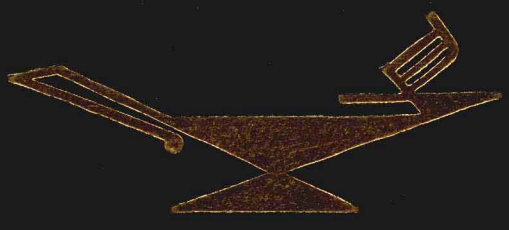
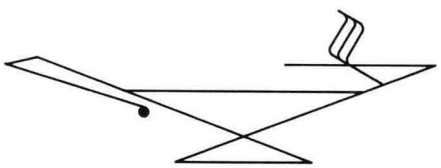


ENCYCLOPEDIA AMERICANA

VOLUME 14

Heart to India



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HEART

CONTENTS

Secton	Page
Evolution of the Heart	1
The Human Heart	2
Heart Rate	2
Stroke Volume	4
Structure	4
Coronary Circulation	5
Diseases of the Heart	5
Infectious Types of Heart Disease	6
Thyrotoxic Heart Disease	7
Congenital Heart Disease	7
Hypertensive Heart Disease	9
Coronary Heart Disease	10
Heart Surgery	11
Bibliography	12

HEART. In ancient times the beating heart was regarded as the very soul or spirit of man and the core or center of the body. Certain words pertaining to the spirit, such as "cordial" and "courage" are derived from *cor*, the Latin word for the heart. St. Valentine's day, which was celebrated as early as the 7th century, has as its emblem the human heart, although it is distorted in shape to resemble that of a diseased heart in which both sides are of equal size. Today, the heart is known as the organ concerned only with pumping the blood through the body, and the soul or spirit of man has been moved to the brain, along with man's thinking, learning, and other higher faculties.

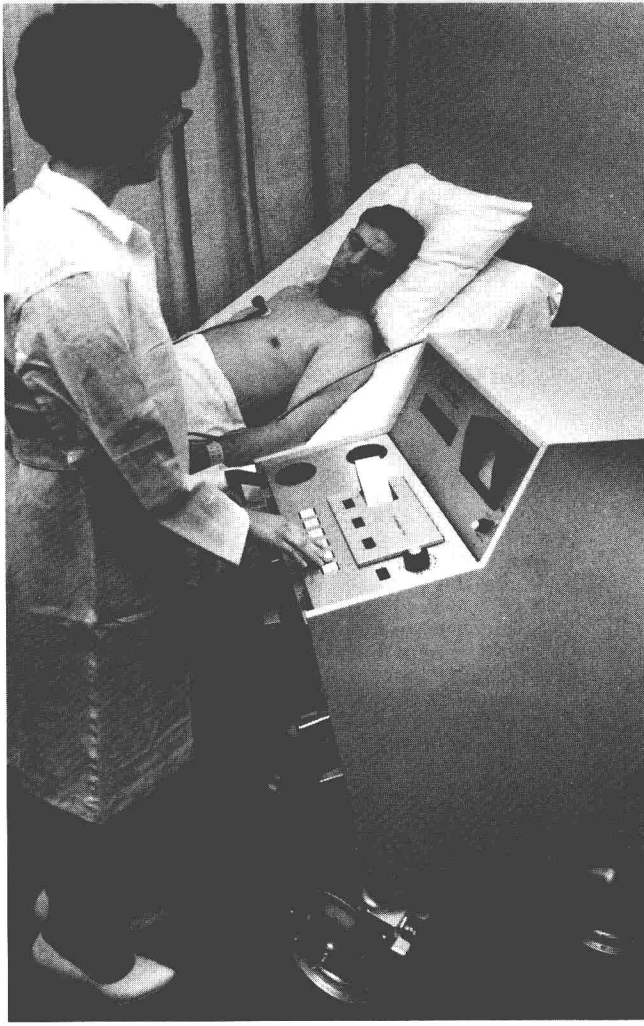
It was obvious even to primitive man that the pulsating heart was a pump distributing to the brain and all other parts of the body important substances, including nourishment from the gastrointestinal tract and liver and a vital spirit from the lungs. However, it was not until the Middle Ages and later that the details of circulation were discovered. First, the circulation of the blood through the lungs was worked out by Ibn al-Nafis, Andreas Vesalius, Michael Servetus, and Realdo Columbus. Next, the general body, or systemic, circulation was worked out by Fabricius ab Aquapendente, Andrea Cesalpino, and William Harvey.

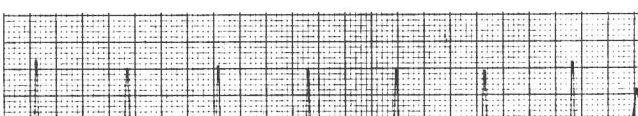
In later years it was found that certain accessory aids help the heart in maintaining an optimal circulation of the blood. These aids are generally not known and not adequately utilized, even by members of the medical profession. Undoubtedly, the most important aid is that of the large muscles of the legs which, on contracting, squeeze the veins in the legs. Since these veins are supplied with valves that prevent the blood from gravitating to the feet, the blood is actually pumped upward when the veins are squeezed, relieving the heart of about one third of its work when the legs are used actively, as in cycling, running, swimming, or jogging.

A second important aid is that of the elasticity of the *aorta*, the large artery that receives a jet of blood from the heart every time the heart contracts and converts these jets into a much smoother, more efficient flow of blood. When hardening of the aorta wall develops as a result of arteriosclerosis, the advantage of the natural elasticity is lost. Preventive measures developed by good health habits in youth undoubtedly delay the onset of arteriosclerosis and lead to a longer and healthier life.

A third aid is a freely moving diaphragm that is unimpeded by abdominal fat. When the diaphragm moves up and down, as it does in breathing, the chest cavity, or thorax, becomes a suction pump that helps to bring blood up from below as well as to bring air into the lungs.

A fourth aid is the arterial system's ability to adjust to the changing demands of different organs. After a meal, for example, during the process of active digestion, the blood supply to the gastrointestinal tract has greater priority than that to the skeletal muscles, which are at rest. During exercise, the situation is normally reversed, with the skeletal muscles receiving priority over the gastrointestinal tract.





CAMBRIDGE INSTRUMENT COMPANY, INC.

HEART DISEASE diagnosis is greatly facilitated by an electrocardiograph (top), which makes a chart, or cardiogram (bottom), of the heart's electrical impulses. The instrument shown also displays the cardiogram on a screen and encodes the data for computer interpretation.

EVOLUTION OF THE HEART

Beginning with the invertebrates, the lowest members of the animal kingdom, the heart is a simple propulsive muscular tube. In the fish,

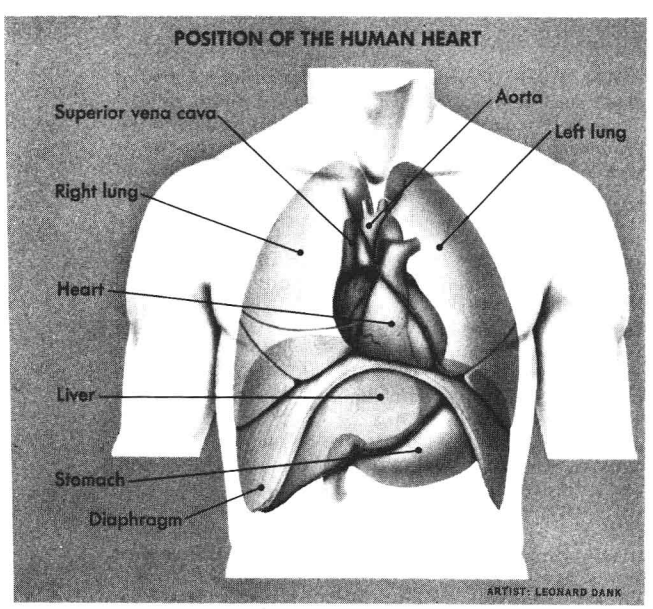
the heart has evolved into a chambered organ with an *atrium,* or receiving chamber, and a *ventricle,* a thick-walled pumping chamber. This development was necessary because the vertebrates, of which the fish are the lowest classes, have a more complicated structure and are generally larger than the invertebrates and require more oxygen. Thus, more energy is needed to pump the blood through the gills as well as through the rest of the body.

With the development of lungs in the higher vertebrates, the atrium became divided into two parts by the formation of a dividing wall called the *septum.* In the higher vertebrates, the right atrium receives the blood from most of the body, and the left atrium receives the blood from the lungs. In amphibians and reptiles there is still only one ventricle, except for the crocodile, which has two. Finally, in birds and mammals, the ventricle also became divided by a septum. The right side of the ventricle pumps blood exclusively to the lungs, while the left ventricle, which is much more muscular and has much more work to do, pumps blood to the rest of the body.

Thus, in man and other animals with a 4-chambered heart, blood from the body cells enters the heart at the right atrium, and from here it passes into the right ventricle, which lies directly below the right atrium. From the right ventricle the blood is pumped through the *pulmonary artery* toward the lungs, where carbon dioxide is released and oxygen is picked up. The oxygenated blood is carried back to the heart through the *pulmonary veins,* which empty into the left atrium. From the left atrium, the blood passes into the left ventricle, which then pumps it into the aorta for distribution throughout the body.

