first to establish the experimental support of traumatic valvular endocarditis by injecting bacteria into the circulation following injury to a valve. Litten[24] and others were of the opinion that there is a thrombus formation on the site of the small endocardial pathology with the accumulation of vegetations. There appears to be sufficient evidence that a traumatized valve becomes vulnerable to infection, especially one of rheumatic etiology, and thus leads to scarring, thickening, contraction, and adhesion of the cusps with the production of stenosis. It is probable that injury uncomplicated by infection will not, in the healing process, create sufficient thickening to produce stenosis. It is easy enough to accept the probability of the entrance of bacteria into the heart following penetrating wounds of the chest but quite difficult when the injury is a nonpenetrating wound. There is considerable argument as to just how the bacteria enter the heart when other organs such as the spleen, when contused or ruptured, rarely develop infection. It would be a rare coincidence in which a trauma to the heart occurred at exactly the same time that there were bacteria in the blood stream. Nevertheless, subacute bacterial endocarditis can be superimposed on a traumatic lesion as readily as on one that is rheumatic or congential.

Valvular rupture is not associated with sudden death but invariably results in permanent damage, however, it is not necessarily fatal. It is a serious type of injury and the duration of life is shorter in rupture of the aortic than of the mitral valve. Traumatic insufficiency of a valve produces a sudden strain on the chamber behind the valve with not sufficient time for compensatory hypertrophy of the myocardium and heart failure may follow rapidly. The collection of fibrin or small blood clots on a torn valve leaflet may act as a focus for emboli which may cause sudden death. The prognosis is grave if the injury is complicated by an inflammation

of the endocardium, myocardium, or pericardium. Minor injuries to the valve cusps, such as hemorrhage, may later, by the healing process, produce incompetent valves but the majority recover with slight, if any damage.

The prognosis in myocardial contusion, fragmentation, and hemorrhage depends on the number of lesions and their extent. These contusions have a definite tendency to heal, especially when they involve only one layer of the heart wall. The possibility of cardiac rupture is greater with the more extensive lesions. Sudden death from cardiac rupture does not always occur and there are numerous cases reported and observed that have lived for two to three days. The prognosis is more serious when the contusion involves the intrinsic nervous mechanism of the heart and produces permanent or immediately fatal arrhythmias. Isolated internal lacerations may be survived, such as rupture of the septum, especially when the injury does not affect the conducting mechanism of the heart; but the work capacity of the heart is definitely diminished. The ruptures of the heart wall which are most unfavorable are those in the auricles because of their thinness which prevents closure with muscle contraction. A high percentage of the cases that do not die suddenly have a rupture of the strong muscles of the left ventricles.

The smaller pericardial and subpericardial lesions heal uneventfully, but extensive pericardial adhesions or obliteration of the pericardial sac may produce heart failure. Frequently adhesions result in partial or complete cardiac fixation and permanent disability. Cardiac tamponade frequently causes sudden death or rapid failure. The heart can also be embarrassed by clot formation. When the wound is small, or when the pericardial sac has been obliterated by adhesions, or when there is also a rupture of the pericardial sac allowing the blood to escape into the pleural cavity and defeating tamponade, death is delayed for some time. Slight pericardial injuries heal more readily than those of the endocardium, probably because no blood enters the small tears causing dissection.

As a general rule, in cases of uncomplicated, mild, and moderate cardiac injury, the prognosis for partial, or even complete recovery, is good. Uncomplicated hemorrhages have the best prognosis, certainly better than contusion or rupture. Likewise injuries that are confined to one cardiac chamber have a more favorable prognosis than those involving several chambers, especially when these are nonperforating. Anderson[25] summarized the possibilities following myocardial contusion as complete recovery, reduced cardiac capacity, pain on effort, congestive failure, or rupture.

The possibility of injury producing death or aggravating an

already present heart disease is revealed in the case, reported by Flerow,[26] of an individual with fatty degeneration of the myocardium and mitral valve leaflet thickening who, following a blow to the chest, died almost immediately from a rupture of the right auricle. Thorel[27] was of the opinion that heart rupture occurred infrequently where there are but minimal skin injuries or blood effusions, and in such cases the heart was usually diseased before the injury. It is easily understood that in a case of coronary artery occlusion with myocardial infarction, myocardial necrosis or marked thinning, that even a slight blow to the chest under proper conditions could cause a rupture which might not otherwise have occurred. Recent clinical and experimental tests have shown that a normal heart can be injured and even ruptured without external signs of violence.

