# The Heart and Circulation

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## SYMPOSIUM ON TRAUMA AND HEART DISEASE

The following article is one of three planned as a symposium dealing with the medical and legal problems arising from the effect of injury on the heart. The purpose here is to acquaint the attorney with the medical background necessary to the preparation of scientific proof required in personal injury and compensation cases. Later articles will deal with the legal aspects, particularly proof and causation, and finally with the problems confronting the expert witness.

# THE HEART AND CIRCULATION

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#### I. Normal Anatomy

The human heart is a dual pump — right heart and left heart — arranged in series, so designed that it looks and acts as a single organ. It consists of four chambers, each "heart" having two: one, a thin muscular reservoir sitting most superiorly, which is called the atrium; and the other, which sits inferiorly is the thick muscular actively contractile part of the pump, called the ventricle. The two "hearts" are joined into one unit by having a common vertical partition which separates right from left.[1]  Each atrium is marked by a number of openings through which blood enters. The atrium is connected to the ventricle by a single opening which is guarded by a valve called the atrio-ventricular valve; in the right heart known as the tricuspid valve, and in the left as the mitral valve.  Each ventricle, by contraction, forces the blood (stroke volume) into the circulation. The outflow ·of each ventricle is guarded by a semilunar valve which consists of three leaflets. These prevent the blood from returning into the ventricle during the filling phase. The blood from the right ventricle is propelled into the lungs (pulmonary circulation), and then is delivered to the left atrium through four openings.  From the left atrium, it passes through the mitral valve into the left ventricle. The output of the left ventricle is propelled into the great vessel known as aorta and there is distributed to all parts of the body in which the various organs are hooked up in "parallel." Blood returns to the right atrium through two large veins, superior and inferior vena cava. The right and left heart, therefore, are "in series."

The "right" heart has the smaller capacity, has a thinner wall, creates less pressure than the "left," and is known as the pump for the pulmonary circulation. The "left" heart is a part of the systemic circulation. The heart is encased in a fibrous sac called the pericardium.

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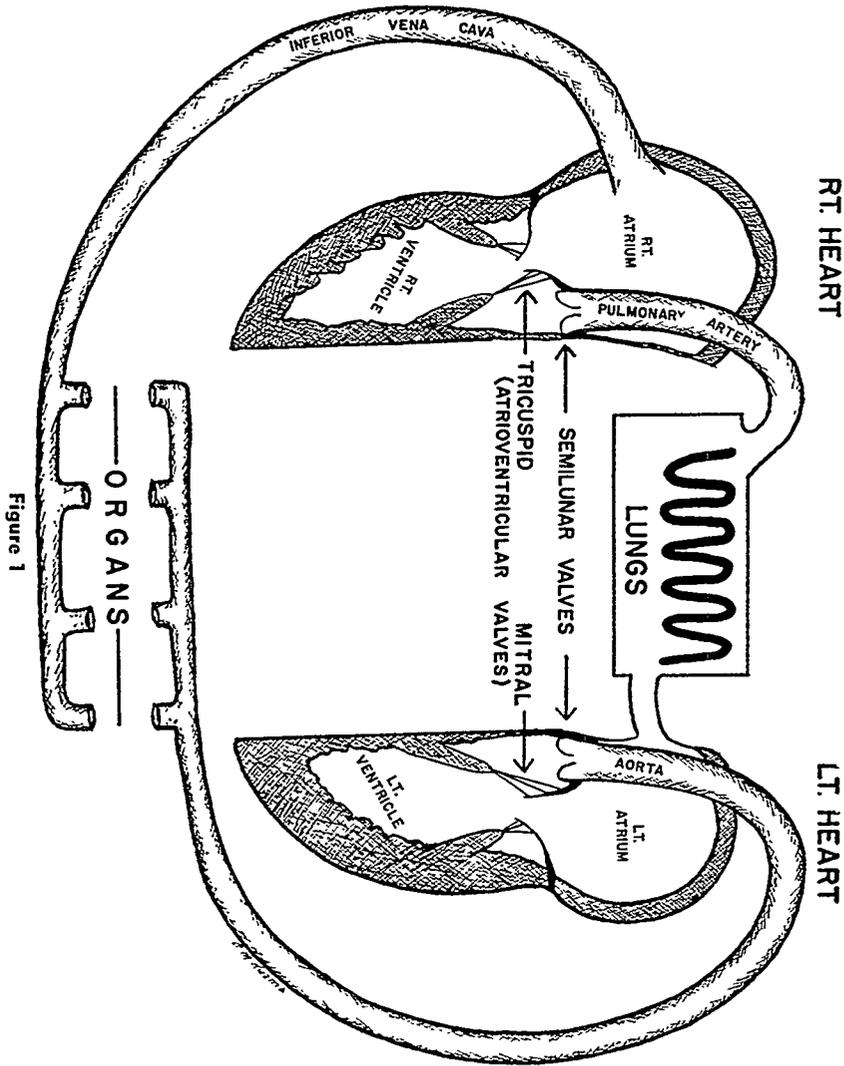[1] Figures 1 and 2.