THE HUMAN HEART

The heart of an adult person is approximately the shape and size of the person's two fists fitted together so that the knuckles of one hand are against the knuckles of the other and the two wrists are touching. The bigger the person, the bigger the heart. Thus, there is a great variation in the size and weight of the human heart. The normal weight of the heart in both children and adults is approximately half of one percent, or a little less, of the total body weight. The professional athlete's heart is a little heavier, with slightly more muscle, but has only a slightly greater volume. Thus, a normal, nonobese man weighing 150 pounds (68.2 kg) may have a heart weighing up to ¾ of a pound (340 grams). This ratio holds more or less for all members of the mammalian order, except that a very athletic animal, such as a racing greyhound, may have a larger and heavier heart than a less active animal, such as a dog that is kept as a house pet. It has been estimated that the heart of a 100-ton (90-metric ton) and 100-foot (30-meter) long blue whale, the largest animal that has ever lived, weighs 1,000 pounds (450 kg). This ratio also applies to smaller whales, such as the grey whale, which weighs about 30 tons (27 metric tons) and has a 300-pound (135-kg) heart.



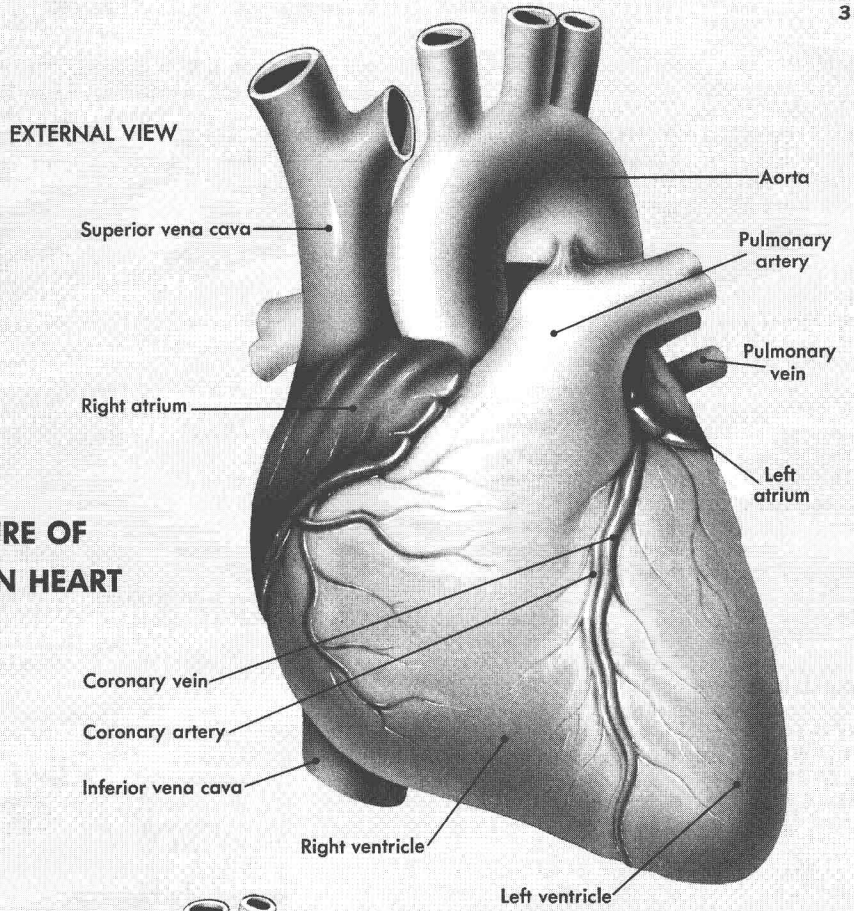
The Heart Rate. Just as the size and weight of the heart vary, so does the rate at which the heart beats. In some persons, especially trained athletes, the heart rate may be as low as 45 beats per minute when the person is at rest. Under the stress of vigorous exercise, the heart rate may reach as high as 170 or 180 beats per minute. The normal heart rate is considered to be about 72 beats per minute. At this average rate, the heart contracts 4,320 times in an hour, 103,680 times in a day, 58,579,200 times in a year, and 4,100,544,000 times in a lifetime of 70 years.

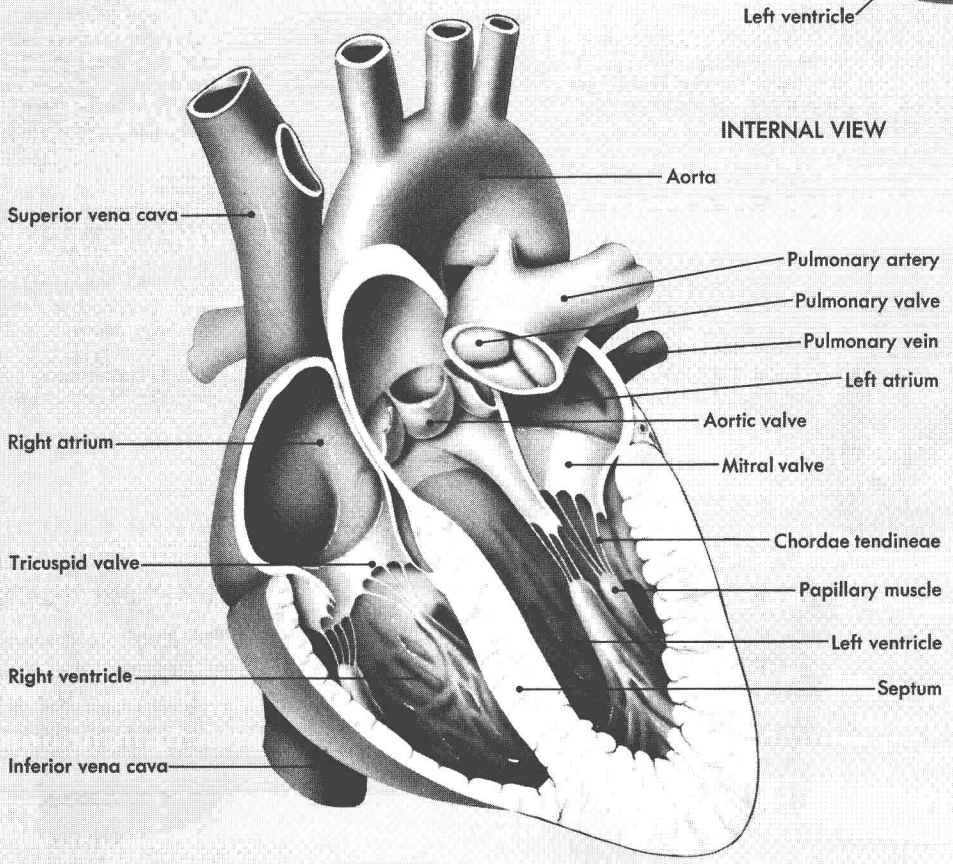
The small heart of a baby beats much more rapidly than that of an adult. At birth, the heart may beat up to 150 times a minute, but the heart rate settles down during the first year to about 100 times a minute. This relationship of heart rate to heart size applies to all mammals. For example, the heart rate of the shrew, the smallest mammal, has been clocked at nearly 1,000 beats per minute, while the heart rate of elephants at rest averages 30 beats a minute, and the rate of an excited small beluga whale beats about 15 times per minute. On the basis of these comparisons, the heart rate of the blue whale is probably about 5 or 6 beats a minute (although the actual heart rate is yet to be recorded).

Although it is true that nerves arising from ganglia outside the heart do have a function in quickening and slowing the heart rate, the heart can beat on its own by means of a natural *pacemaker,* as proved by both animal and human heart transplants. And undoubtedly, the heart's pacemaker can be stimulated by certain chemical substances, called *catecholamines,* which are secreted into the bloodstream by the adrenal glands and nerve endings for emergency action throughout the body.

The heart's pacemaker is located where the *superior vena cava,* the large vein carrying blood to the heart from the upper parts of the body, joins the right atrium. It is made up of a collection of muscle cells, blood vessels, and nerves and is technically known as the *sinoatrial node,* or the node of Keith and Flack, the two men who discovered it in 1907. Every second, or even more often, an electrical impulse originates in the pacemaker and travels down over the two atria in a wave of about $\frac{1}{10}$ of a millivolt, causing the atrial muscle to contract. The impulse then proceeds to another specialized node located at the fibrous junction of the two atria

STRUCTURE OF THE HUMAN HEART





ARTIST: LEONARD DANK

and the two ventricles. This node is called the *atrioventricular node of Tawara*, named for its discoverer. This node sparks the electrical stimulus that causes both ventricles to contract simultaneously. The electrical impulse is carried from the atrioventricular node by two special muscular branches that penetrate the muscle sheets of the ventricles.

The Stroke Volume. Each of the four chambers of an average-sized human heart holds about 2.5 ounces (80 cc) of blood; this is about the volume of blood pumped through and out of the heart with each heartbeat. This volume is called the *stroke volume*, and multiplying it by the heart rate, the *minute volume* is obtained. For example, if the heart rate is 60 beats a minute, the minute volume would equal 5 quarts (nearly 5 liters). During exercise, this figure can be greatly increased, even quadrupled, by two mechanisms, a doubling of the stroke volume and a doubling of the heart rate. Thus, it is possible, for at least short intervals, for the minute volume to increase from 5 quarts to 20 quarts (nearly 19 liters) of blood.

Assuming that during an hour at complete rest the average heart pumps 300 quarts (over 280 liters) of blood, in 24 hours this would amount to 1,800 gallons (7,200 liters). In a year, the heart would pump 657,000 gallons (2,688,000 liters) and in a lifetime of 70 years, nearly 46,000,000 gallons (more than 188,000,000 liters) of blood would be pumped. Since these figures are for a resting heart, they should be doubled for a normal person who is not always at rest.

