Operative Results in Aortic Valve Surgery

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SUCCESS in aortic valvular surgery has approached that comparable to other cardiac defects. This has resulted from the development of competent artificial valves, increased safety of prolonged hypothermic perfusion to allow ample time for the operative procedure, and the proper selection of patients.

During the past 2 years 90 patients with aortic valvular disease have had surgical correction of their valvular defects by means of artificial valvular replacement. A critical analysis of morbidity, mortality, and results has indicated pitfalls to be avoided and has led to modifications in surgical management. There were 15 operative deaths for an over-all operative mortality of 16 per cent, ranging from 12 per cent in patients with pure aortic regurgitation to 21 per cent in patients with calcific stenosis and 33 per cent in patients with multivalvular disease. There were 24 factors relating to the patient, the heart, or the perfusion responsible for death in these 15 patients. More than one factor was present in the majority of the patients (table 1). This report is an analysis of the surgical management in relation to the factors contributing to this mortality.

Factors in Operative Results

The Patient

Age. Age did not appear to be a very significant factor. The youngest patient to undergo successful correction was 6 years of age, and the oldest was 63. The average age of patients having successful operations was 38 years, as compared to 41 years for those who died. There were more complications in the older age group associated with probable vascular changes, particularly with renal involvement.

Coronary Artery Disease. Approximately 50 per cent of the patients showed evidence of myocardial ischemia as a result of the disproportion between available coronary supply and myocardial mass. In seven patients electrocardiographic evidence of myocardial infarction was found in the absence of organic coronary artery disease. Three patients with moderately extensive coronary artery obstruction died following surgery.

The Heart

Cardiomegaly with Left Ventricular Failure

The status of the heart was the major determinant of the operative result. The cardiac volume of patients with predominant aortic regurgitation was greater than in predominant stenosis. The largest hearts were associated with myocardial dilatation, failure, elevated left ventricular end-diastolic pressures, mitral regurgitation, and pulmonary hypertension. The operative mortality in patients with extensive cardiomegaly and elevated left ventricular end-diastolic pressure was over double the mortality of patients in whom the left ventricular end-diastolic pressure was not elevated above 12 mm. Hg. The myocardial factor played an important role in operative success. Twenty-two patients had left ventricular end-diastolic pressures greater than 12 mm. Hg, eight of whom (36 per cent) died following the operative procedure.

Valvular Pathology

The operative mortality in patients with pure regurgitation was approximately one-half that in calcific disease and one-third that of multivalvular lesions. This was largely a reflection of operating time. It took considerably longer to remove a calcified valve, particularly if the calcification extended into the aortic wall and myocardium, and to prepare the bed for the valvular insertion and
fixation. The operating time was also extended if more than one valve had to be surgically corrected. The operating time for patients with pure aortic regurgitation averaged 127 minutes, for calcific stenosis 180 minutes, and multivalvular disease 184 minutes. Greater experience and changes in valve design have resulted in shorter perfusion times.

### Myocardial Protection

Patients with aortic valvular disease showed varying degrees of myocardial hypertrophy, dilatation, fibrosis, and ischemia. If surgery were to be successful, maximal myocardial protection had to be employed during the bypass procedure to prevent oxygen deprivation.

From the maze of experimental data on myocardial metabolism under varying conditions of cardioplegia, anoxia, and hypothermic cardiac arrest, it has been difficult to learn what constitutes adequate myocardial protection.

Bing and co-workers\(^1\) demonstrated changes in the transmembrane potentials 4 minutes after anoxia and no transmembrane potentials after 20 minutes of complete myocardial ischemia. Glycogen and high-energy phosphate disappeared rapidly during anoxia. This explains the inadequate myocardial protection afforded potassium and acetylcholine arrest.

To obviate the disadvantages of potassium or anoxic arrest, we instituted myocardial protection by means of direct coronary artery perfusion with normothermic blood in 1958.\(^2\) The coronary perfusion rate was regulated by the appearance of the myocardium and the electrocardiogram. Coronary flow varied between 300 and 500 ml. per minute. The heart was kept beating during the operative procedure, which was largely palliative. All of these patients survived surgery but five died during the postoperative period. Postmortem examination revealed incomplete correction of the aortic regurgitation in these patients and, in addition, multiple petechial hemorrhages throughout the myocardium. It was postulated that these petechial hemorrhages resulted from the rupture of capillaries from the constant coronary perfusion pressure throughout the cardiac cycle. Compression of the deeply located coronary vessels and increased ventricular pressure cause reduction in coronary flow during systole. Continuous flow by the coronary perfusion pump in the face of myocardial resistance during systole led to rupture of the smaller vessels. By induction of ventricular fibrillation at present, this phasic increase in coronary artery pressure is abolished so that petechial hemorrhages are no longer encountered.