Stern[28] believed that a heart disorder can be aggravated in three different manners due to trauma, (1) by the psychic excitement (2) by strong mechanical shock to the body and (3) by strong movements of the muscles, such as the effects of sudden effort that will lead to raising the pressure on the inside of the thorax and also lead to a compression of the heart and the intrathoracic vessels, or the effects of the muscle activity which increases the work of the heart. Thoracic deformity resulting from injury also has an occasional influence upon the heart function. This, of course, would be possible in marked depressed deformity of the sternum or ribs so as to displace the heart from its normal position.

Kahn and Kahn[29] expressed the opinion that direct violence to the chest may predispose the heart to the effects of heart strain. The resulting condition must be attributed to the direct violence rather than to the strain for the reason that the aggravation of symptoms by effort usually develops within a short time after the violence. To illustrate this they cite the case reported by Schuster,[30] of a healthy laborer forty-three years old who received a blow to the chest by the harness bar of a wagon and was thrown to the ground. He immediately recovered except for some pain which persisted for two weeks. At the time, following a hard strain of lifting he developed a sudden and severe epigastric pain radiating to the chest with a choking sensation. He was pale, cold, cyanotic, and the heart action and tone was weak. He died suddenly after a few hours. The autopsy revealed hemorrhage into the anterior and posterior mediastinum, pericardium, and the aortic wall was torn horizontally just below the semilunar ring.

In discussion of this case Kahn and Kahn were amazed to find that with such extensive injury to the heart muscle and aorta the patient was able to do even strenuous work for a time and felt

only moderate pain; that rise in blood pressure entailed by the additional lifting two weeks after the original accident apparently produced the severe symptoms and total disability that terminated in his death; and that in such cases the aggravation produced by strain was secondary. The essential liability falls upon the direct injury by violence.

Clark[31] studied the effects of accidents on persons with heart disease by reviewing forty-five cases which he collected. He concluded that:

> As a rule, the diseased heart is unaffected by accident, but in a certain number of cases the exception occurs, especially in old workers, who have cardiac disease, or in the case of those who are on the edge of heart failure. In these cases, following an accident the heart may show signs of failure which persists and progresses. Also, there appears to be no special type of accident, which is invariably followed by heart weakness and failure.

Heart strain is not trauma in the usual and common meaning of the word, however, the result of this physiological condition is internal injury with disability of varying duration and degree. Labor should be critically examined and separated into two categories; that of sudden, or continuous effort. Very often the distinction is almost impossible.[32, 33] Strain may be that of a conditioned individual in an athletic event, or the intense unexpected effort of a man that is not trained. In the latter type the unexpected effort usually occurs when the glottis is closed and the chest is fixed and is most likely to damage the heart. Wilson[34] believed that he recognized dilatation of the heart after effort with a quick return to normal, and Wilkinson[35] suggested that the dilatation was only apparent, and that neither dilatation nor the strained heart existed. This is supported by Smith[36] who believed that all cases diagnosed as acute dilatation from strain were due to a cardio-neurosis. Barber[2] suggests that it is reasonably possible for the untrained average man to have acute dilatation while undergoing exceptional strain with the glottis closed. However, he also mentions the possibility of an unrecognized lesion of the heart being present and therefore the organ was not truly normal, which is probably a true appraisal.

An orthodiagram or teleoroentgenogram taken immediately after the questionable overstrain, which when compared to one made previously or later, showed increased heart size would be probably the only medical evidence worthy of consideration in favor of acute cardiac dilatation of a normal heart. A diseased heart would be more easily dilated by over-exertion, especially when there is a narrowing of vessels or valve orifices interfering with the escape of blood, or degeneration of the muscle walls.

During exertion there is a deep inspiration, closing of the epiglottis with tense contraction of the muscles of the chest wall which places the heart under increased pressure and thus is exposed to greater danger from injury by a force applied to the thorax. It should be remembered, however, that many believe that even with the most severe exertion it is impossible to cause a normal heart to fail or dilate. I share this opinion.