Figure 1

The lining of this sac is lubricated by a small amount of fluid. The heart is attached at its superior most portion by means of the vein channels that enter, particularly the right atrium. It occupies the position between the two lungs in the center of the chest. However, the muscular ventricles form a blunt pointed structure which projects to the left of the midline in the area of the left nipple or breast. (It is the size of a man's fist and weighs about 300 grams, or 10 ounces. Much of the heart, however, is in the midline and even extends to the right.)

These two pumps are connectel to a closed system of pipes or

blood vessels. The vessels which carry blood away from each ven-
tricle are called arteries. These are thick-walled muscular elastic
vessels and ordinarily carry blood rich in oxygen. The artery from
the right ventricle, however, carries blood poor in oxygen, and sends
it to the lungs to be oxygenated. The vessels which return the blood
to the heart are called veins. These are thin-walled elastic and mus-
cular vessels with valves, placed at various intervals along the way,
directing the flow of blood toward the heart. Arteries are normally in-
terpreted by lay people as those vessels which pulsate and carry red
blood. Veins on the other hand, are interpreted as those superficial
vessels which are blue in color and do not pulsate. Small arteries are
called arterioles, and the small veins are called venules. Between the
arterioes and venules is an extensive plexus of very tiny, extremely
thin-walled vessels (capillaries) which give the skin its pink color.
The great mass of blood circulating through the body is in these
capillaries.

In normal circulation and function of the heart, each portion of
this large circulatory system has a volume of blood which is related
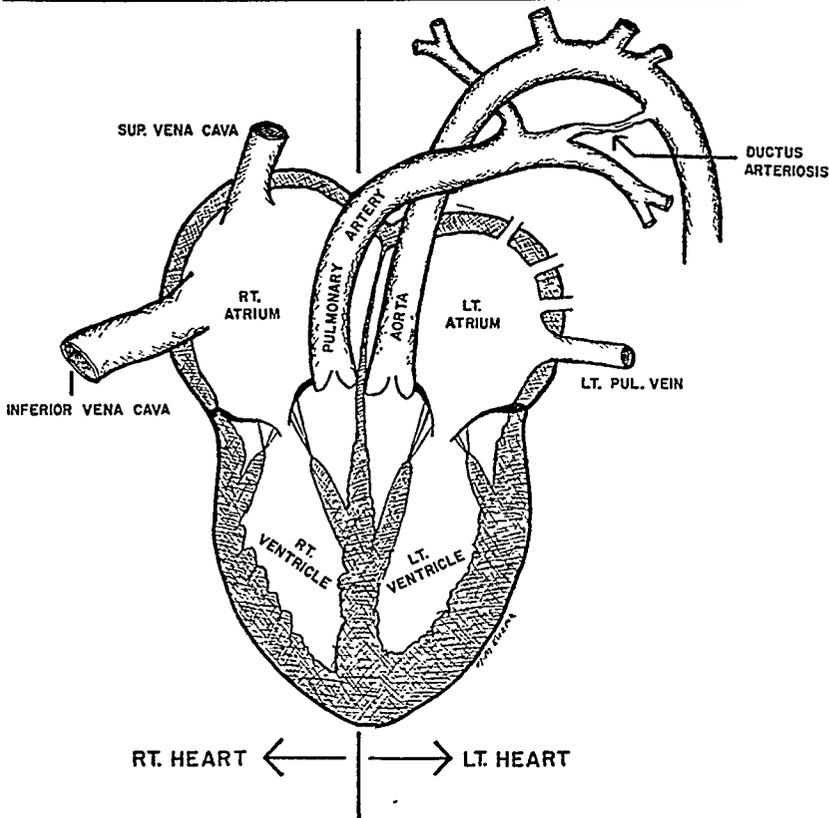


Figure 2

to that in other portions; that is, the volume within the chambers of the heart, in the arteries, in the veins, and in the capillaries, is all correlated so that there is a uniform circulation of blood without abnormal accumulation in any particular area.