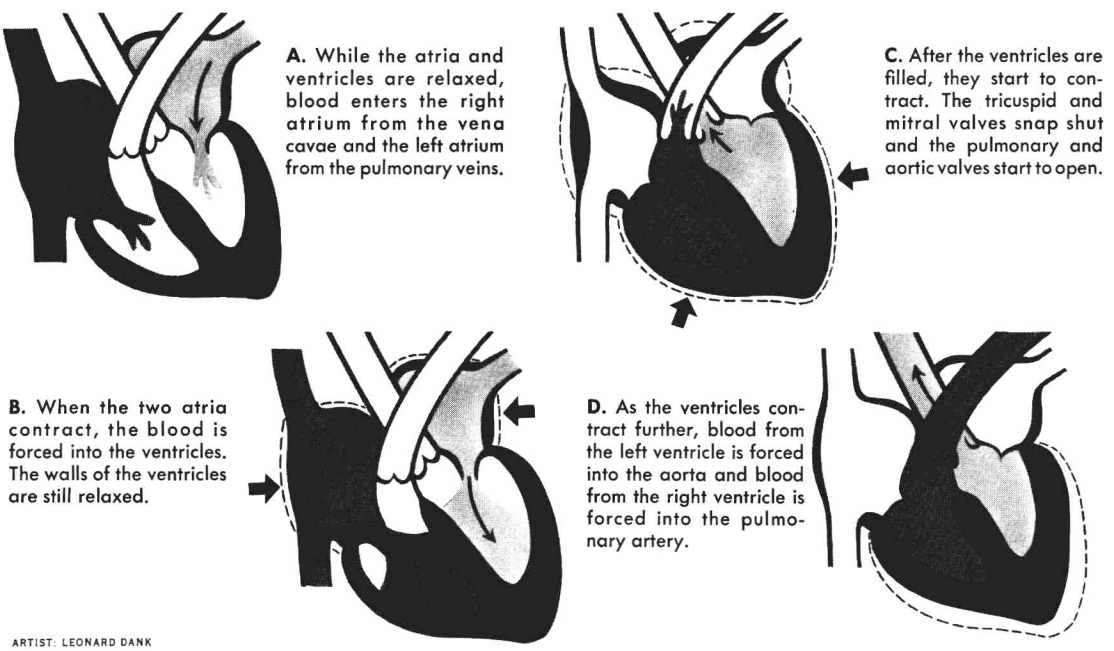
Structure of the Heart. The innermost lining of the heart wall is called the *endocardium*. It is a strong layer of fibrous tissue and endothelium over which the blood moves smoothly. When the endocardium becomes diseased or damaged the blood may clot on it, producing an intracardiac, or endocardial, *thrombosis* (clot).

In addition to coating the inside of the heart, the endocardium coats the four heart *valves*. Two valves are in the right heart chambers, and two are in the left. Each valve is made up of a thin strip of fibrous and elastic tissue and operates by opening and shutting with the heart beat. The valve on the right side that separates the right atrium from the right ventricle has three cusps, or curtains, and is called the *tricuspid valve*. It closes when the ventricle contracts, during a phase known as *systole*, and it opens when the ventricle relaxes, a phase known as *diastole*, allowing the blood to pour in from the right atrium.

The valve that separates the right ventricle from the pulmonary artery is called the *pulmonary valve*. It opens during systole, to allow the blood to be pumped toward the lungs, and it closes in diastole to keep the blood from leaking back into the heart from the pulmonary artery. The pulmonary valve has three cusps, each one resembling a crescent, and it is known as a *semilunar valve*. The pulmonary valve does not need any cords to keep the cusps in place, but the tricuspid valve does. These cords are called *chordae tendineae*, and they are attached to the *papillary muscle*, a muscular outgrowth inside the right ventricle.

In the left ventricle, the valve that separates the left atrium from the ventricle has only two cusps and is called the *mitral valve* because it is shaped like a bishop's mitre. It opens during diastole to allow the blood stored in the atrium to pour into the ventricle, and it closes during systole to prevent the blood from leaking back into the atrium. In order for the cusps to be taut during systole, they are equipped with chordae tendineae that are anchored in two papillary muscles. The fourth valve of the heart is called the *aortic valve* because it separates the left ventricle from the aorta. It has three semilunar cusps and closely resembles the pulmonary

PATH OF BLOOD FLOW THROUGH THE HEART



A. While the atria and ventricles are relaxed, blood enters the right atrium from the vena cavae and the left atrium from the pulmonary veins.

B. When the two atria contract, the blood is forced into the ventricles. The walls of the ventricles are still relaxed.

C. After the ventricles are filled, they start to contract. The tricuspid and mitral valves snap shut and the pulmonary and aortic valves start to open.

D. As the ventricles contract further, blood from the left ventricle is forced into the aorta and blood from the right ventricle is forced into the pulmonary artery.

ARTIST: LEONARD DANK

valve in structure. The aortic valve opens during systole to allow the jet stream of blood into the aorta, and it closes in diastole to prevent any of the blood from leaking back into the left ventricle.

With each heartbeat, or systole, the tricuspid and mitral valves close more or less simultaneously. Their closure produces a sound, which has been described as resembling the sound "lub." While the tricuspid and mitral valves are closing, the pulmonary and aortic valves are opening rather quietly, but when diastole begins the pulmonary and aortic valves snap shut while the tricuspid and mitral valves quietly open. The sound made by the closing pulmonary and aortic valves resembles the sound "dup." Thus the normal heartbeat heard through a stethoscope or even with the ear next to the chest sounds like "lub-dup," "lub-dup," "lub-dup." These sounds may be doubled on occasion if the contractions of the ventricles are not exactly simultaneous, a condition that has no important effect on the heart's functioning. Generally, at a heart rate of about 60 beats per minute systole lasts one third of a second and diastole lasts two thirds of a second.

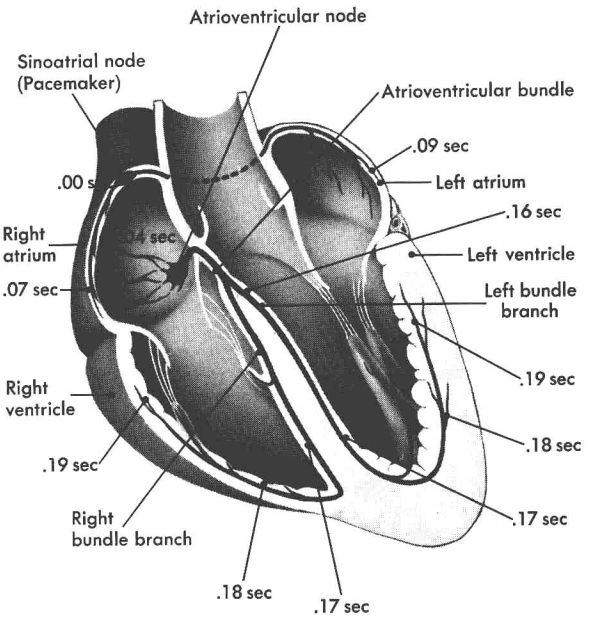
Whenever blood is allowed to leak through any of the four heart valves characteristic sounds, called *murmurs,* are produced. Murmurs vary considerably in timing, intensity, duration, and character. They may be blowing, humming, rasping, or whistling sounds. Sometimes a heart murmur indicates a disorder of the heart, such as a narrowing of the valves and the heart chambers. Some murmurs do not indicate heart disease and are found in normal hearts.

The middle layer of the heart wall, called the *myocardium,* is made up of thick heart muscle arranged in more or less concentric layers so that their full pressure is exerted on the two ventricles simultaneously. The thickness of the wall of the left ventricle averages slightly less than half an inch (10–12 mm) in the normal adult heart, and that of the right ventricle averages $^{12}/_{100}$ to $^{16}/_{100}$ of an inch (3–4 mm). The weight of the left ventricular muscle mass is a little less than twice the weight of the right, but in both ventricles the thickness tapers toward the apex at the bottom of the heart.

The outer layer of the heart is called the *pericardium,* and it forms a sac in which the heart lies. Although this sac affords some lubrication to the moving heart, it is not essential to life or even to good health. Occasionally, the pericardium becomes infected, a condition known as *pericarditis.* Sometimes in pericarditis, the two pericardial layers—the outer layer of the sac and the epicardium, the layer attached to the surface of the heart—become fused. This condition is known as *adhesive pericarditis,* and if the sac becomes greatly thickened and fibrous it can constrict the heart and seriously interfere with its functioning.

Coronary Circulation. The blood supply of the heart muscle itself is called the coronary circulation; this is because the two major arteries and their branches that descend over the outside surface of the heart from their origin at the base of the aorta are thought to resemble a corona, or crown. These two small arteries, called the right and left coronary arteries, arise from the aorta just above the aortic valve, their openings, or *ostia,* located in the outer edges of two of the valve cusps. In diastole, after blood has been

CONDUCTION PATHWAY THROUGH THE HEART



ARTIST: LEONARD DANK

pumped into the aorta, some of it is forced into the coronary arteries and thence through an elaborate network of smaller and smaller arterial branches and arterioles. Finally the blood enters the capillaries that supply each muscle cell. The left coronary artery is generally larger than the right one and quickly divides into circumflex and anterior descending branches.

The coronary circulation undergoes a great deal of stress and strain due to the constant bending and straightening of the arteries with each beat of the heart. This wear and tear of the coronary arteries, combined with their small size, provides the background for the disease atherosclerosis which, when complicated by thrombosis (clotting), is such a menace to many young and middle-aged men. Connecting the three chief coronary branches are tiny arterioles that become functional only if the chief arteries become narrowed. These tiny arterioles, under the pressure put on them, develop into a *collateral circulation,* which allows the blood to the muscle cells to bypass the blocked arteries. This collateral circulation has saved countless lives.