Sudden physical exertion will produce auricular fibrillation in a heart already diseased and even if this irregularity would be eventually expected, as in mitral stenosis, the strain if it caused its occurrence only a short time sooner than it otherwise would have occurred, must be considered as an aggravating factor. This is important, because functionally the heart in auricular fibrillation does not have the efficiency of one in normal rhythm.

Heidenhain[37] expressed the opinion that endocardial ruptures that occur either on the right or left side of the heart were due to the inner force from over exertion. However, this observation has not been confirmed in fact by experimental and autopsy material. Clowe, Kellert and Gorham[38] analyzed fifty-five cases of rupture of the aorta and were of the opinion that exertion, if ever, produced this condition but found that the attacks developed suddenly while the patient was in bed, walking, standing, or engaged in his usual activity.

A great number of the cases reported of rupture of the aortic valve cusps, or as a matter of fact any valve cusps, due to muscular strain, are in reality not ruptures of normal valves but due to endocarditis, spontaneous ruptures or ulcerations. This mistake is very easily made, in fact, the great clinician Gerhardt reported such a case of supposed rupture of the aortic valves and later Sinnhuber[39] stated that it was shown at autopsy to be a recurring endocarditis and not rupture. Howard[10] collected sixty cases of aortic valve rupture due to strain, but only thirty were proven by autopsy.

> Pepper[40] believed that, immediately before great effort, a deep inspiration is taken which aids in filling the cavities of the heart to their utmost, and then in order to afford fixed points for the contraction of the muscles of the arms, shoulders and back, the chest is held rigidly fixed. The violent contractions of the neck which follow, compress the carotid arteries, while the muscles connected with the arms impede the free flow of blood through the subclavians and their branches. Thus, while the tension within the chest is greatly increased and the heart is stimulated to violent contraction, there is also an enormous elevation of arterial tension. The strain which results must extend itself directly upon the walls of the left ventricle, which must over-exert to press for-

ward the blood, and indirectly upon the aortic valves, which are compelled to bear the shock of a recoil of the blood stream.

Injury to the great vessels is frequently due to the violent displacement of the heart in the chest. Laceration of the inferior vena cava is most frequently accompanied by fractured ribs, which very likely caused the damage. The aortic attachment of the ligamentum arteriosum acts as a fixed point and the arch as a whole is rather fixed by the great arterial trunks. The hinge-like action of this attachment is responsible for the localized point of injury. It has been found that aviators who had fallen from various heights had transverse aortic lesions at or near this attachment. The anterior wall of the aorta is not as thick as the posterior and the latter may be crushed against the vertebral column thus sustaining the blunt of the impact.

It is interesting that in traumatic ruptures, and in those due to other causes, the site of predilection is in the ascending portion of the arch and at the duct of Botalli because these areas are comparatively fixed, and also the aorta may be congenitally weak in these regions. Rupture of the aorta from any cause frequently results in the formation of a dissecting aneurysm. The mechanism of rupture of the aorta as the result of falls from a height is explained by the sudden increase of blood pressure within it, due to the impact and the doubling up and distortion of the body as it strikes the ground. It is this explosive force of the blood contained in the artery that rips through the wall. This, of course, may occur in a normal as well as a diseased aorta.

The wall of the aorta is frequently injured in accidents resulting from direct violence to the chest, such as a blow. A sudden and violent compression of the systemic arteries which distends the aorta to a marked degree at the time of cardiac systole can cause the intima to rupture and the blood to extravasate gradually distending the elastic coats, paralyzing the nerves, and occluding the vasa vasorium with resulting sac or fusiform aneurysmal formation. Schnabel[41] reported the case of a worker who received a severe blow to the chest from a wagon tongue. For two weeks he appeared to be recovering when, after the exertion of carrying a heavy burden, he collapsed and died the next day. There were subpericardial, endocardial, and myocardial hemorrhages of the heart and a rupture of the ascending aorta with a dissecting aneurysm. Stern[28] in discussing this case suggested that the blow may have produced the hemorrhages, while later the rupture of the aorta with further hemorrhage occurred as a result of the severe exertion. It is evident that when the aorta is dilated by an aneurysm and predisposed to rupture no great increase in blood

pressure is required to produce rupture, and it is evident also that after a dissecting aneurysm has started it can continue in a normal media and under normal blood presure.