Each organ has its own artery from the main aortic circulation, and is hooked-up to the feed-line aorta in "parallel" arrangement. Nourishment of all tissues comes from the blood supply. Although the heart is bathed with blood on its internal surfaces, it does not receive sufficient nourishment just by contact on the lining of its chambers. Accordingly, it has its own arterial blood supply through two vessels, called coronary arteries, right and left. These originate from the aorta immediately above the left ventricle of the heart and encompass the base of the heart as a crown might fit on a head; therefore, they are referred to as "coronary" arteries. From the crown type of arrangement, branches extend longitudinally along the chambers of the heart towards its very point or apex.

## II. Normal Physiology

The actual contracting and functioning actions of the heart are exceedingly intricate. There is innate natural contractility of heart muscle and it may show muscular action for some time after it is separated from the body. However, the normal intact heart has a built-in "communications" system which fundamentally controls its rate and force of contraction, and coordinates the action of all parts. This is called the conduction system, fibers of which run in all four chambers but begin in the right atrial chamber. The heart is also governed by the nervous system through the parasympathetic vagus nerve, and by outside regulators such as the carotid sinus in the neck, and the adrenal glands which produce adrenalin. In normal action, all chambers of the heart are synchronized and effectively propel blood along the way. Force of contraction is governed in part by the tension on the heart muscle itself. When a chamber (ventricle) is slightly over-distended, it will contract with greater force to expel the blood which it had acquired. Under usual circumstances, the heart will contract approximately 72 times each minute and will have ejected several liters of blood.

The pathway of the circulation is somewhat as follows: blood from various portions of the body is brought to the right atrium by two principal veins called the superior and inferior vena cava. The right atrium will by contraction, by gravity, and by increasing load, pass the blood to the right ventricle through the tricuspid valve. The right ventricle, upon contraction, propels the blood into the pulmonary artery through the semilunar valve, and the blood is thereby distributed through the ramifications of the pulmonary artery within the lung. After passing through the capillaries of the lung, the blood is re-

collected into larger channels called veins and four such veins empty
into the left atrium. The blood is then passed on to the left ventricle,
through the mitral valve, and on contraction of the left ventricle, it is
sent into the aorta through the aortic semilunar valve. The aorta has
many branches and each major organ has a principal artery coming off
directly from the aorta. After passing through the capillary bed of
the various organs, the blood is recollected into the venous system and
brought back to the right atrium and the cycle is repeated. Within this
circuit of propelled fluid, a certain level of hydrostatic pressure is
maintained. The pressure created by the contraction of the ventricles
in propelling blood into the arteries is known as the systolic pressure
and ordinarily is in the neighborhood of 120 mm. of mercury. Im-
mediately after contraction, the pressure in the vessel falls. A certain
level is then maintained until the ventricle contracts again and creates
a systolic pressure. This sustained pressure within the blood vessels
between contractions of the ventricles is known as diastolic blood
pressure and is roughly in the neighborhood of 70 mm. of mercury.
It is rather clear, therefore, that systolic pressure in great measure
results from the force of the contraction of the ventricle, and that the
diastolic pressure depends in the main on the state of the blood vessels,
namely, how much resistance the vessels present to the circulation of
the blood (constriction increases resistance), and how effective they
are in dissipating the systolic pressure by their elasticity. Exercise
and effort increase blood pressure; rest and sleep are associated with
a decrease.

The pressure in the outgoing vessels from the heart is gradually
diminshed as the finer ramifications are reached so that in capillaries,
the blood is propelled through this network with a very small head of
pressure. On the other side of the capillary bed where the blood is
being collected into veins to be returned to the heart, the pressure is
low and at times is a negative pressure; particularly as created by
actions of respiration so that blood is, in effect, "lifted" to the heart
from the lower extremities rather than forced by intraluminal positive
pressure.