DISEASES OF THE HEART

Heart disease was not recognized until about 1500 A. D., when postmortem examinations were first allowed by the church. Prior to that time the heart was considered so delicate and sensitive that death was believed to be inevitable if the heart was injured. Gradually, postmortem examinations became more numerous, and in the 17th and 18th centuries structural defects of all kinds were discovered, including enlarged atria and ventricles, deformed valves, scarred heart muscle, and disorders of the endocardium and pericardium. There was also much interest concerning blood clots in the chambers and blood vessels of the heart, but it was not known whether they had occurred before death or after

death. It was finally the German pathologist Rudolf Virchow who, in the middle of the 19th century, solved most of the riddles concerning thrombi (clots) and emboli (clots that travel through the circulatory system and block arteries in the lungs, brain, or other organs).

The structural diagnosis of heart disease, both clinical and pathological, held the limelight until the time of Sir James Mackenzie who, at the end of the 19th century and during the beginning of the 20th century, insisted on the addition of another category to the diagnosis of heart disease, namely that of function. This category included degrees of competency, or sufficiency, of the heart muscle to do the work required of it, and of the coronary circulation to supply the heart muscle with enough blood and oxygen to carry on its work. Among the disorders included in Mackenzie's category were congestive heart failure, which occurs when the heart muscle does not function sufficiently; and coronary insufficiency, which occurs when the coronary arteries are obstructed by atherosclerotic thickening of their walls or by blood clots. Also included under the heading of disorders of function are the various cardiac arrhythmias, or irregular heartbeating.

In 1914 there appeared in the *Journal of the American Medical Association* under the authorship of Richard C. Cabot of Harvard University an important article entitled "The Four Common Types of Heart Disease." In this article the author introduced the idea, now quite obvious and widely accepted, that understanding the causes of heart disease is much more vital than understanding either the actual structural defects or the functional conditions. He maintained that the causes of heart disease deserve first priority since the prevention of heart disease, the ultimate goal, depends upon determining the causes. In a follow-up to this article, the American cardiologist Paul Dudley White published the first edition of his textbook *Heart Disease* (1931), emphasizing the etiological, or causative, diagnosis first, followed by structural and functional diagnoses in that order.

Although most of the causes of heart disease observed in the first quarter of the 20th century are still present, their relative proportions have changed considerably, and some causes that were mentioned only occasionally or not at all at that time are considered common today. This is especially true of the virus infections. In some parts of the world the diagnosis of "heart disease of unknown origin" is a common one. In the United States such a diagnosis exists but is uncommon, and a few kinds of heart disease that are common in certain other countries are rare or nonexistent in the United States. For example, Chagas' disease (myocardial trypanosomiasis) is a parasitic infestation of the heart common in South America.

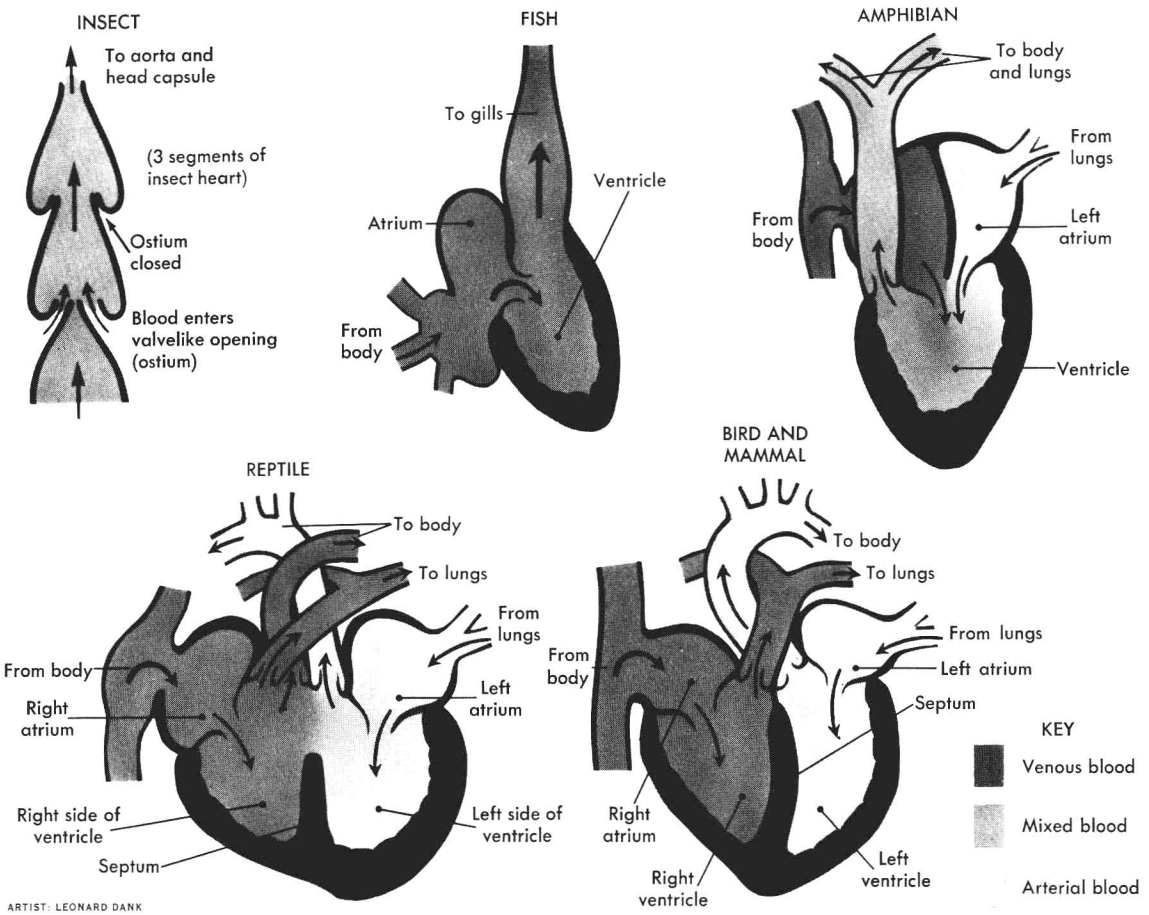
Infectious Types of Heart Disease. During the early 1900's infectious types of heart disease were the greatest scourge, making up much more than half of all the cardiac cases. Today they account for less than a fourth of all cases in the United States and other countries that are equally prosperous and equally able to control infectious diseases. The two major infectious heart diseases in the United States are rheumatic heart disease and syphilitic heart disease.

Rheumatic Heart Disease. This disease occurs as a complication of rheumatic fever, and it is still somewhat of a menace; but wherever the causative bacterium, hemolytic streptococcus, is well controlled by penicillin, hygienic measures, or both, rheumatic fever is being wiped out. Already, it has dropped to third or even fourth place from its position of first place in the early 1900's. At that time there were long waiting lists for hospital beds for acute rheumatic patients, mostly children, and as the need for hospital beds for tubercular patients decreased in the 1920's and 1930's, their beds were occupied by rheumatic fever patients who were often very ill. With improvement in living conditions and the discovery of penicillin and other antibiotic drugs in the 1940's, rheumatic heart disease began a steady decline.

Although the hemolytic streptococcus bacterium does not itself invade the heart, its toxic reaction can seriously damage all parts of the heart. Involvement of the myocardium, especially in the acute stage, may lead to heart muscle failure and death. Involvement of the pericardium may cause pericarditis and effusion (collection of fluid) in the pericardial sac. Often, the endocardium is affected, producing endocarditis. This condition leads to scarring and deformity of the heart valves, especially the mitral and aortic, resulting in stenosis (narrowing) or regurgitation (leaking).

The most common chronic scarring effects are those of mitral stenosis and aortic regurgitation. *Mitral stenosis,* when acute, leads to enlargement of the left atrium, congestion of the lung circulation, and enlargement of the right ventricle. *Aortic regurgitation,* when pronounced, leads to left ventricular enlargement and failure. A common complication of mitral stenosis is atrial fibrillation, a rapid irregular heart rate often with edema of the lungs. Both mitral and aortic valves may be deformed in chronic rheumatic heart disease in youth and middle age, and thus the combination of mitral stenosis and aortic regurgitation is quite common. Rheumatic *mitral regurgitation* frequently occurs with mitral stenosis but can also occur alone. *Aortic stenosis* often accompanies aortic regurgitation, but it too may also occur alone. Aortic stenosis is generally less serious than regurgitation and is not always rheumatic in origin; it may be a congenital defect, or it may develop in older people as a result of arteriosclerosis. Damage from rheumatic fever to the valves of the right side of the heart—that is, the tricuspid and pulmonary valves—is uncommon, at least to any important degree. Fortunately, in many cases the lesions are so slight that they do not shorten the patient's lifespan. However, protection with penicillin against subacute bacterial endocarditis is always important for such individuals who are candidates for heart complications throughout their lives, especially when exposed to infection through extensive dental repairs or extraction.

Since the late 1940's cardiac surgery has advanced to such a degree that it is now possible to replace seriously damaged mitral, aortic, and even tricuspid valves by artificial valves or valves taken from animals or other people. The early operations consisted of a relatively simple splitting of the stenosed mitral valve by the surgeon inserting his finger through it. These operations led to much more complex operations, made possible by the use of the pump oxygenator and open-heart surgery, with the heart at a complete standstill for as many minutes as may be



necessary to replace one, two, or even three valves.

Syphilitic Heart Disease. Although the incidence of syphilis involving the ascending aorta causing an aneurysm (bulging out of the vessel wall due to a weakening of the inner lining) or a serious leak of the aortic valve has decreased greatly since the 1920's, there has been an increase of the primary lesion, with a threat of an increase of the tertiary stage, the stage that does involve the aorta. It is extremely important, therefore, to diagnose and treat the onset of syphilis with its very effective antidote, penicillin.