McFadzean[42] reported a most unique case of four saccular aneurysms of the abdominal aorta in an old man who had been a trapeze and hard bar performer. There was no history of syphilis or evidence of it at necropsy but there were four aneurysms occupying the whole extent of the abdominal aorta and the greater part of the external and common iliac arteries. All of the large arteries of the abdomen from the origin of the ciliac axis to the termination of the external iliac arteries on either side were degenerated, calcified, irregularly dilated and aneurysmal. Almost every day for twenty years he had spent some time swinging and turning on a hard trapeze.

Practically all traumatic aneurysms of the aorta are dissecting and the prognosis must be considered in the light of this condition. Death may occur immediately from complete rupture, however, the small number that survive the period of injury have a fair chance of recovery. The aneurysm may rupture back into the aorta, but most frequently the dissecting sac fills and is closed by clotted blood, creating a false channel which results in the so-called healed aneurysm. If the injury is to the ascending portion an associated cardiac contusion is more likely and there is also a greater posibility of rupture into the pericardial sac with cardiac tamponade.

Traumatic aneurysm of the pulmonary artery is occasionally seen, but rupture is quite rare, and there are no symptoms peculiar to this condition. Frequently there is an increase in the size of the right ventricle with a to-and-fro murmur and a thrill felt over the pulmonic area. These signs, together with the observation of a pulsating dilatation of the pulmonary artery, which can be differentiated from the aorta, are of great assistance in making the diagnosis. The following case illustrates a method by which this lesion is produced. A white coal miner, age 56 years, was struck by a fall of slate with such a force against his back and lower thorax that his body was flexed anteriorly until his head was driven between his legs. He developed a marked thrill over the pulmonic area with a to-and-fro murmur. A Roentgen ray examination a few weeks later revealed a pulsating aneurysm of the pulmonary artery and the electrocardiogram showed a 2 to 1 heart block.

In my opinion, from a medicolegal standpoint, a history stating the patient was previously healthy, was able to do hard work, and served in the Army, etc., is not sufficient because it is common knowledge that severe heart disease with marked damage may

remain for a long time in a symptom-free latent state. However, in regards to myocardial injuries it is generally sufficient to trace a definite relationship between an injury and the following heart affliction, and it matters not whether the heart was normal or diseased, but the important fact to determine is the degree of disability and reduction in productive capacity. It is, of course, quite beneficial in establishing a diagnosis of traumatic heart disease if it can be shown that within two or at least three years previous to the injury the individual had been examined by a capable and experienced physician. This should be at least a physical examination and much better if it included an electrocardiogram and the results showed a normal heart.

Beck[43] is of the opinion that since the differentiation between myocardial infarction and myocardial contusion is so difficult, frequently the only essential differential point is that of the trauma and because it is frequently irrelevant in the history of medical lesions the physician does not hesitate to consider the injury as only coincidental in the production of the cardiac lesion. Following an injury, the possibility or probability of traumatic heart disease should be kept in mind and every attempt must be made to ascertain the condition of the heart prior to the injury without placing too much reliance on the testimony that the patient was apparently in good health and able to work previously.

Traumatic injury to the normal heart usually results in recovery even when there are gross electrocardiographic changes and arrhythmias. The possibility of sudden death due to cardiac rupture following a silent myocardial infarction or other causes resulting in a fatal accident and establishing circumstances which might appear to show death as the result of trauma must be considered, but the truth is invariably revealed by autopsy. The case reported by LeRoy and Snider[50] illustrates this point. "A man aged fifty-six ran into a tree while driving home from work. He was dead when a passerby reached the wreck. Three days previously he had consulted a physician because of a 'cold in the chest' and had received some sort of medication. The necropsy disclosed a moderate hemopericardium, the origin of which was a large rupture of an infarct of the posterior surface of the left ventricle and a complete occlusion of the more distal portion of the circumflex branch of the left coronary artery by a thrombus was observed."