It should be relatively clear at this point, that variations in the
number of contractions that the heart makes in a period of time, the
resistance against which the heart must work to propel the blood, and
abnormal accumulations of blood in various systems within the body,
may adversely influnece the effectiveness of the circulation. Never-
theless, the heart has a very marked ability to compensate for stresses
placed upon it, but the compensation may not be effective over an
indefinite period of time. The ability of the heart to perform effectively
during states of increased demand is called cardiac reserve. Reserve
depends on increase of heart rate, increased filling of ventricles, in-

creased coronary circulation, and perhaps hypertrophy (within limits) of the heart muscle. When the heart chambers do not empty completely, there accumulates an increase of blood within such chambers. This leads to over-distention of the chambers which is known as cardiac dilatation. Each individual heart muscle fiber is thereby lengthened and this is associated with a greater contracting power. This is compensatory for a period of time, but when the stretching is beyond optimum length, effectiveness of heart contraction decreases. Continual over-stretching of the heart muscle over a period of time increases the size of the heart muscle fiber which is known as cardiac hypertrophy. However, as the fiber becomes larger it requires more nourishment to maintain its own metabolism, and when the circulation cannot effectively meet the increased demand, heart failure sets in. This is the state in which the heart is unable to perform its functions without causing symptoms of illness and is known as cardiac decompensation. This implies the inability of the heart, as a pump, to supply sufficient blood for the various needs throughout the body.

### III. Signs and Symptoms of Decompensation

The findings in a case of cardiac decompensation might be listed for better appreciation of the concomitant physiological and anatomic abnormalities:

A. *Pulmonary vital capacity is decreased* — the volume of air contained in the lung is reduced whenever the pumonary vessels have an increased pressure and are distended. This is both mechanical replacement of air space as well as interference with the elasticity of the lung to accommodate larger volumes of blood.

B. The *circulation time of blood* within the lungs is increased since pulmonary pressure is greater and the failing heart cannot overcome the increased pressure. Hydrothorax, or the accumulation of fluid in the chest cavities, results. It occurs also in the abdominal cavity (ascites) as well as in the extremities, particularly in the dependent portions. Much of this is related simply to the hydrodynamics in which the pressure within a vessel is increased sufficiently to force fluid to the outside, causing edema or "dropsy."

C. *Shortness of breath, or dyspnea,* is related to the slowness of circulation through the lungs, the increased pressure within the veins of the lungs, and the decreased ability of the lungs to contain an adequate volume of air. Orthopnea means shortness of breath when in the lying position. This is in great measure related to congestion and increased pressure within the vessels of the lung. Upon assuming a sitting or standing position, much of the pressure may be relieved by gravity and the circulation through the lungs might be improved.

D. The actual *volume of blood* is increased in heart failure. Stimulation of bone marrow to form more blood and retention of salt

which attracts water is the explanation. Any organ of the body may become enlarged as its venous pressure is increased and the condition of "passive congestion" develops.

E. Blueness of color, or *cyanosis,* is related to the presence of blood with insufficient oxygen. This may be due to stagnation of circulation in heart failure, or may be related to disease of the lungs wherein normal oxygen exchange cannot take place.

## IV. DISEASE STATES OF THE HEART

Disturbances of the heart and blood vessel system, known as pathological states, are basically of one of three types. These will be given below and a short explanation of the various important categories within each type will be mentioned:

A. *Principally physiological disorders of the heart which lead to failure:* Irregularities of heart function, principally those of rate and rhythm of the pump action are significant. There is a wide range of normal or physiological cardiac activity within which range the rate of contraction (number of beats per minute) of the heart as well as the output of each contraction sustain life without symptoms of distress. Increased rate of heart action takes place with exercise and emotional disturbances. Such ranges, of course, are considered to be within physiological normals. However, if one speeds the rate of heart contraction beyond 150 times per minute, then incomplete filling of the ventricles may take place and this, of course, would be reflected in reduced output of each contraction, and therefore reduced coronary circulation. However, the heart in most instances requires more nourishment. A very rapid heart rate (tachycardia) will seriously reduce the cardiac output and thereby cause increased venous pressure and distress, or evidence of heart failure. Serious abnormalities in rate leading to distress and even death may develop from intoxication, such as belladonna poisoning.