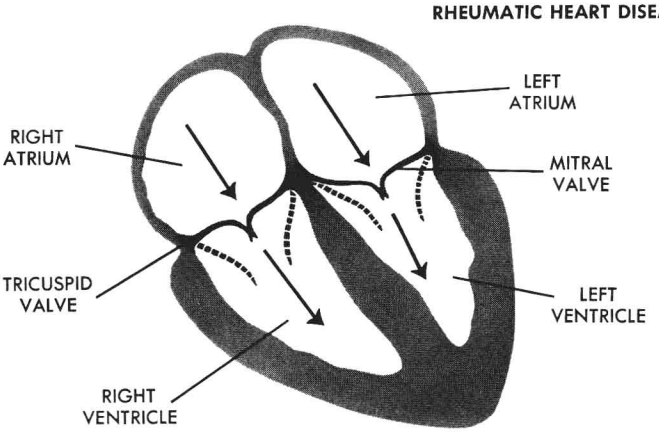
Thyrotoxic Heart Disease. Thyrotoxicosis is a condition caused by oversecretion by an enlarged thyroid gland; it sometimes affects the functioning of the heart. Exophthalmic goiter causing thyrotoxicosis was responsible for 3% of the 3,000 cardiovascular cases analyzed in New England by T. Duckett Jones and Paul Dudley White in the 1920's. That was before any cure for the disease was known. Later, when surgical operations and drug therapy, especially the use of radioactive iodine, were introduced, thyrotoxicosis was quickly treated and cured. Thus, this kind of heart disease has been practically eradicated, although rare cases are still encountered in elderly people. Other endocrine disorders affecting the heart are very rare.

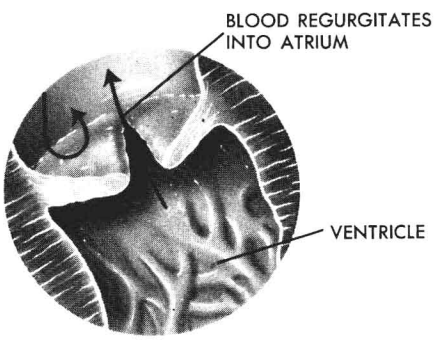
Congenital Heart Disease. With the increasing control of rheumatic heart disease it is probable that congenital cardiovascular disease will soon outstrip it in incidence, and with the increasing control of high blood pressure, congenital heart disease will take second place. An interesting statistical fact is that in the 1920's, T. Duckett Jones and Paul Dudley White found that congenital heart disease made up only 1.5% of all of 3,000 patients with signs or symptoms of heart disease. Thirty years later this figure had risen to 8%. This increase was due largely to improved diagnosis with the aid of the electrocardiograph, the X-ray camera, cardiac catheterization, and more complete history and physical examinations. It was also due to the fact that more patients, mostly babies and young children, were being brought to medical centers because it had become possible to correct or at least improve many of the defects by surgical operations.

There are two factors causing congenital defects: genetic defects and disorders that affect the mother during pregnancy—that is, during the fetal life of the infant. Their relative responsibility has not yet been ascertained, and further research on these two causes is essential before preventive measures can be devised. So far, surgical correction is the only method for treating congenital defects.

The most common of the congenital defects of the heart and great vessels found after infancy are septal defects, patency of the ductus arteriosus, coarctation of the aorta, and transposition of the great arteries of the heart. Aortic and pulmonary valve stenosis, which have been discussed earlier under rheumatic heart disease, are also sometimes caused by congenital defects.

RHEUMATIC HEART DISEASE





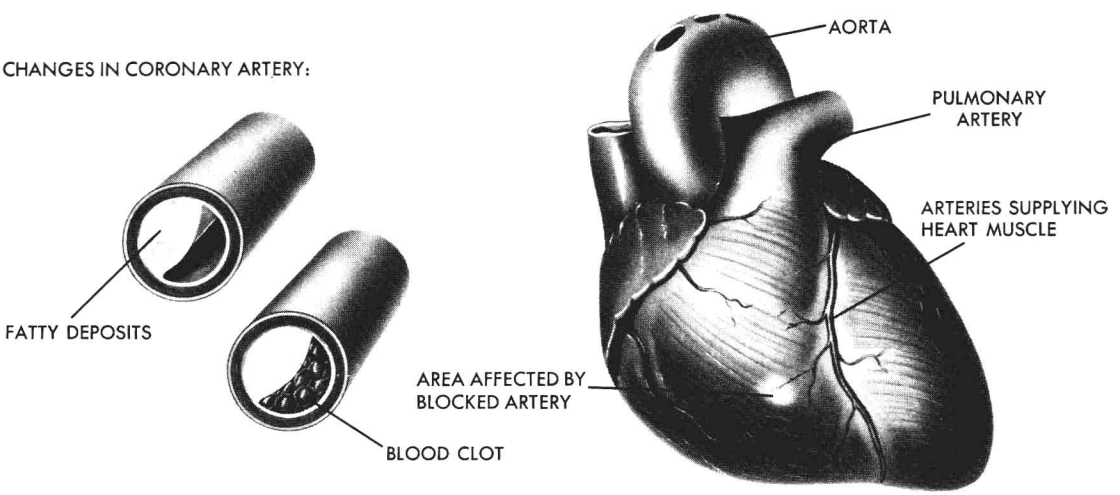
THE NORMAL HEART

In the normal heart, the valves between the atria and ventricles open to permit blood to flow through and close when the ventricles contract, thus preventing the blood from flowing back into the atria.

THE RHEUMATIC HEART

In rheumatic heart disease, the valves may not close tightly enough to keep the blood from leaking back into the atria. The aortic valve, between the aorta and left ventricle, may also be affected.

CORONARY HEART DISEASE



This disorder is caused by atherosclerosis of the coronary arteries. In atherosclerosis, deposits of fatty materials line the walls of the arteries, partly obstructing the flow of blood. If a blood clot lodges in an artery and blocks it completely, the tissue that is normally supplied by the artery dies, causing a severe, sometimes fatal, malfunctioning of the heart.

Septal Defects. Ventricular septal defects, when isolated and small, cause little harm and allow a long life if there is adequate protection against bacterial infection of the defect. When complicated by a high degree of right ventricular infundibular or pulmonary valve stenosis, it gives rise to the *morbus ceruleus*, or *blue baby*. The blue coloration of the baby's skin is due to a lack of oxygen resulting from much of the blood bypassing the lungs. Instead of traveling to the lungs from the right ventricle, the blood passes through the septal opening directly into the left side of the heart, from where it is pumped into the aorta. This condition requires surgical correction if the child is to survive.

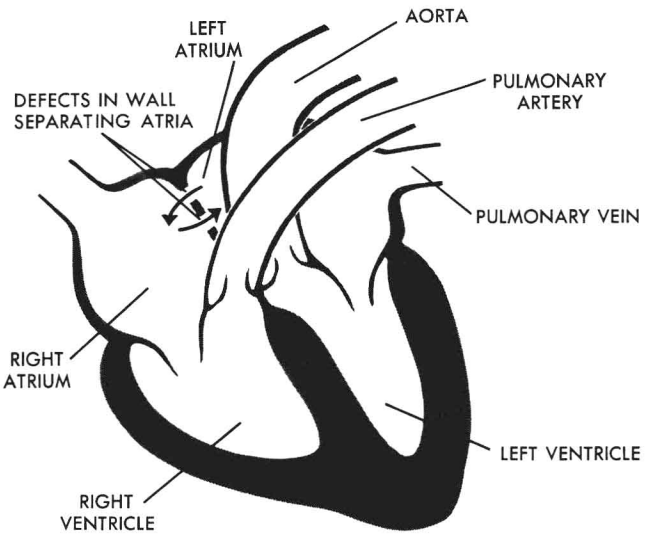
Atrial septal defects, when isolated, may allow the patient to live an active healthy life to middle age despite much enlargement of the right ventricle. However, it is best to correct the defect surgically during either childhood or adolescence.

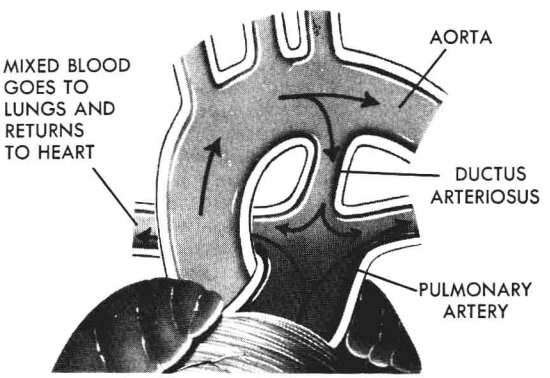
Patency of the Ductus Arteriosus. The ductus arteriosus is a tube which, during fetal life, allows blood to bypass the nonfunctional lungs and travel from the right ventricle directly into the aorta. Normally, the ductus arteriosus closes shortly after birth, but sometimes it persists and remains patent, or open. If the ductus arteriosus is large, it may cause heart failure by aortic regurgitation into the pulmonary artery. If it is small, the patient may lead a normal life. Most often, a persistent ductus arteriosus should be surgically treated to relieve the strain on the heart and prevent a bacterial infection of the ductus.

CONGENITAL HEART DEFECTS

SEPTAL DEFECTS

Openings in the septum, the wall that divides the heart into right and left sides, are among the most common congenital heart defects. Shown here are openings in the septum between the left and right atria, but they may also occur between the two ventricles. When atrial or ventricular openings are fairly large, the blood may travel directly from the right side of the heart to the left, bypassing the lungs and depriving the body tissues of oxygen. As a result, the infant's skin may turn blue, producing the so-called blue baby. In such cases, immediate surgery is necessary to save the child. In cases where the openings are small, the child can lead a fairly normal life but the openings should be surgically closed sometime during childhood or adolescence.



