The Biology of Myocardial Infarction

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SUMMARY

Myocardial infarction is a dreaded companion of the civilization of our time. The "heart attack," the coronary thrombosis, the myocardial infarction lurk behind the corner, striking strangers, patients, friends, and ourselves. The toll taken by this single "disease" in many hospitals equals or even exceeds that from almost all other diseases together. Must it be so? This is the question many physicians, and the lay public, are asking themselves today.

Identification of some areas where our knowledge is particularly deficient is stressed. We must concentrate our efforts on reevaluating practices and procedures that have brought both the greatest success and the worst failures, at the same time that we ask ourselves what goals could be considered realistic.

If myocardial infarction is the evil of our time, what does this mean in a greater biological context—in the evolution of human life and endeavor as viewed in perspective? Are we dealing with a "disease" in the restricted sense of the word, or are we dealing with a way of life and a consequent mode of death? If so, what can be done to achieve the goals of health as defined by the World Health Organization? In what terrain are the forces to be assembled and the battle to be fought—and by whom.

Additional Indexing Words:
Coronary heart disease
Hospital mortality from coronary heart disease
Intensive care units

Angina pectoris
Statistical approach

THE American Heart Association has asked me to give the first Paul Dudley White International Lecture. For this honor I express my sincere thanks.

I pay tribute to this great nation as a leader in science, in medicine, in cardiology, and to Paul Dudley White, scientist, physician, teacher of cardiology, and ambassador of good will. He is known to the whole world as an inspiring example of the American people at their best. I have had the good fortune to know Paul White for more than 20 years as teacher, research companion, and friend.

A visitor from abroad cannot be expected to bring much new information on the subject of myocardial infarction to the cardiologists of this country. Your nation is at the top of the list both with regard to prevalence and incidence of that disease and with regard to research concerning its causes, effects, treatment, and prevention. And yet, the subject proposed to me, "The Biology of Myocardial Infarction," is close to my heart; something I have been living with the last 25 years, and with which I have been wrestling as biochemist, physician, sociologist, and hospital administrator. I have been forced to analyze the problems at different levels and with various techniques. I shall try to convey some of my experiences, thoughts, and unanswered questions.

Coronary Heart Disease and Myocardial Infarction

Myocardial infarction is an acute event in the course of a chronic disease of multifactorial origin. The "biology" of this event relates both to the predisposing causes and mechanisms, and to possible precipitating factors (fig. 1).
Causes and clinical manifestations of coronary heart disease.

**Figure 1**

Causes and clinical manifestations of coronary heart disease.

**Figure 2**

Trends in mortality from coronary heart disease (B 26) in Sweden (Reprinted with permission from Biörck, G.: Läkartidningen 64: 5231, 1967.)
BIOLOGY OF MYOCARDIAL INFARCTION

What we know about myocardial infarction we know from clinical case material, from autopsy studies, and, in addition, from epidemiological field studies and international statistics based on death certificates. We have reason to believe that morbidity and mortality patterns in coronary heart disease are not everywhere congruent.

Many issues are still ambiguous. An infarct has been considered to be caused by an occlusion of a coronary artery, mainly by a thrombus. However, in many instances, no thrombus is found despite microscopic evidence of damage to the myocardium. Some patients present the classic clinical picture of myocardial infarction. Others succumb in a few minutes to fatal arrhythmias. Some develop angina pectoris; others, progressive heart failure indistinguishable from myocardial weakness from other causes.

Figure 3

Mortality for men from all causes and from coronary heart disease (B 26) in the United States and in Sweden for 1963-1964. Death rate levels per 100,000 mean population for men in the U.S.A. at the age of 40, 45, 50 years and so on, and the corresponding ages of men in Sweden with equal death rates (age in years in the circlets).
Clinical research is generally based on hospital patients. Infarct patients brought to the hospital may live long enough for us to engage in a battle whose outcome is always uncertain. In sudden death the battle is over before it has begun. In progressive cardiac failure of the aged the acute dramatic element is often missing. This is why, in part, we know little about many coronary manifestations outside the hospital, and why classical infarcts have stolen the show.

What Makes the Myocardial Infarct Such a Big Problem?

At my hospital in Stockholm, the Seraphimer, deaths from myocardial infarction now make up 40% of all medical service deaths. The number of patients admitted with myocardial infarction has been increasing every year during the last 30 years. This is evident in data from both the General Hospital in Malmö and the Seraphimer Hospital, the two medical services in Sweden where this development has been most closely studied.

In Malmö, we found that the 4-week hospital mortality from cardiac infarction fluctuated around 30% in each 5-year-period between 1934 and 1959.

In Stockholm, there has been an alarming rise in the mortality figures back to and even exceeding levels I remember from the early 1940's in Nylin's department.