There are also irregularities of cardiac *rhythm* (relationship of recurrence of heart contraction) which may adversely influence the heart in doing its work. In auricular fibrillation, instead of a uniform synchronized contraction of the atrial heart muscle, there are irregular convulsive movements which are not effective in propelling blood. This timing disorder is frequently associated with other diseases of the heart that are anatomic in nature, such as rheumatic fever. A heart suffering from auricular fibrillation is at a disadvntage since the actions of the atrium are not synchronized and fully effective. Great energy may be expended with little work accomplished. Mural thrombosis (blood clot on atrial walls) develops in arrythmias and presents a threat for infarction of other organs by embolic occlusion of vessels. (Clots loosened and carried in the blood stream plug a vessel and cause death of the tissue supplied by that vessel).

A condition similar to auricular fibrillation is called ventricular fibrillation. In this instance, the heart muscle of the ventricle itself is undergoing tremulous or convulsive movements without effective contraction. This condition is not compatible with life and, unless corrected immediately, death usually results in a matter of a few minutes. Occurrence of ventricular fibrillation and cardiac arrest during anesthesia and surgical procedures has recently been emphasized.

A decreased rate of heart contraction is called bradycardia. Some degree of slowing of the rate occurs with rest and sleep, however, an extreme degree may be brought on by various disturbances, such as jaundice, typhoid fever, and tumors within the skull. In a greater number of patients, slow heart rate develops from interference with the conduction system within the heart itself. This may be produced by drugs or by scar tissue that involves the conduction system. As heart rate is decreased, a point is reached wherein the brain receives insufficient blood supply and the individual suffers from fainting, or syncope. In any instance of sudden decrease of blood volume or pressure, syncope may develop. Many of the disorders which relate to consciousness or syncope have much to do with nerve innervation and emotional disturbances. The vagus nerve and vaso-vagal reflexes which govern heart action are intimately tied into this particular phenomenon. It is important to note that a normal cardiovascular system recovers quickly after such syncope and the individual is restored to his full health in a matter of minutes. However, in disease states of the cardiovascular system, the return to normal may be slower than usual and, in fact, death may occur from cardiac standstill or ventricular fibrillation.

Closely related to the condition of syncope is the problem of shock. This is frequently defined as an altered state of health characterized by a markedly decreased blood pressure, decreased circulating blood volume, decrease of cardiac output, rapid weak pulse, pale, cold, moist skin, and loss of consciousness. Such a state may result from nervous influence, severe trauma, and loss of blood. From a teleological standpoint, the reduction in blood volume, the drop in blood pressure, and the syncope which attends the state of shock, are beneficial. An organism suffering blood loss, on becoming unconscious, assumes a horizontal position which improves cerebral circulation, and at the same time reduces the demands of other organs. As blood pressure and blood volume are decreased, there is a tendency for hemorrhage to stop. Shock, therefore, may be effective in saving life; however, sometimes shock is an irreversible phenomenon. In these instances, the continued decrease in cardiac output leads to oxygen starvation in many tissues, and the brain being extremely sensitive to oxygen want, may suffer irreparable damage.

B. *Chiefly anatomic conditions which place an undue work load on the heart:* The outstanding example of this is the disease of hypertension, or high blood pressure. This is characterized by an increase in both systolic and diastolic pressure and is usually related to constriction and inelasticity of the small vessels called arterioles. The disease frequently begins with a disorder in the kidney characterized by decreased kidney circulation. Under such circumstances, a substance is produced in the kidney which upsets the humoral balance controlling arteriolar size and causes the blood vessels to constrict. Continued constriction of the arterioles, leads to degenerative changes within the vessel walls. Degenerative changes consist of loss of elastic tissue and muscle fibers, and replacement by a rather dense uniform inelastic material. The channel of the vessel is thereby greatly reduced. Reduction in the calibre of the small vessels throughout the body places a severe load on the heart. The heart finds it difficult to empty its chambers against an increased resistance interposed by constricted arterioles. The heart, first, stretches (dilates) to accommodate the increased volume of blood which accumulates as the chambers cannot completely empty; and as a result, the muscle fibers eventually enlarge (cardiac hypertrophy). After a time, the dilatation and hypertrophy cannot cope with the increased work load, and gradually the heart accumulates more and more blood within its chambers, and more and more of it backs up into the venous side of the heart, elevating peripheral venous pressure. All signs and symptoms of heart failure then become evident (left heart failure). Similarly, diseases of the lung which reduce the blood vessel volume within the lung, such as progressive fibrosis, place an undue load on the right ventricle, leading to decompensation (right heart failure). Terminally, total failure occurs and one does not distinguish between right and left failure.