### Selective Hypothermia

Myocardial protection is provided by hypothermia. According to Urschel and Green-
myocardial oxygen consumption is reduced to 50 per cent at 30 C. and to 10 per cent of normothermic consumption at 10 C. Gott et al. demonstrated a minimal breakdown of glycogen and adenosine triphosphate during selective hypothermic arrest in contrast to the utilization of these substances during potassium arrest.

Selective Hypothermic Arrest

Six patients with aortic valvular disease were operated upon under selective cardiac hypothermia (10 to 20 C.) with oxygenated lactated Ringer's solution by perfusion of the coronary arteries through the base of the aorta and by local application of slush. Coronary circulatory arrest by this technic was successful for periods up to 60 minutes even though a myocardial acidosis occurred. In no instance in which the arrest was longer than 60 minutes, however, was the operation successful. Microscopically, the myocardium in these hearts showed diffuse and patchy areas of petechial hemorrhages and focal necrosis.

Myocardial protection for periods less than 60 minutes could not always be safely provided. For example, a 34-year-old man with calcific aortic stenosis had a valvuloplasty with removal of calcium under selective cardiac hypothermia (12.5 C.) for 26 minutes of coronary circulatory arrest. Postoperatively he did well with hemodynamic evidence of a good-functioning valve. On the fourth postoperative day, he died of left ventricular failure. Postmortem examination of the myocardium showed multiple necrotic areas. Deep hypothermia may produce detrimental myocardial effects and be unable to provide sufficient myocardial protection in patients with marked left ventricular hypertrophy for even relatively short periods of arrest.

Technic of Cold Arrest and Coronary Perfusion

Studies of myocardial metabolism revealed metabolic acidosis during hypothermic cardiac arrest even for relatively short durations. Such an oxygen debt is considered inadvisable for prolonged periods in the presence of severe myocardial hypertrophy. Since artificial valvular replacement of the diseased aortic valve has required a minimum of 1 1/2 hours, myocardial protection by hypothermia alone appeared inadequate.

Greater myocardial protection could be provided for longer periods by employing hypothermia and coronary artery perfusion. This technic has now been employed in 90 patients having valvular replacement. In only four patients was it impossible to re-establish normal cardiac function; one had a markedly enlarged heart and postmortem evidence of an extensive pre-existing subendocardial infarction, one had congenital anomalies of the coronary arteries that prevented adequate perfusion, and two had associated coronary artery disease. In our opinion perfusion of both coronary arteries should be performed to provide maximum protection.

Total body perfusion was maintained by a Kay-Cross rotating disc pump oxygenator. A heat exchanger was placed in the venous line to provide general body hypothermia with esophageal temperature about 25 C. Cardiac arrest was accomplished by perfusing the coronary arteries through the proximal aorta with oxygenated cold Ringer's lactate (0 to 3 C.) and by local application of slush until the heart went into standstill. Ventricular fibrillation usually occurred around 20 C., and complete arrest about 12 C. From 500 to 1,000 ml. of fluid were required to obtain standstill in 15 to 30 seconds. Ordinarily, hypothermic arrest is obtained by both coronary perfusion as well as local application of ice-cold Ringer's solution. When hypothermic arrest is obtained only by external application of cold to the hypertrophied heart, the ventricular septum cools more slowly than the external myocardium. Decompression cannulae in the right and left sides prevented overdistention of the ventricles.

The aorta was then opened and the coronary arteries were cannulated and perfused with hypothermic blood regulated by a second heat exchanger in the coronary perfusion line to maintain myocardial temperatures between 12 and 20 C. Each coronary line was indi.
AORTIC VALVE SURGERY

Individually controlled and monitored by its own pump-perfusion circuit. A common pump or gravity circuit was found inadequate to assure the desired individual coronary flow. Individual coronary flow rates between 100 and 300 ml. per minute were used to maintain coronary perfusion pressures between 80 and 120 mm. Hg.