Impressions from hospitals may be misleading and have to be correlated with information about the population served by the hospital. Such data are scarce. Malmö is a useful example because there is only one large hospital serving a community of ½ million inhabitants. Framingham is another example.

Figures from the Central Statistical Office in Sweden (fig. 2) show a moderate increase in mortality from coronary heart disease (B 26) in men over recent years, and a corresponding decrease in women—a trend observed almost everywhere.

Despite our high hospital mortality rate our figures on death from coronary disease are, internationally, quite low (fig. 3). Sweden and Denmark seem to follow the same pattern. Norway, while starting lower, is now in the process of overtaking us. Finland is far above us all, holding a position as an equal to the Anglo-Saxons: United States, United Kingdom, Canada, Australia, New Zealand, and Ireland. Why that is so, we do not know.

These figures relate to deaths from all coronary disease (B 26), and not particularly from myocardial infarcts.

The fraction of all coronary death certificates in which infarction was stated as the cause of death has been shown to vary considerably between age-groups, being high in younger groups and low in older groups. For a long time I was inclined to interpret these findings as indicating that a greater proportion of older persons die from progressive cardiac failure, ascribable to atherosclerotic heart disease.

What has begun to worry me increasingly is the knowledge about sudden death unravelled parallel with new techniques of cardiac resuscitation and coronary care in much the same way that the cardiac catheter paved the way for fuller recognition of congenital heart disease. The best source of information still seems to be the Framingham study in which one out of six men who developed symptoms of new heart disease during a 12-year observation period died unexpectedly within less than an hour. These deaths appear to have made up 40% of all male coronary deaths during the period of observation.

Even more impressive figures have recently been collected by Morris on a population of 7,000 male British physicians, aged 40 to 64 years, in whom sudden death appears to be more than twice as frequent as other types of death ascribed to ischemic heart disease. If this is true, what is the place of the myocardial infarct? We do not know how to classify sudden death in relation to an infarct. In contemporary lingo it may represent "electrical failure" rather than "power failure," and this may escape the notice of even the
most careful pathologist. But there is a fair suspicion, substantiated by findings in a recent analysis from our department, that the better we prepare for the instant care in our hospitals of patients with "heart attacks," whether living, dying, or already dead, the greater will be the transfer of the sudden death cases now in the death-certificate group outside the hospital to the hospital sector. There they may be given a diagnosis of myocardial infarction (especially in case of survival). Such a process will make considerable impact on statistics and planning, particularly as the older citizens who represent the vast number of victims of coronary heart disease avail themselves of Medicare or other inventions of our great societies. We should not forget that although much emphasis is commonly placed on infarcts among middle-aged executives, the President's commission in this country stated that 72% of all cardiovascular deaths in the U.S. occurred in persons 65 years or over. The corresponding figure in Sweden today would be 84%.

In this connection attention should be drawn to the fact that mortality from coronary heart disease (B 26) shows a fair correlation to mortality "from all causes." The high coronary death rates in some Western countries are found in countries with a high "biological age" (for example, a high overall mortality from all causes) and vice versa.

The percentage of coronary deaths in relation to total deaths in age groups over 60 years is rather similar in all Northwestern European countries, irrespective of differences in the younger age groups, whereas Italy, Portugal, and Japan have much lower relative frequency of CHD-deaths at all ages. The United States and Canada have a higher proportion of CHD-deaths.

This observation reflects another aspect of coronary artery disease. It has frequently been stated that more and more of its victims belong to younger age groups. Even though the upward trend in the youngest male age groups is getting steeper in my country, the numbers are still small and count little in the total picture. Rather the average age of Swedes who become hospitalized with a first myocardial infarct has been rising over the last 25 years, about 5 years for men and 10 years for women. This rise is of the same order as the increase in life expectancy at birth over a corresponding period and is far in excess of the increase in life expectancy at age 50.

At what time in your life do you get your extra years added or subtracted? It may be in early life, as suggested by Hardin Jones. I think it is important to bring this question into any consideration of the biology of myocardial infarction.

I have repeatedly stressed that most of the information from vital statistics concerns coronary heart disease overall and not myocardial infarction. We cannot easily project hospital data onto the population at large. But now that we are mobilizing forces to combat more effectively the acute manifestations of coronary heart disease, we ought to know more of the incidence of sudden death, myocardial infarction, and other clinical manifestations of coronary heart disease in our communities. This will necessitate some kind of coronary heart disease registry in defined populations, a world-wide Framingham study, without upper limits as to age and with emphasis on types of "new disease," on modes of death, and on disability and rehabilitation. A recommendation to that end was recently forwarded by a scientific group within the WHO.

The Course and Prognosis of Myocardial Infarction

While it is difficult to deal with the natural history of myocardial infarction as an isolated phenomenon, something may nevertheless be said about its course and prognosis.