A congenital narrowing of the aorta, called coarctation, also produces a severe load on the heart. In such instances, the heart may dilate and hypertrophy over a long period of time and years may elapse before heart failure, "decompensation," becomes evident.

Ability of the heart to empty itself may be seriously hindered by diseases of the heart valves. These commonly are the result of rheumatic fever, syphillis, and bacterial inflammations of the lining of the heart (endocarditis). Such diseases produce two types of valvular dysfunction: one is called stenosis or narrowing of the valve opening. This is especially true of the mitral valve with the disease of rheumatic fever. The other is enlargement of the valve opening to such an extent that it is incompetent. This is called valvular insufficiency and is best demonstrated in the disease of syphillis. Acute inflammation (Aschoff nodules — collections of cells of inflammation about degenerating supporting connective tissue) of the heart valve in rheumatic fever is

followed by adhesions between the leaflets of the valve, thickening and stiffening of the leaflets, and severe distortion produced by gradual shrinkage of the scar tissue. This takes place over the course of many years.

In syphillis, on the other hand, there is a degeneration of the elastic and muscular tissue of the aorta at its origin. This weakens the wall to the point of a blowout. However, weakening at the heart valve causes stretching and pulling apart of the leaflets so that they cannot make contact and close.

A chamber which must empty itself against a narrowed valve opening undergoes dilatation and hypertrophy and it is only a matter of time before this will no longer compensate for the increased load and, thereafter, the heart will be in "failure." Some of the largest hearts (greatest hypertrophy) are seen with stenosis of the aortic valve. When the mitral valve is narrowed as by rheumatic fever, the inability of the left atrium fully to empty itself through the narrow opening into the ventricle is reflected backwards into the lungs, into the pulmonary artery, and into the right ventricle, which becomes enlarged, since it is the chamber that must overcome the load. Valves which are incompetent (allow blood to go in retrograde direction) are accompanied by dilatation and hypertrophy of the chamber which doesn't empty itself fully since blood returns to the chamber rather than being directed forward during the resting phase (diastole) of the pump action.

A condition quite similar to overloading of a chamber by an incompetent valve may also result from malformations of the heart during its development. Such are known as congenital anomalies, and the important ones are associated with the abnormal shunting of blood to chambers and vessels where the added volume creates extra load. A disorder of this kind is known as the persistent patent ductus arteriosus which means a channel communicating between the pulmonary artery and the aorta. It serves as a physiological shunt (pulmonary artery to aorta) during intrauterine life. However, after birth this shunt is no longer needed and spontaneously closes since the lungs are expanded with air and can take all the blood which the right heart expels. In postnatal life, the pressure in the aorta is greater than in the pulmonary artery. If the ductus remains open, then with each contraction of the heart, the blood will be sent from the aorta through this ductus, into the pulmonary artery. Eventually the pulmonary artery will dilate and may even rupture.

An opening between the two ventricles through the interventricular septum is called Rogers disease, (patent interventricular septal defect) and it too is associated with increased volume and load in the right heart since after birth the pressure of the left chamber is greater

than the right.

Pulmonary stenosis is a congenital anomaly with narrowing of the outlet of the right heart at the pulmonary artery. This, of course, places a severe work load on the right ventricle each time the ventricle attempts to empty itself against the narrowed opening. This causes dilatation and severe hypertrophy of the right ventricle. Cardiac dilatation and hypertrophy compensate for the above difficulties for a period of time. However, decompensation is the final chapter.

Another serious obstacle to health and life in individuals with rheumatic valvular disease and congenital anomalies is bacterial endocarditis. Erosions of heart lining membrane (endocardium) on damaged valves and at congenital defects permit bacteria to grow therein. Unless the bacteria are controlled, death is certain from *septicemia* (blood stream poisoning by bacteria). However, even eradicated bacterial disease of heart valves has left its injurious mark on the previous disorder and life is frequently shortened.