During coronary perfusion the decompression cannulae were connected to a sump, and the blood from the heart was returned to the venous side of the oxygenator. In effect, two separate circulations with a common oxygenator were established. The depth of hypothermia was dependent upon the adequacy of coronary perfusion and the degree of myocardial hypertrophy. No attempt was made to defibrillate the heart until completion of the operation because of the theoretically reduced oxygen requirements of the fibrillating heart, better constant coronary perfusion without the systolic phase and the quieter operative field and reduced blood loss in the absence of the pumping action of the ventricles.

During the operative procedure, general body hypothermia with esophageal temperature about 25 C. was maintained. Thereby lower flow rates have been possible with maintenance of basic physiologic requirements within normal ranges for periods up to 4½ hours bypass time without any significantly increased hemolysis (fig. 1).

During closure of the aorta, the patient and the heart were rewarmed. After the aortic clamp was released, both sides of the heart were vented by gravity into a reservoir to prevent overdistention of the myocardium and flooding of the pulmonary vascular bed. At that time the pump output was usually increased to improve coronary perfusion and to facilitate rewarming. When the myocardial temperature reached 32 to 34 C., electric defibrillation was performed. The left ventricular decompression cannula was then drawn back into the atrium and decompression was gradually decreased as the heart took over the circulation.

Coronary Artery Flow

There was an average left coronary flow of 260 ml. per minute as compared to 165 ml. per minute for the right coronary artery. The left coronary flow exceeded the right in 70 per cent of the patients. The coronary flow was purposefully kept high because of the increased oxygen extraction with higher flow rates, even in the presence of hypothermia. The adequacy of myocardial protection during the operative procedure is attested by the fact that an average of only 10 minutes additional support time by the pump was required before termination of the perfusion.

General Body Perfusion

With increasing experience the deleterious effects of extracorporeal perfusion have become lessened. During the first years of open-heart surgery, operations longer than 1 hour of perfusion were not likely to be successful. Perfusion time has now been extended with safety to 3 to 5 hours. Hypothermia has added considerably to the safety of prolonged perfusion but it can in itself complicate the procedure. The main asset of hypothermia is
the resulting lessened oxygen requirements of the body that allow the perfusion to proceed at lower flow rates. Little clinical information is available on prolonged hypothermic perfusion performed under the varying clinical conditions. Successful results depended upon complete correction of all hemodynamically significant valvular deformities. In the case of aortic valvular disease, correction can be realized in the majority of instances only by artificial valvular replacement—a time-consuming procedure, particularly with a largely destroyed calcified valve.

Operating Time

The average operating time for patients who survived operation was 134 minutes as compared to 177 minutes for those who did not. If the four patients are omitted in whom the heart could not be restarted, the average additional perfusion support time was 10 minutes for those who survived against 9 minutes for those who died. The majority who required the additional 43 minutes operating time consisted of patients with extensive calcific stenosis or multivalvular lesions. The short duration of the pump support following surgical correction of the valvular deformity attests to the high degree of myocardial protection it afforded. The over-all operative mortality was 15 per cent when the operating time was 2 hours or less, and 17 per cent when the operating time was within 2½ hours. However, if the operating time was greater than 2½ hours, the operative mortality increased to 50 per cent. Operative time obviously is not the only factor involved in this comparison. The majority who required longer than 2½ hours comprised those with multivalvular disease in failure.

Hemolysis

Excessive hemolysis undoubtedly is a factor in morbidity and mortality. Excessive hemolysis can result from incompatible blood among donors as well as between donors and the recipient, from insufficient rinsing of the detergent used in cleaning the oxygenator system, improper siliconization, excessively long perfusion, and excessive use of sump suction. To avoid undue hemolysis from incompatible blood, samples from all donors are pooled and tested for hemolysis. The oxygenator is then primed and rotated for 15 minutes when hemolysis is determined again. This level should not be significantly increased. Upon two occasions in the past 2 years excessive hemolysis (above 100 mg.) was noted in the second sample: the increase was thought to result from improper rinsing of detergent or improper siliconization. In these instances the oxygenator system was replaced.

With proper care of the oxygenator excessive increase in hemolysis is not expected, even for perfusions of 3 to 4 hours unless excessive sump suction has been necessary. The average hemolysis for those patients who survived increased from 22 to 70 mg. per cent as compared to an increase from 21 to 71 mg. per cent in those who died. No bleeding problems were encountered in either group except perhaps for several who had re-operations and who had extensive vascular adhesions. Secondary operation for hemostasis was required in only one patient.