Myocardial contusion has a definite tendency to return to normal, so permanent damage should not be considered unless twelve to eighteen months have elapsed. The degree of disability of course depends upon the capacity for effort. There is no reason why a diseased heart cannot be injured as readily as a normal one, as shown in a case reported by Beck, in which a man, aged forty-

nine, struck his chest against the steering wheel when the car he was driving hit a bridge. He sustained fractures of the ribs and on the third day after the accident he became very ill and had a rapid pulse, and following this he had slight cyanosis, slight dyspnea and a pulse rate of one hundred thirty-six per minute with frequent premature ventricular contractions. The electrocardiogram showed evidence of myocardial injury and the heart sounds were reduced in intensity. The sternum was slightly depressed and had a transverse fracture line in its lower third. There were also fractures of the 4th, 5th, and 6th ribs on the left. He died on the sixth day from myocardial failure and at autopsy there were two contusions 18mm in diameter in the posterior wall of the right ventricle with a laceration of the myocardium between these two areas but no rupture. There was also found old pericardial adhesions, coronary sclerosis and some fatty infiltration of the heart.

It is conceivable that certain pathological changes such as marked hypertrophy, because of the increased bulk of the heart and the larger area of proximity to the thoracic cage, would amplify the susceptibility from lesser degrees of external violence. When previous heart disease is present and myocardial contusion has occurred, the myocardial injury may have recovered completely, but by its occurrence and the length of time required to heal, acts as a sufficient burden to aggravate the previous heart disease. The time interval before the onset of symptoms is of utmost importance in determining whether the heart disease, especially coronary artery occlusion, is only coincidental to the injury or whether a true myocardial contusion exists.

The absence of microscopic or macroscopic evidence of damage to the heart at autopsy following external chest injury cannot be taken to mean that death was not caused by cardiac standstill or ventricular fibrillation, especially if the death immediately followed the blow. The experiments on dogs showed that death may be due to cardiac standstill, ventricular fibrillation, complete heart block or other cardiac arrhythmias and occur so suddenly that by our present methods no demonstrable pathological changes are found.

The hesitancy of the medical profession, individually or as a whole, in making a diagnosis of cardiac injury may delay or prevent justice and interfere with individual rights. This is exemplified in the case, reported by Bishop,[44] of paroxysmal auricular fibrillation which became permanent following external chest injury. The case was decided in the New York Supreme Court in favor of the claimant and appealed on the grounds that claim should have been made within four months after injury.

Brahdy and Kahn[45] stated, "That no conclusion of a traumatic

heart case should be given without a consideration of the following items: (1) The physical and psychic condition of the patient prior to his injury. (2) The type and site and severity of the injury. (3) The immediate effects of injury both objective and subjective. (4) Bridging symptoms or course of the traumatic symptomatology from the injury to the onset of the disease. (5) The latent period of the disease, that is, the time interval between the occurrence of the injury and the appearance of the disease. (6) The diagnosis of the disease, its mode of onset, the site of the injury and its course." Kahn and Kahn[29] have also given the following compensation criteria: "In considering the subject of trauma to the thorax with intrathoracic cardiovascular injury from the point of view of the compensability of the accident, several important criteria must be established.

1. From a labor standpoint, the heart is healthy if a man is able for a long period of time to pursue his occupation without distress and without long periods of absence from work.

2. If following direct or indirect violence to the chest, signs of an intrathoracic cardiovascular lesion develop, which are incapacitating, they must be considered the result of an aggravation of a previously existing asymptomatic lesion, or the result of damage to a previously normal heart.

3. As in heart strain, the time that elapses between the accident and the development of disabling symptoms is very short. There must be immediate pain with its dyspnea, rapid irregular pulse, faintness and cold sweat, and immediate partial or total disability, in order that causal or aggravating relationship be clearly established. Temporary improvement, with return to usual or lighter work, followed by a recurrence of the condition may occur. But in these cases, the reappearance of the symptoms and signs should be attributed to the original injury." I personally do not entirely subscribe to opinions No. 1 and 2.

Kauffman[46] stressed an important legal point pertaining to dissecting aneurysms in that they do not form as an immediate effect of an accident and that the elapsed time is generally great enough to relieve an insurance company of any responsibility. Conversely, a large aneurysm noticed immediately after an accident generally speaks against any relationship between the aneurysm and the accident.

Schneider[47] in discussing cardiac rigor mortis states that there are various factors influencing the onset and duration and that after a mechanical injury of the heart such as a gunshot wound, contusion or rupture, an intense and long rigor is noticed, especially if the heart involved was a powerful and normal organ. This observation requires further investigation because by this means,

if it can be made an accepted fact, cardiac trauma could be established in those cases in which external violence caused sudden death by auricular fibrillation or cardiac standstill without demonstrable cardiac or other pathological lesions.