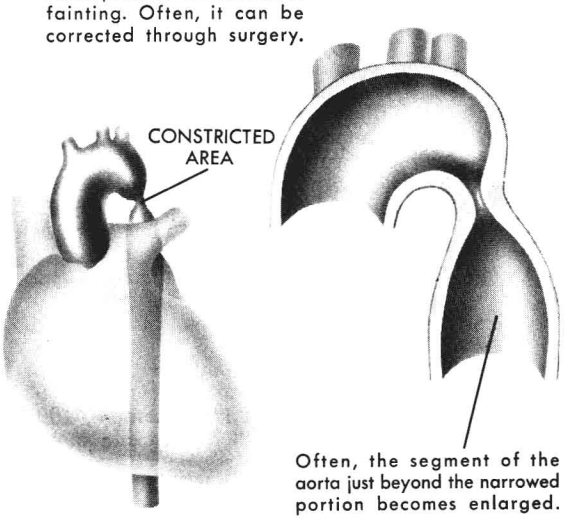


PATENCY OF THE DUCTUS ARTERIOSUS

The ductus arteriosus is a blood vessel which, during fetal life, allows blood to pass from the pulmonary artery directly to the aorta, bypassing the nonfunctioning lungs. Normally, the ductus arteriosus closes off shortly after birth, but sometimes it remains open, or patent. When this happens, blood from the aorta may flow back into the pulmonary artery and be recircuited to the lungs and back through the heart. Most often, this defect can be corrected through surgery.

COARCTATION OF THE AORTA

This defect is an abnormal narrowing, or constriction, of the aorta. It may cause high blood pressure and attacks of fainting. Often, it can be corrected through surgery.

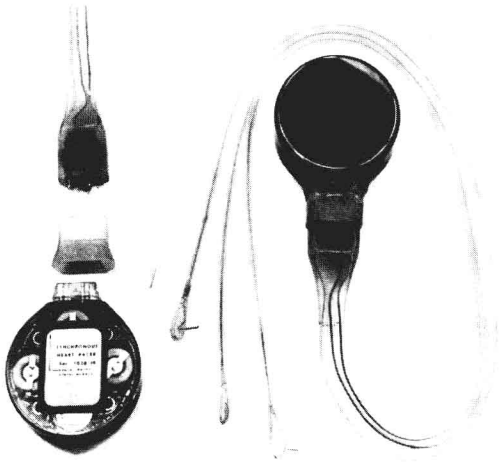


Coarctation of the Aorta. This condition is a malformation characterized by a severe narrowing of the aorta. It is a cause of hypertension (high blood pressure) in children and should be corrected through surgery.

Transposition of the Great Arteries. In this condition the two major arteries from the heart, the aorta and the pulmonary artery, are reversed; that is, the pulmonary artery arises from the left side of the heart and aorta from the right side. This very serious defect is most common in the earliest months of life. Sometimes it can be corrected through surgery, but rarely do the infants survive.

Hypertensive Heart Disease. Hypertension (high blood pressure) and hypertensive heart disease were common in the decades before the 1950's, ranking second in incidence to infectious types of heart disease. Although these diseases have not decreased in incidence, their mortality rates have been strikingly decreased as a result of the introduction of drug therapy for hypertension. In the 1940's, before medicines for lowering blood pressure had become widespread, there were two methods for treating severe hypertension. One method was an operation in which certain nerves were cut (thoracolumbar sympathectomy); the other was a rigid and very unpleasant low-sodium diet.

Sometimes, hypertension can be traced to a known cause, such as chronic glomerular nephritis, a defective arterial supply to one or both kidneys, disease of the pituitary gland, pheochromocytoma (a type of tumor of the adrenal



NATIONAL HEART INSTITUTE

ARTIFICIAL PACEMAKER can be implanted on the heart to provide long-term correction of complete heart block.

gland), or a congenital coarctation of the aorta. However, the most common cause of hypertension, called essential, or idiopathic, is still of unknown origin and is the subject of continued research. It must be noted, however, that many people who have an elevated blood pressure (for example, over 150 mm of mercury systolic pressure and over 90 mm of mercury diastolic pressure) at the time of an examination are not truly hypertensive. Such people are called *hyperreactors*. Under the stress of an examination, especially by a physician new to the patient, both blood pressure levels, particularly the systolic, are elevated; but with time and relaxation they will usually drop to normal. Such a person has an elevated blood pressure only a few times a day for a few minutes at a time and has normal blood pressure most of the time. Such hyperreaction does little or no harm to the heart and arteries and should not be confused with or treated as hypertension. However, it may lead to hypertension.

High blood pressure, if persistent much of the time over many years, and especially hypertension of the diastolic level (for example, 190/125), eventually causes the left ventricle to hypertrophy (enlarge) and unless it is adequately treated, the left ventricle may fail in the course of time. If the hypertension is severe enough and sustained long enough, no complications are needed to produce heart failure. Very often, however, as the patient grows older, atherosclerosis of the coronary arteries, the aorta, and the carotid arteries in the neck complicates the hypertension and is accelerated by it. In fact, the combination of hypertensive and coronary heart diseases is very common.

Just as generalized, or systemic, hypertension can in time cause enlargement and failure of the left ventricle, pulmonary hypertension can cause enlargement and failure of the right ventricle. Pulmonary hypertension is most commonly caused by congestion in the lung circulation due either to the back pressure of a failing left ventricle or to mitral valve stenosis. A somewhat less common cause of pulmonary hypertension is an obstruction in the pulmonary circulation that has nothing to do with the left ventricle. This condition is known as *cor pulmonale*, and it is most often caused by blood clots from veins in the legs. Such clots may in time block over half of the pulmonary arterial tree. A second known cause of cor pulmonale is very extensive scarring of the lungs from an infectious disease. A third cause is silicosis, which is due to the inhalation of silica dust. Silicosis was once common among miners and certain industrial workers but has decreased greatly as a result of preventive measures. Some cases of cor pulmonale are due to unknown causes.

Coronary Heart Disease. Coronary heart disease is responsible for about 55% of all the deaths due to heart disease in the United States. It is caused by atherosclerosis of the coronary arteries, the small but vital blood vessels that supply the heart muscle with blood and oxygen. Atherosclerosis is the most common and most important kind of arteriosclerosis. The term "atherosclerosis" is derived from two Greek words, *athero*, meaning soft material or pudding, and *sclerosis*, meaning hardening. Both of these processes involve the inner lining, or *endothelium*, of the large and medium-sized arteries throughout the body. It should be noted that young and middle-aged men are generally more prone to atherosclerosis than women. This is because women, until they reach menopause, are relatively well protected by female hormones.

Although atherosclerosis of the aortic wall comes somewhat earlier and is often more extensive than that of the coronary arteries, the much smaller coronary arteries are more easily reduced in size and can be easily blocked by the collection of fat and fibrous tissue cells deposited on their inner lining—as occurs in atherosclerosis. This blockage results in *ischemia* (lack of adequate blood supply) of the heart muscle, which may cause *angina pectoris* (oppressive pain across the front of the chest) for a few minutes when the person is excited or physically active. This pain can be relieved quickly or prevented by nitroglycerin tablets that are easily dissolved under the tongue. The angina is a warning to the patient to stop the exertion because of the possibility of a cardiac standstill or ventricular fibrillation, a fatal irregularity of the heart rate. If angina pectoris occurs when the person is resting, or if it awakens him at night, the condition demands treatment by quiet rest at home or in a hospital and by other measures, such as sedatives, long-acting nitrites, and sometimes anticoagulants. Some patients with angina pectoris may live for years and may completely recover in the course of months or years, owing to the development of the heart's collateral circulation.

In addition to the symptom of angina pectoris, further evidence of myocardial ischemia from coronary artery obstruction usually appears in an electrocardiogram (ECG or EKG), a graph on which the electrical impulses of the heart muscle are recorded. Sometimes, the EKG indicates definite abnormalities even though the heart may seem perfectly normal in a physical examination or in X-rays.

The next degree of involvement by atherosclerosis of the coronary arteries is usually due to the complication of a blood clot that completely obstructs one of the main arterial branches, causing a scar, or *infarct*, in the heart muscle. Such a scar, like a broken bone, takes 3 or 4 weeks to heal; during this time the patient should

be at almost complete rest, preferably in a hospital and, if considered necessary by the doctor, in an intensive care unit for the first few days. The speed of recovery and extent of rehabilitation depend largely on the size of the scar, the age of the patient, and various complications, especially an early state of shock, congestive heart failure, serious arrhythmias, or rupture of the heart wall. The prognosis of the patient varies according to these factors. Death may come almost at once, in a week, a month, or a year, but the patient may live an ordinarily active life for many years after recovering.

Since coronary heart disease has become a leading cause of death in young and middle-aged men in the United States and since there is no really effective cure for it, either surgical or medical, it is essential that the causes of the disease be appraised so that preventive measures can be devised. Studies show that several factors are involved in coronary heart disease, with no single cause being entirely responsible, but the degree of responsibility of each of the many causes is not known. Among the probable causative factors now under investigation are heredity, diet (especially the intake of animal fat), obesity, the lack of physical exercise, stress, and the use of tobacco and coffee. Medical science will have reached a major goal when it can discover the role of these and other factors and develop the methods necessary for preventing coronary heart disease.

HEART SURGERY

For centuries, the heart, like the brain, was considered to be territory forbidden to surgeons, and it was not until after World War II that cardiovascular surgery emerged as a valuable method of treating heart disease. Prior to World War II there were only a few cases in which surgical procedures succeeded in curing or partially correcting diseased hearts. In 1925, Henry S. Souttar in England successfully split a mitral valve in a patient with mitral valve stenosis (narrowing), but this operation was not repeated or successfully duplicated by other surgeons. In 1928, Edward D. Churchill in Boston cured a serious case of chronic constrictive pericarditis, and in 1938, Robert E. Gross, also in Boston, performed the first successful operation in which a patent (open) ductus arteriosus was ligated (tied off).