Pitfalls of Long Hypothermic Perfusion

Seven patients died postoperatively from renal, cerebral, or intestinal complications thought possibly due to hypoxia occurring during perfusion. All perfusion records were therefore analyzed to determine any common denominators responsible for these complications. In most instances, no one factor, but rather a combination of factors was found whose summation over a long period undoubtedly caused hypoxia responsible for the fatal complications. The standard method of determining oxygen consumption calculated on arteriovenous oxygen differences is dependent upon oxygen extraction. Oxygen consumption values under hypothermia do not indicate that tissue requirements have been met. It is obvious from oxygen dissociation curves that less oxygen is available to the tissues with decreasing temperature. This may be influenced by variations in acidosis and oxy-

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gen tension. The assumption that adequate oxygen has been provided to fulfill tissue requirements, based on theoretic and experimental studies on oxygen consumption, is fraught with danger. A discussion relative to factors or combinations of factors producing hypoxia in prolonged hypothermic perfusion follows.

Cooling Speed

In an adult the efficiency of cooling is less than in a child. Gradients may exist in varying regions of the body that cool at different speeds. Esophageal temperature alone may be misleading, for the esophagus, being adjacent to the cold pericardium, is cooled considerably faster than other tissues. If flow rate is lowered before hypothermic equilibration, the oxygen available to some organs may be insufficient. The cooling speed is dependent upon the size of the individual, the rate of perfusion, and the efficiency of the heat exchanger. It is significant only when the flow rate is prematurely reduced so that insufficient oxygen is available to the warmer tissues. Rectal or peripheral temperatures as well as esophageal should determine when flow rate is to be reduced. Too early a reduction in flow rates in two patients with a rapid cooling speed may have been a factor producing hypoxia.

Potential Factors Producing Hypoxia

Rate and Duration of Flow

Factors that may be responsible for hypoxia include not only the rate of flow but also its duration. The shorter the operative procedure, the lower is the flow rate that can be tolerated. Alterations in metabolism will be corrected with resumption of adequate flows if conditions are otherwise satisfactory. In our experience flow rates of 50 ml. per Kg. per minute have always been adequate to fulfill the oxygen requirements in adults at normothermia. Experimental evidence based on arteriovenous oxygen difference shows the oxygen consumption at about 25 C. to be approximately 30 per cent that at normothermia. A reduction in flow rate of 50 per cent to 25 ml. per Kg. per minute should provide ample oxygenation at 25 C.

Analysis of our perfusion records demonstrated the fallacy of basing tissue oxygen requirements on oxygen consumption. Specific levels of oxygen consumption do not indicate that the tissue requirements for oxygen were completely fulfilled. The amount of oxygen consumed is known to depend upon the flow rates, the temperature level, the oxygen tension, and the pH of the blood. In addition, other factors difficult to specify determine whether complete oxygen protection had been provided.

In complicated operations in which prolonged perfusion was required, it was thought advisable to reduce flow rates to minimize trauma to the blood. Consequently, flow rates of 25 ml. per Kg. per minute were employed at 25 C. in some instances, with the thought that adequate oxygen protection was being provided. These flow rates could be tolerated without metabolic acidosis and complications of hypoxia if the flow at this rate was for 60 minutes or less. If the duration was longer than 60 minutes, 45 per cent of the patients died or showed hypoxic complications. That 55 per cent were not so complicated would indicate the oxygen availability of flow rates of 25 ml. at 25 C. was just borderline in supplying the oxygen requirements.

It also appeared that older patients tolerated the lower flow rate of 25 ml. less well than younger ones, perhaps due to the increased vascular resistance in the older age group and the increased vascular resistance and viscosity of the blood inherent in hypothermic perfusion. In such instances there frequently was an elevated mean arterial pressure associated with lessened venous return thought to be due to difficulty in circulating the blood through the smaller arteries and capillary system. In the presence of an elevated mean arterial pressure there was a tendency to perfuse at the lower rates. An elevated mean arterial pressure at 25 C. is not necessarily an index of a satisfactory perfusion. It may indicate a precarious per-
fusion in the face of undue vasoconstriction and indicate either rewarming of the patient to a lesser degree of hypothermia to lessen the effect of vasoconstriction, or to increase further the flow rate to push through this added vascular resistance.