C. *Ineffective pump musculature:* In this category is a group of conditions which are characterized primarily by heart muscle diseases which render contraction and pump action ineffective. Some of these are related to nutritional disturbances, such as the disease of beriberi, or Vitamin B1 deficiency. Likewise, in chronic alcoholism, the development of a heart muscle filled with fat may be attended by loss of vigorous contraction. The heart muscle in diseases of high fever (typhoid, meningitis, etc.) frequently is at a disadvantage in its ability to contract, (toxic myocarditis). There are unusual instances wherein the heart muscle itself is the seat of infectious disease, such as Fiedler's myocarditis. The severe infiltration of the heart muscle by cells of inflammation precludes its function as a contracting, work-performing organ. Serious disturbances of the heart action may also result from changes in the concentrations of certain essential substances in the blood and tissues, namely the elements of calcium and potassium.

The most important disease affecting the ability of the heart muscle to contract and propel blood is that which is known as myocardial infarction. This is a condition in which a portion of the heart muscle undergoes death and eventual replacement by connective tissue scar. Infarction is caused by sudden loss of blood supply to the heart muscle as occurs in occlusions of the hearts' vessels (coronary thrombosis). The sudden loss of the blood supply to the heart muscle may cause ventricular fibrillation or cardiac standstill and sudden death — without anatomic change in the heart muscle. If, however, such an individual survives, the heart muscle goes through a chain of events in the process of dying and being removed. The muscle is severely changed as evidenced by its appearance in a matter of 24 hours. At

this time, cells of inflammation and hemorrhage are prominent within the muscle which is undergoing death. Towards the end of the first week, the heart muscle is completely dead, it is being digested in the process of removal, and at this time it is extremely weak. The heart, in contracting, may actually break the wall and propel blood into the pericardial sac. Such an occurrence is called cardiac tamponade. In a matter of just a few minutes, the pressure within the unyielding fibrous pericardial sac may equal that within the chamber of the ventricle and as soon as this happens, the ventricle is no longer an effective pump and cannot propel blood to the organs of the body. Sudden death is the result of this. However, if the heart wall does not rupture, then it is gradually replaced by scar tissue over a period of weeks. While this is going on, however, the lining of the heart at the point of the muscle damage, may develop a blood clot. This is called a mural thrombosis. The clot which develops may be broken off by the actions of the heart and be carried in the blood stream to any other organ of the body where it occludes a blood vessel and causes an infarction just like that of the heart muscle itself. In the brain, this is evidenced by loss of motor function (paralysis), loss of speech, loss of consciousness, etc. Heart failure may occur at any time after myocardial infarction, but many months or years after such an attack, the scar tissue of the heart wall may begin to bulge externally. The pressure within the chamber causes the bulge which is called an aneurysm.

The story of myocardial infarction is not at all complete without discussion of its cause. The coronary arteries must be patent and carry a sufficient amount of blood at a sufficient pressure to maintain the nourishment of the heart muscle. The coronary arteries are subject to the pressure which is quite the same as that of the aorta and undergo a degenerative disease called arteriosclerosis. This means that the elastic and muscle fibers of these arteries are lost and replaced by scar tissue, by fatty deposits, and by calcium. Such calcium and fatty deposits may actually build up to a mass in the wall and project into the channel itself. This may reach the proportions of occluding the entire lumen of the vessel. More commonly, however, the bulging part of such a mass suffers loss of the lining of the vessel and a blood clot suddenly develops on such an area. This is called coronary thrombosis. Hemorrhage within the wall of the coronary artery, when it is diseased and replaced by fatty materials, causes a lifting of the lining and pushing of it to the opposite side; thereby, effectively occluding the circulation at that point. The result of coronary occlusion, irrespective of how it develops, is myocardial infarction.

A condition, frequently confused by the clinician as myocardial infarction, is a tearing of the aorta, called dissecting aneurysm. It

begins as a weakening and destruction of the elastic and muscle fibers of its wall (cystic medial necrosis). Hemorrhage develops in this site and then tears apart the inner and outer layers of the aortic wall. A double-barrel aorta is thereby produced; one channel (the normal one) with moving blood, and the other within the wall with a trapped collection of blood. Eventually, the blood collected within the aortic wall may tear an opening through the inner wall into the normal lumen or rupture the outer wall and produce a fatal hemorrhage into the chest cavity or into the pericardial sac (cardiac tamponade).