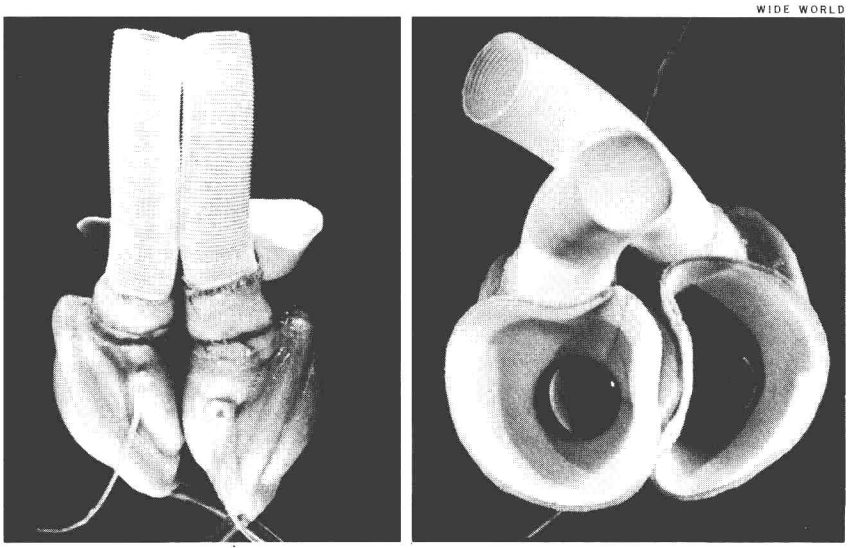
It was the experience of American surgeons working with thousands of chest wounds during World War II that provided the evidence that heart surgery was feasible. One of the first heart operations performed after the war was the opening of a narrowed mitral valve. In this operation, which was pioneered by Dwight Harkin and Charles Bailey in the late 1940's, the surgeon's finger was inserted into the valve through the left atrium. This relatively simple procedure was more or less successful in treating pulmonary edema and congestion in thousands of patients throughout the world during the next decade. Other valve deformities, however, were then still unapproachable.

Also during the 1940's and through the 1950's, congenital defects of the heart and blood vessels became subject to surgical treatment. Gross' method of closing off a patent ductus arteriosus became an almost routine and safe operation, and it is still widely used today. This operation is performed for two basic reasons: it spares the patient's overworked heart, and it prevents a bacterial infection of the ductus arteriosus, which is always a threat in untreated patients.

Perhaps the most severe congenital defect that has been in the limelight since the 1940's is the tetralogy of Fallot, a combination of four heart defects that is responsible for most blue babies. In 1945, Alfred Blalock of Baltimore, with the advice and help of Helen Taussig, introduced the first operation to enable blue babies to survive. The following year, Willis J. Potts of Chicago introduced a different surgical technique to achieve the same end, but it was not until the advent of open-heart surgery in the 1950's that it was possible for this complicated anomaly to be completely corrected.

During the time that heart surgery was being developed and perfected, there was also considerable progress in the correction of abnormalities of the arteries and veins. Various techniques were developed to treat varicose veins, and sometimes even the large veins leading to the heart—the inferior and superior venae cavae—were objects of surgical attention. Operations on the larger ar-

WIDE WORLD



MECHANICAL HEART, a double pump designed to replace the entire human heart, was developed at Texas Medical Center in Houston. The open tubes shown in the front view (*right*) and rear view (*left*) connect to the circulatory system.

teries, however, constituted a more dramatic chapter in the history of cardiovascular surgery. Aneurysms (bulging enlargements) of the aorta excited much interest, and efforts, sometimes successful, were made to prevent aneurysms from bursting. The technique used in the early 1900's involved the insertion of many feet of fine wire into the artery to produce a blood clot in the bulging sac so that the aneurysm could not burst. In the 1940's, Clarence Crafoord of Stockholm and Robert Gross independently pioneered the operation that has now become routine for curing congenital coarctation (narrowing) of the aorta, generally by removing a greatly narrowed portion and inserting a piece of artificial tubing or a piece of a large artery or vein from a blood vessel bank. During the 1960's, often with the help of a pump oxygenator, or heart-lung machine, many operations were performed to repair or replace large sections of the aorta and its major branches.

The introduction of the pump oxygenator in 1953 by John H. Gibbon of Philadelphia was the next dramatic step in the development of cardiovascular surgery. Pump oxygenators have slowly improved since then, and today they are used widely in open-heart surgery and other extensive cardiovascular operations. In a pump oxygenator, venous blood from the body is oxygenated and then pumped into the patient's arterial system so that the body cells, including the cells of the heart muscle, are not deprived of oxygen even though the heart has stopped beating. With this instrument, the heart valves, especially the aortic valve, can be repaired or replaced while the heart is standing still. Following an operation, which may take as long as three hours, the heart is revived with electric shock.

Probably the greatest achievement in heart surgery during the 1960's was the introduction of surgical techniques to transplant the human heart. The first operation of this type was performed by Christiaan Barnard of South Africa in 1967, and this operation was quickly followed by a score of similar operations in the United States and elsewhere. Many of the recipients died within hours, days, weeks, or months following the operation, but others survived. (See also TRANSPLANTS, TISSUE AND ORGAN.)

The procedure of heart transplantation is still in its experimental stage, but it is certain to have a definite place in the future after several problems, including the rejection phenomenon and the establishment of heart banks, have been overcome. It is to be hoped, however, that extreme measures such as heart transplants will not be necessary if medical science achieves its ultimate goal—the prevention of heart disease in people under the age of 80.

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HEART ATTACK is the popular term for an acute failure of the pumping action of the heart, accompanied by lung congestion and the accumulation of fluid in the dependent parts of the body. The symptoms of a heart attack include coughing, copious frothy spittle, discomfort when lying down, labored breathing, rapid heart action, blueness of the skin and lips, swelling of the legs, and fatigue. Heart attacks are a leading cause of death, particularly among men, and more than 500,000 people in the United States die from heart attacks every year.

Causes. A major cause of heart failure is coronary thrombosis. In this disorder, a blood clot in one of the small coronary arteries obstructs the delivery of blood to a portion of the heart. If the affected muscle segment dies, the heart may no longer be able to function properly. Other leading causes of heart attacks include severe hypertension (excessively high blood pressure) and obstructions or leaks in one or more of the heart valves. Heart failure may occur within a few minutes after a coronary thrombosis or may develop slowly over several weeks as when a valve is obstructed.

Pathology. No matter what the underlying cause of heart failure may be, the heart's main pumping chamber, the left ventricle, becomes overburdened and cannot pump away the volume of blood that returns to it. As a result, blood becomes dammed back in the left atrium and in the veins that drain the lungs, and as the pressure rises in the lung blood vessels, the distended lung capillaries protrude into the air spaces and become covered with fluid seeping out of them, thereby diminishing the uptake of oxygen by the blood. The excess fluid in the lungs causes coughing, air hunger, and restlessness. Fluid may also accumulate in the chest cavity, sometimes causing a lung to collapse.

Once the lungs are congested with blood, the blood pressure in the lung arteries rises, overloading the right ventricle and sometimes causing it to fail. The pressure then rises in the large veins that empty into the right atrium. This elevated venous pressure increases the pressure in the capillaries throughout the body, and fluid seeps out of them, especially into the dependent parts of the body. When the patient is lying down, the fluid accumulates in the area of the sacrum; when he is standing, it accumulates in the legs. As much as 10 liters (over 10 quarts) excess of fluid may accumulate in the body. When the patient goes to bed, much of this fluid is returned to the blood and is transferred to the lungs. Within an hour after going to bed, the lung congestion causes labored breathing and fits of coughing, which often awaken the patient. If he gets out of bed and walks around, he experiences relief because the fluid becomes redistributed to his legs.

Treatment. Heart failure is treated with bed rest, usually in a sitting position, removal of excess blood from the circulation (by bleeding or by temporary tourniquets at the extremities), and the administration of oxygen, morphine, and heart stimulants. Fluid loss is encouraged by drugs that increase urine output and by a low sodium diet that reduces the body's tendency to retain fluids. After acute symptoms are relieved, the specific cause of the attack is diagnosed and, if possible, treated.

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City of Hope Medical Center, Duarte, Calif.

HEART MURMUR is a rumbling noise produced by the heart in addition to the normal sounds of the heartbeat. Heart murmurs usually can be heard only through a stethoscope, but they may be recorded on paper by means of an instrument known as a phonocardiograph. Although some murmurs result from heart disorders, others occur normally and do not indicate disease.

In many cases, heart murmurs occur when the blood flowing through a heart valve flows too fast because the valve is too narrow. The high velocity of the blood causes the valve leaflets to flap like flags waving in the breeze. Some heart murmurs occur when a valve does not close tightly enough, allowing blood to leak back into the chamber from which it was pumped. Heart murmurs also occur in certain congenital heart disorders, such as when there is an opening in the wall between the heart's right and left sides or when the vessel between the aorta and pulmonary artery remains open after birth. In children, heart murmurs are often not a sign of heart disease but a phase of rapid heart growth. Such murmurs usually disappear in later life.

Heart murmurs may be associated with either the pumping phase (systole) of the heart or the phase of heart muscle relaxation (diastole). Systolic murmurs appear when the aortic and pulmonary valves are too small or when either the tricuspid or mitral valve allows blood to flow backward. Diastolic murmurs occur when the tricuspid or mitral valve is so narrow that the blood flow is impeded.