Hemodilution

In a recent attempt to reduce the requirements for blood, the oxygenator system was primed with 50 per cent blood and 50 per cent glucose in 1/4 per cent saline solution. The hematocrit level was lowered from an average of 42 to 32 but returned to normal in 30 to 45 minutes. The oxygen content of the blood was reduced from approximately 16-18 to 12-14 mg. To compensate for this reduction in the oxygen-carrying capacity of the blood, flows of 30 ml. per Kg. per minute were used. There was no increased mortality in patients in whom hemodilution was employed when the flow rate was maintained above 30 ml. per Kg. per minute at 25 C., in contrast to a 40 per cent mortality in patients perfused at 30 ml. per Kg. per minute from what appeared to be hypoxic complications.

Oxygen requirements were found to be greater than theoretical calculations. This increased operative mortality occurred only in patients perfused for over 60 minutes at less than 30 ml. or at 30 ml. in association with hemodilution. Subsequent to this analysis flow rates of 35 ml. per Kg. per minute or greater have been employed without any sustaining of hypoxic complications. This should lower the over-all operative mortality in the future.

The Valve

Fibrin deposition on the artificial valve was minimized in experimental studies by lightly filming the Teflon fabric with polyurethan as well as by postoperative heparinization. Fibrin deposition has not been a problem clinically. A three cuspid aortic valve patterned after the normal aortic valve has been used. A gradient has not developed across the artificial aortic valve during the 2-year period of its use, which might be indicative of a stenosing factor occurring. Valve design and the technics of insertion have not led to acute operative mortality. Only one episode of an embolic phenomena occurred postoperatively. It did not appear that the valve was the source of embolism.

Difficulties with valve design and technics of fixation have led to regurgitation in seven patients. These patients were reoperated on with two survivals. Two factors were responsible for this recurrence. These were insecure fixation of the commissures and failure of tissue in growth into the valve.

Of 45 patients with pure aortic regurgitation, 42 have had surgical correction by means of sewing the artificial tricuspid valve onto the existing aortic leaflets. In an occasional patient the thickened free edge of the leaflet was first excised, but in the majority it was not considered necessary. In only one instance did the leaflet tissue fail to hold the sutures and regurgitation recurred. This patient was reoperated on with use of a full-depth artificial valve and survived.

The valve must be of proper size. If the valve is too small, regurgitation may take place after the aorta dilates with restitution of aortic pressure because the leaflets cannot approximate. If the artificial valve is too large, improper suspension of the free edge of the leaflet from the commissure results, so that the center of the leaflet may herniate downward with diastole and fail to fit properly. These complications are prevented by measuring the area size of the aortic valve with a calibrated dilator which is slightly distended at the time of this measurement. Valves are available in sizes from 2.8 to 3.4 cm.

The greatest stress on the artificial aortic valve appears to be at the commissure level. The diastolic pressure is reflected into the sinus of Valsalva and causes a downward and inward pressure upon the leaflets as well as a lateral pressure upon the aortic wall. The greatest strain resulting from these opposing pressures is at the level of the commissural attachment. Commisural detachment was found to be responsible for recurrence of...
regurgitation in four instances. The valve design was changed (fig. 2) so that permanency of fixation could be assured. Extensions of the valve leaflet were fashioned 5 mm. in width and 4 inches in length. Teflon fabric can be stretched in the horizontal plane double its length. These extensions were pulled into cords, threaded on a needle, and passed through the aortic wall behind which they were tied with the corresponding extension from the adjacent leaflet over pledgets of Teflon felt. They may be further secured by tying each pair from the three commissures around the aorta. There have been no recurrences of regurgitation over 8 months in 40 patients.

The second factor—failure of tissue ingrowth and fixation at the site of suture of the valve at the aortic annulus occurred in valves in which Teflon felt was used at the suture edge of the leaflet in the belief that this might act as a seal. In three instances tissue failed to grow completely over and into the Teflon felt. The suture eventually broke. Again, when this complication became apparent, the use of the buttressing edge of felt was discontinued. A 4-mm. edge of the Teflon leaflet is left free of the polyurethan for suture fixation. This porous area allows tissue ingrowth. The valve is further secured with extension of the leaflet at the base like the one at the commissural level. It is thought that with the present valve design and technique of fixation the likelihood of recurrence because of insecure fixation has been eliminated.

All calcium deposits on the valve must be removed for tissue ingrowth to occur. During this procedure, calcium particles may fall into the ventricle and later become embolic. In one instance several hours postoperatively embolic occlusion of the left coronary artery occurred in this way. This complication can be avoided by packing the ventricular chamber with one-inch gauze through the residual slit in the stenosed orifice. Following removal of the cal-

Figure 2

*Present valve design employing extensions of leaflet Teflon fabric into cords passed through aortic wall to assure permanency of fixation.*
cified valve, the pack is removed before insertion of the aortic artificial prosthesis.