The increase in the scope of what constitutes compensable disability under the various state industrial commissions, to the extent that aggravation is included, has led to more physical examinations before employment. This has worked a definite hardship upon the compensated cardiac seeking employment because of the employer's fear that minor trauma may aggravate the already present heart condition and produce a permanent total disability when ordinarily only temporary, or at most, partial disability would have resulted. The simple answer to this problem appears to be waiver, but a complete analysis reveals this to be impossible within our present legal concepts. The second-injury clause offers some hope of an answer but still falls far short of an equitable solution.

Occasionally the evidence will show that a lesion is produced by exertion and a finding will be accordingly made by the court when it is incompatible with medical facts. As in the case of *Ohio Industrial Commission v. Smith.** Smith while working had to frequently climb a long flight of steps to repair a belt. The condition was diagnosed by a physician as a mitral lesion of the heart from which he later died. The decision of the court was against the defendant because no injury was shown and there was no trauma, but on appeal this verdict was reversed and it was held, "Over exertion causing a heart lesion would be compensable since the lesion was not caused by the prolonged action of any ulterior force of condition but, as far as the evidence was concerned was a direct result of over-exertion in the frequent succesive climbing of the steps." The heart lesion in this case was a mitral lesion and it is common knowledge that it is caused by a disease, rheumatic heart disease.

It is hardly equitable for compensation to be awarded in the meaning of the Act, when the employee is suffering from such an advanced degree of heart disease that death may occur at any time from any exertion and the deciding factor is only that it occurred while doing the ordinary and usual exertion or the work of his employment. There is no doubt that exertion and strain have caused numerous deaths, but compensation in some states is allowed only when it is extraordinary or unusual although there are many conflicting legal opinions and decisions. The legal aspect has deteriorated in some states to such a degree that the employee need only be on the premises of the employer.[48]

* 45 Ohio App. 362, 15 Ohio L. Abs. 703 (1933).

From a medical point of view there is but slight difference between strain or over-strain in angina pectoris because angina pectoris is a symptom syndrome which is characterized by a pain on effort. In the case *Ohio Industrial Commission v. Harney,** the employee died as the result of an attack of angina pectoris after pushing heavy cars of clay. There was evidence that it was extremely hard work, and that the employee was an overseer and was not accustomed nor required regularly to do the heavy work he had been performing. A verdict for the Claimant was affirmed and it was held, "That such death is compensable." The coroner clearly testified that "It was his opinion as a physician that the exertion which the deceased was subjected to brought on the attack which caused his death and the court takes notice that there was no testimony submitted by the defendant to show that this condition could have been caused by anything else, and therefore the record stands uncontradicted that the defendant's death was due to some exertion and strain to which he was subjected, according to the evidence." It is interesting to speculate on the medical argument in this case. Since death occurred in a short time the correct diagnosis was probably a coronary artery occlusion with myocardial infarction due to a thrombosis rather than angina pectoris. If this is true then the question of strain or overstrain must be reconsidered because at least fifty percent of all coronary artery occlusions occur while the individuals are at rest.

The solution of most of the medicolegal problems especially legal liability is based upon the establishment of the proximate cause of death. The definition given by the court in the case of *Laidlaw v. Sage,** was that, "The porximate cause of death must be understood to be that cause which in natural and continuous sequence, unbroken by any new cause, produces that death, and without which it would not have occurred." The proximate cause may be the last or nearest act to death but this is not necessary, neither need it be the sole cause but must be a concurring cause** such as might reasonably have been contemplated as involving a result under the attending circumstances, as illustrated by the following case of a man who had a normal heart, but developed an auricular fibrillation following a blow to the chest from the steering wheel of an automobile during an accident. This irregularity resulted in an embolism which resulted in his death quite some time later. Since the question of proximate cause is one of fact it is a question for the jury to decide, except when the facts are such

* 30 Ohio L. Rep. 559 (1929).

* 158 N.Y. 73, 79, 52 N.E. 679, 688 (1899).