## V. FORENSIC CONSIDERATIONS

In general, there are only a few situations in which trauma or stress have dire consequences in the absence of preexistent cardiac disorder of some kind or other. Certainly a direct injury, notably penetrating wounds, such as stab or bullet. need not be related to preexistent cardiac disease. At times, nevertheless, one must weigh the results of such injury in a normal heart versus one with pre-existent disease; for example, a stab or bullet wound which does not completely penetrate the heart chamber, may be tolerated without evidence of decompensation or permanent illness by a normal heart. However, a wound of like depth on the surface of a thinned myocardium following infarction, may cause perforation and cardiac tamponade with sudden death. Likewise, the ability to repair a wound of the myocardium, may be related to the coronary artery supply and its efficiency. In such instances, healing of the myocardium might be retarded in the cases where interstitial fibrosis has resulted from coronary insufficiency.

In Group A, of part IV, the disturbances which may be aggravated or may result in fatality (the broad consideration of stress and trauma) have to do with ventricular fibrillation, shock, and cardiac standstill. These may be called physiological death with very poor, if any, anatomic correlation. Notable in this might be the example of the individual who collapses and dies while being interrogated by a police officer following a minor automobile accident. He may have been only a witness of the accident. There are also the instances of witchcraft and voodooism which have caused sudden death in presumably normal individuals. This must be related to severe nervous influence of the heart that may produce cardiac standstill or ventricular fibrillation. The effect may be mediated through chemical substances such as adrenalin which is sharply elevated by severe fright. Such people undoubtedly have an unduly excitable heart as the basic disorder.

In Group B, any situation which has produced an exaggerated work load on the heart, may be further augmented by effort or stress. This, of course, may be reflected in the usual signs and symptoms of cardiac decompensation, or may be associated with sudden death.

Sudden death may be caused by rupture of a blood vessel in hypertension, (cerebral hemorrhage or stroke) by rupture of an aorta with coarctation, by rupture of a dissecting aneurysm and cardiac tamponade, by sudden dilatation of the heart and by ventricular fibrillation in diseases of valvular character.

In Group C, the general disorder of diseased myocardium and its ability to act as an effective pump, may be seriously hampered by effort.   Certainly, physical effort may produce an increased blood pressure, increased coronary artery pressure, rupture and hemorrhage of a fatty deposit in the coronary artery and injury of the endothelium covering such a plaque followed by the development of a blood clot (thrombus).   On the other hand, sudden drop in blood pressure in the state of shock or even the physiological lowering during sleep may precipitate myocardial infarction in people with coronray artery disease. (These need a certain "head of pressure" to maintain adequate circulation through diseased coronary vessels).  Blunt injury to the chest wall may fracture a brittle coronary artery with its fatty and calcified plaques and lead to obstruction of the coronary artery.  Injury may also cause a severe bruise of the heart muscle.  Once a myocardial infarction has taken place, continued physical exertion may lead to perforation of the wall of the heart at the point of infarction, as is so well evidenced by its development in mentally ill people who do not seek medical help or do not abide by the orders of bed rest during this phase of illness.  Blunt injury to the chest and back and severe physical exertion have been known to cause either the inception of a dissecting aneurysm of the aorta or its perforation into the pericardial sac.  Nevertheless, it must be emphasized that the greater number of myocardial infarctions occur *without* physical exertion (during sleep, watching T.V., etc.) and that the overwhelming majority of dissecting aneurysm cases are unrelated to effort or injury.

It must be clearly understood, in conclusion, that all diseases of the heart, except those of external physical violence, occur as "natural" phenomenon.  Coronary thrombosis, myocardial infarction, dissecting aneurysms, rupture of cerebral vessels associated with hypertension, congestive heart failure associated with valvular disease — all of these produce invalidism and death without any consideration of "injury," external influence, work or effort.  It follows, therefore, that any external influence which adversely affects this system, may exaggerate or aggravate a naturally existing disease and hasten demise.  Serious consideration, therefore, must relate to quantitative relationships between natural disease and its aggravation by external influence.  Except for direct physical injuries and intoxications, practically all other influences of forensic consideration do not adversely affect the ability of the normal heart to carry on its work.