Clinical Results

It is too soon for an opinion about late results of surgical correction of aortic valvular disease with artificial valves; nevertheless, the status of 30 patients 6 to 24 months after operation is presented in table 2.

There has been a significant roentgenographic reduction in heart size, averaging 2 cm. in patients with pure regurgitation and 1 cm. in patients with calcific stenosis or multivalvular disease. This reduction probably represents reduction in myocardial dilatation rather than in myocardial mass.

As might be expected, little change has been noted in the electrocardiogram, which still reflected left ventricular hypertrophy. Auscultation and phonocardiographic examination revealed a slight systolic murmur in the majority of patients but no diastolic murmur. This slight systolic murmur was heard immediately postoperatively and did not change over the ensuing months.

One hundred per cent of the patients evaluated who had only the aortic valve involved, had clinical and hemodynamic improvement. In patients with multivalvular disease, physiologic studies demonstrated a normal-functioning aortic valve: in two instances persistent mitral valvular disease or myocardial irritability gave rise to problems in digitalization.

Competency of the aortic valve has been demonstrated by normal arterial tracings as well as by cineangiography.

Over this period of time, there has been no clinical or hemodynamic evidence of undue fibrin deposition and development of stenosis. Combined tracings of left ventricular and aortic pressures demonstrated no evidence of a gradient across the artificial aortic valve up to at least 24 months postoperatively.

In this group of 90 patients with artificial valvular replacement there has been no evidence of adverse effect upon the blood components, increased hemolysis, or anemia. One patient, however, developed evidence of subacute bacterial endocarditis approximately 10 months postoperatively following a lung infection. There was one other probable instance of such an infection developing 2 months postoperatively, although never proved. Since such a complication may exist, precautionary measures should be taken during septic periods.

Summary

An analysis of the surgical management in relation to factors contributing to morbidity, mortality, and results in 90 patients having...
artificial valvular replacement for diseased aortic valves is presented. Twenty-four factors occurred in 15 patients which were responsible either alone or in combination for the deaths. These factors fell into five main groups related to the patient, the heart, the myocardial protection, the general body protection, and the valve. Most of these factors contributing to death are preventable. Therefore, the over-all operative mortality of 16 per cent should be materially reduced in the future.

References

"The Usefulness of Useless Knowledge"

The marvel of beholding for the first time a fresh aspect of nature fascinates the investigator and, even through privation and struggle and repeated disappointments, the possibility tends to hold him strictly to the search. This single-mindedness in striving for new knowledge involves a large degree of neglect of the motives for money-getting. After all, new knowledge—knowledge in its unprofitable infancy—obviously does not possess commercial value. The vast majority of mankind are well satisfied with what they know. There is no demand from them that their relative ignorance be increased by further additions to the accumulated information available to the race. During nearly twenty years the Dutch botanist, De Vries, investigated the origin of variations in plants. His discoveries threw much light on the processes of biological evolution. If money were the motive, who would labor twenty years in order at the end to publish a book which only university libraries and a few meagerly paid biologists would care to buy?

No intelligent person would for a moment think that research in "pure" science, which has been motivated by personal curiosity and which, though satisfying that curiosity, has not resulted in immediately useful discoveries, is destined to have little or no economic value. There are too many examples all about us illustrating the fact that increased knowledge of nature yields increased power over her processes. The mathematical predictions of Maxwell and the experiments of Hertz made possible the elaborate modern development of wireless transmission which brings news and entertainment to myriads of homes and protects the lives of travelers on land and sea. The two men were not concerned with possible commercial value of their studies. The motive impelling them was the scientific motive, the desire for understanding. Such men may be grateful that accumulated wealth gave them leisure to carry on their investigations, free from the worries of uncertain livelihood; but they would look upon eagerness for money without envy. This attitude should not be regarded as implying a claim of moral superiority, for the investigator is in the happy position of doing his specially chosen work and receiving a livelihood in addition. This livelihood is not lavish, to be sure; if it is sufficient to meet the simple requirements and tastes of the investigator and his family, it is enough.—WALTER B. CANNON, M.D. The Way of An Investigator. New York, W. W. Norton & Company, Inc., 1945, p. 206.
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