** Gibney v. State, 137 N.Y. 1, 33 N.E. 142 (1893).

that they support only one reasonable inference which then makes a question of law.

Hyman and Parsonnet report,[49] "The case of Moon vs Order of United Commercial Travelers of America (146 N.W. 1037) was an action to recover on a policy. On the day of his death, the assured while going up the steps leading into his yard, accidentally slipped and fell with force and violence, striking his body near the region of his heart upon a large stone with such force as to cause a rupture of the left auricle of his heart from which he immediately died." It was held that the complaint stated a good cause of action and that the death was caused by external bodily injury as contemplated by the indemnity clause of the policy. Though a pre-existing condition of the heart may have more readily permitted rupture of the auricle, it was not shown that death would have occurred at the time it did, but for the accident. The jury found that the accident was the proximate cause of the death. The fact that a person fifty or fifty-five years of age would be likely to have more or less normal degeneration of the arteries in those parts which might tend to bring about a rupture of the heart in a case of a violent accident is not sufficient evidence to show that the accident was not the proximate cause of the death of the assured. It was contended that the walls of the heart, the left and right auricles, were more or less thinned by the effect of advancing years and it was argued that because of their condition, the defendant should not be held liable. To this, the court said:

> If the insurance is to be defeated because of the fact that the walls of the heart grow thinner by advancing years, or the arteries become sclerosed, or the valves of the heart act improperly, and this condition is the result of age; then the collection of the insurance money may nearly always be defeated by the fact of increasing years which inevitably change the condition of the assured.

Hyman and Parsonnet[49] have admirably summarized these problems as follows:

> In actions on policies of accident insurance where the deceased has died as a result of an accident which was the immediate proximate cause of death within the meaning of the policy, a definite liability attaches. Although many diverse and contrary citations are found, in general the problem has centered around the establishment of the proximate cause of death. In this, both the physician and lawyer must meet on a common ground in order to render justice as equitably as possible.

It is very difficult to ascertain when over-exertion occurs if it should be considered an accident, because the slight normal exertion of every day life may be enough to overstrain a diseased heart while another heart may withstand tremendous exertion.

There are many cases of so-called dilatation of the heart occurring at work while the employee is under some particular strain. He may be lifting some unusually heavy load, or doing heavy lifting in an awkward position. Probably the word dilatation should be dropped from the description because it leaves the impression of a pathological change. I doubt if dilatation can be produced in this manner to a normal heart.

The traumatic etiology of infectious endocarditis must have an infection added to the injury. This opinion is more or less universal and is compatible with the pathology of endocardial injury and also of endocarditis. The valve lesion and murmurs produced from the healing of endocardial trauma and tears should not be considered as an endocarditis.

So far as death from heart disease is concerned, the weight of authority is that liability falls on the one who, by his wrongful or negligent act or default, accelerates a diseased condition thereby hastening and prematurely causing the death of the diseased person, even though the disease would probably have resulted in death at a later time without his agency.*

The greatest obstacle in preventing technical knowledge being given by a medical expert witness is that in the majority of hypothetical cases the death certificate is incorporated as a fact and considered prima facie evidence. Many times the physician who signed the death certificate and stated the cause, has not seen the person within twenty-four hours before death. Occasionally they only viewed the body after death. The person probably has been a patient of the physician's for some time but still he must rely on others for the immediate events occurring at the time of death. In many cases he has a reasonably correct cause based on the known mathematical probability. If he does not sign this instrument or if he honestly states that he does not know the immediate cause of death it becomes a case for the coroner. However, this act creates poor public relations between the physician and the deceased's family. Furthermore, he knows from a practical standpoint nothing is obtained because the coroner will, unless some questionable criminal act is involved, receive information from him and others and establish an immediate cause of death.

This can all be corrected by obtaining a necropsy. In my opinion if the physician in charge of the case did not see the patient within twenty-four hours of his death, or states that he does not know the immediate cause of death, a necropsy should be manditory. Further, in all legal cases in which a necropsy was not performed, the signer of the death certificate should be called as a witness so that he can be placed under oath and by the rules of

* McCahill v. N. Y. Transp. Co., 201 N.Y. 221, 94 N.E. 616 (1911).

evidence it can be determined whether or not his statement is a fact or hearsay. This type of procedure would also make vital statistics of the utmost medical importance and reliability.

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