SIMON RODBARD, M. D.
City of Hope Medical Center, Duarte, Calif.

HEART OF DARKNESS, a short novel by Joseph Conrad (q.v.), published in 1902. One of Conrad's best works, the story reflects his experiences during a riverboat voyage in the Belgian Congo.

Marlow, the narrator, reminiscing about his adventures in the Congo as a boat captain for a concern that traded in ivory, remembers the terrible treatment of the native people by the self-seeking company agents. He recalls rumors that were spread about one agent, Mr. Kurtz, once a reputedly kind and idealistic man, who by some mysterious means was able to obtain more ivory than anyone else. Intrigued, Marlow set out to find Kurtz in the jungle and discovered him to be morally corrupted by greed for power and dying of tropical disease. The story's title symbolizes the jungle and also the dark and bestial side of man's nature. T. S. Eliot's epigram for his poem *The Hollow Men,* "Mistah Kurtz, he dead," is taken from this story.

HEART OF MIDLOTHIAN, The, mid-lō′thē-ən, a novel by the Scottish author Sir Walter Scott, published in 1818. It is regarded as one of his best works.

The story is set in Scotland during the reign of George II. The title refers to the Edinburgh prison where some of the most spectacular action occurs. The heart of the plot is the dilemma of an uneducated peasant girl, Jeanie Deans, daughter of an old "true-blue Presbyterian." Her sister, Effie, is charged with infanticide under an inhumane law. Jeanie's conscience forbids her to save her sister from execution by perjuring herself, so she sets out on a heroic walk to London to secure a pardon for Effie. She succeeds through her innocence and frankness and through the aid of the Duke of Argyle, who arranges a dramatic meeting between her and Queen Caroline.

The tale is shapeless and uneven, with romantic and Gothic trappings, especially in figures like mad Madge Wildfire and the picturesque George Robertson, Effie's seducer. Undistinguished in the narrative passages, the novel leaps to life in the dialogue, in the Scots dialect. Scott brings history alive not through description but through Jeanie and her father, their moral struggles and the dramatic situations they produce.

INGA-STINA EWBANK, *University of Liverpool*

HEARTBREAK HOUSE is a play in three acts by George Bernard Shaw (q.v.), who regarded it as his best work. Written at intervals between 1913 and 1919, it was published in 1919 and first performed in 1920 in New York. The scene is a room, built to resemble an old-fashioned ship's cabin, in a country house in northern Sussex, and the main characters are Captain Shotover, his two daughters, and various weekend guests.

There is little action in the play, but the dialogue is sophisticated and the characterizations are shrewd. In it Shaw gives his views on international politics, interweaving typical Shavian witticisms with some of the profoundest passages in his works. Patterned after Chekhov, *Heartbreak House* foresees the breakdown of pre-World War I European society. The house of the title is a symbol of England.

HEARTBURN is a burning sensation under the breastbone (sternum) and the upper portion of the abdomen. It usually occurs after eating and may be associated with nausea, cramps, and other symptoms of indigestion.

Causes. In many cases, heartburn is caused by minor digestive disorders, such as gastritis, or by spasms of the muscle at the upper end of the stomach. The burning sensation is due to a backup of the acidic contents of the stomach into the esophagus, or gullet.

Sometimes heartburn is an early symptom of a more serious disorder of the digestive system. It may be a sign of a hiatal hernia, which is a translocation of a portion of the stomach into the chest cavity. When a hiatal hernia is the cause, the heartburn is felt mostly after the person has eaten a heavy meal and is lying down. Other disorders that may cause heartburn include esophagitis (inflammation of the esophagus), peptic ulcer, and diseases of the gall bladder and pancreas.

Because heartburn may also be caused by heart disease, it is important that a person suffering severe heartburn have a complete medical checkup. All too often, the pain caused by angina pectoris and even a heart attack is dismissed by the patient as indigestion.

Treatment. Heartburn caused by gastritis or spasms of the stomach can be relieved by antacids or antispasmodic drugs. If caused by a hiatal hernia, heartburn can sometimes be controlled by having the patient refrain from overeating or eat more slowly and remain in an upright position after eating. In severe cases, the hernia may be treated through surgery. Heartburn caused by other diseases cannot be cured until the underlying disorder is treated.

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HEARTS is a card game for three to seven persons, each playing for himself, based on the reverse principle of striving to lose tricks rather than win them. A standard deck is used, the cards in each suit ranking from ace (high) to deuce. There is no suit ranking and no trump. (Hearts are often called trumps but do not have the trump privilege.) Cards are drawn for the deal and low card deals. The deal rotates to the left, and the deck is dealt one card at a time. With other than four players, some small cards must be removed to equalize the hands. For example, if six play, the ♣ 2, ♣ 3, ♦ 2, and ♠ 2 are set aside; if five play the ♣ 2 and ♦ 2 are discarded; and if three play, the ♣ 2 is removed.

Eldest hand (player to the dealer's left) leads to the first trick, and the winner of each trick leads to the next. Each player in turn must follow suit if he can, otherwise any discard may be made; the trick is won by the highest card of the suit. Each heart taken in a trick adds one minus point to a player's score. Players try to avoid winning (being *painted* with) any hearts and thus keep their score low.

In hearts there is no standard number of points that constitutes a game, and any number of deals may be played. At the end of each deal the number of hearts taken by each player is recorded. At the end of the game the totals of all players are added to a grand total and divided by the number of players to determine the average score. Then, if chips or money is used, each player pays or collects for the difference between the average and his own score.

An alternative method of scoring is called *sweepstake*. Before the game an equal number of chips is distributed to each player. Then, after the play of a deal, each player puts a chip in the pot for each heart he wins. If one player won no hearts, he is *clear* and wins the pot. If two are clear they divide the pot. If every player is painted the pool is a *jack* and is added to the next pot. If one player takes all 13 hearts, the pot is likewise a jack.

Hearts has many variants. In *spot hearts* the rank of the heart card is counted: ace costs 14, king 13, queen 12, jack 11, and the others their pip value. In *joker hearts* the ♥ 2 is set aside and a joker added. The joker ranks between the ♥ J and the ♥ 10. The joker may not be played by discard, unless a high heart has previously been discarded upon the same trick. In scoring the joker costs five points. In *heartsette*, the full 52-card deck is dealt out evenly as far as it will go. The odd cards form a *widow*, which goes to the player winning the first trick.

The most popular variant is *black lady*, or *black Maria*. In this game, besides each heart counting one, the ♠ Q counts 13 against the one who wins it. After the deal each player passes face down any three cards of his original hand to his right-hand adversary. (With five or more players, only two cards are passed.) If one player wins 13 hearts and the ♠ Q, it is a *take-all*, and 26 points are subtracted from his cumulative total. *Omnibus*, a variant of black lady and generally played with four, counts the ♦ 10 as 10 plus points for the player winning it in a trick. For a *take-all* a player must win all 13 hearts, the ♠ Q, and the ♦ 10, which totals 36 points. The game lasts until one player reaches minus 100 points.

FRANK K. PERKINS
"Boston Herald"

HEAT is a term often applied to the entire branch of knowledge comprising thermometry, thermal expansion, calorimetry, phase transformations, and the thermal flow processes of conduction, convection, and radiation. It is in this broad sense that the term is used in this article. In a narrower sense, "heat" is the name for energy as it passes from one body to another by thermal flow processes. This restricted meaning of the term is better understood when it is related to the scientist's concepts of work and internal energy (see the section *Laws of Thermodynamics*).

CONTENTS

Section	Page	Section	Page
Temperature and Thermometry	14	Phase Transformations	18
Early Theories of Heat	15	Thermal Flow Processes	19
Laws of Thermodynamics	16	Conduction	19
Thermal Expansion	17	Convection	19
Units of Heat	18	Newton's Law of Cooling	20
Calorimetry	18	Radiation	20
		Bibliography	21

TEMPERATURE AND THERMOMETRY

Although a metal cup *feels* colder to the touch than a wooden bowl, after both have been cooled in a refrigerator for a long time, this does not mean that the cup is at a lower temperature than the bowl. How cold an object feels depends not only on the difference in temperature between it and the fingers, but also on the thermal conductivity of the object. Furthermore, the sensation is easily modified by pretreating the fingers; thus, the cup would feel warm if the fingers were immersed in ice water for several minutes just before the test. We conclude therefore that physiological methods for observing temperatures are unreliable. Fortunately, many physical properties, such as the length of a liquid column, the electrical resistance of a wire, or the average ion density in a gas, change reversibly with temperature in a highly reproducible way. A suitably calibrated device that utilizes one of these thermal properties for detecting and measuring temperatures is called a *thermometer* (q. v.). Most common household thermometers depend for their operation on the differential expansion with heat of either liquid-in-glass or one metal on another—for example an oven bimetallic thermometer.

The origin of the thermometer is somewhat obscure. There is evidence that its precursor, the air thermoscope, may have been invented as early as 1592 by Galileo Galilei at Padua, Italy, and independently at about the same time by Cornelis Drebbel of Holland. When scales were attached to them they began to resemble modern thermometers. However, because one surface of the liquid in these devices is exposed to the atmosphere, variations in the readings are affected by changes in atmospheric pressure on the open surface as well as by changes in temperature of the closed bulb. It should be recalled that the existence of atmospheric pressure had not been clearly established at the time. This effect was eliminated fortuitously several years later by artisans of the Florentine Academy who sealed off the tubes so as to prevent evaporation of the liquid. All that these protoypes of the common liquid-in-glass thermometer needed were standardization and calibration of their scales.

The 17th century English scientist Robert Hooke and his contemporaries had observed that the level of the liquid in a thermometer immersed in a mixture of ice and water reaches a steady