In the large groups of cases with primary myocardial infarction in Malmö, to which I have referred, the future course of the "disease" could be empirically described in figure 4.

Other large hospital statistics give similar results. Information on the subsequent course of patients with angina pectoris, which is
Some aspects of the natural history of myocardial infarction. (Reprinted with permission from Börck, G.: Course and prognosis in some cardiac diseases. 1962, Pergamon Press.16)

sparse since the early reports of White and associates22 and of Block and co-workers23 indicates that the prognosis in angina pectoris is equivalent to that for survivors of an acute myocardial infarct.24 However, the average course and prognosis give little information on the many factors that influence the fate of the individual patient. Rather they include and obscure such factors. Some of these relate to the patient, others to the treatment given. We know that age is very important in determining the mortality risk during the first weeks of the acute infarct, mortality here being almost linearly related to age.25
The apparent higher mortality in women can mainly be explained by their being older at the time of attack. For this reason, mortality figures on infarction materials always must be related to the age of the group. International comparisons are confounded by the fact that "biological age" in any two populations may correspond to different chronological ages.

When it comes to long-term prognosis, it is clear that older age-groups have a shorter absolute survival after infarction than the younger groups. But if comparisons of remaining life-expectancy are made between survivors of a myocardial infarct and the uninvolved population of the same age, younger persons fare less well than older ones, that is, for them the mortality risk is greater (table 1).

Other factors that seem to influence long-term survival in infarction are antecedent diabetes—overt or latent—and hypertension. This may be explained merely as the addition of two risks to each other.

The size of the infarct, judged from determination of the enzymes GOT and LDH, appears to influence the immediate prognosis as well as the long-term prognosis. These data may become obsolete in view of present trends in treatment, which is the other big factor influencing course and prognosis. We cardiologists have not been able to show improvement, either in immediate or in long-term prognosis over the past 25 years. Immediate prognosis with the patients may even have worsened in recent years, probably as a result of patients with more severe conditions being admitted to the hospital. Both in Malmö and in Stockholm hospital communities, there is a continuous increase in the mean age of patients at their first infarct which per se would increase the immediate mortality. Therefore, even a constant mortality rate might represent some improvement. On the other hand, several recent reports from special coronary care units indicate considerable reductions of immediate mortality. This is a new feature which must be taken into account in future predictions of the course and prognosis in patients with coronary heart disease. It has been true also of our own coronary care unit.

It is fair to say that we simply do not know what effects our previous endeavors have had on long-term prognosis in myocardial infarction and in coronary heart dis-

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**Table 1**

*Excess Mortality in Patients Surviving the Acute Phase of Primary Myocardial Infarction*†

<table>
<thead>
<tr>
<th>Age (yr)</th>
<th>No. of patients</th>
<th>Number of deaths/expected deaths after 1 yr</th>
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*Number of expected deaths calculated from census data (age and sex specific death rates, Swedish town population 1941-1950).
†Reprinted with permission from Biörck, G.: Course and prognosis in some cardiac diseases. 1962, Pergamon Press.*

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ease at large. But at the present time, when coronary care units are mushrooming in many countries, and when the U. S. is contemplating a 50 million dollar venture into preventive dieting, it may be useful to consider what we are aiming at in our fight against this disease.

**Which Are Our Aims?**

The challenges are: (1) to postpone the first manifestations of coronary artery disease (especially myocardial infarction and lethal arrhythmias), (2) to reduce the acute mortality (especially in myocardial infarctions and arrhythmias), (3) to postpone repeated attacks (especially of myocardial infarction and arrhythmias), and (4) to improve the condition of the survivor (rehabilitation).

With these in mind should we aim primarily at fighting coronary heart disease or myocardial infarction? Shall we attempt the long range elimination or reduction of the causes of atherosclerotic processes in the coronary vascular wall, which is an enormous undertaking, or should we aim at the instant and energetic battle against the occluding thrombus, the lingering arrhythmias, and the myocardial weakness as they present themselves in the acute event? It seems to me that there has recently been a shift of emphasis within the medical profession away from preventive aspects of coronary heart disease that have been so favored for a decade or so, and toward the coronary care unit and the philosophy of immediate action, a move from strategy to tactics.

The strategical approach, if I may say so, derives from a biological philosophy based on animal experimentation and epidemiological surveys. It tries to modify and counteract pathogenic mechanisms, particularly in persons earmarked as especially vulnerable candidates for future disease. The tactical approach is an expression of a humanitarian philosophy: to fight death and save life, vigorously, unyieldingly, and at almost any price.

Although these goals are, of course, not mutually exclusive, we must be aware that for years to come in many countries, including the United States, medicine will have to decide on priorities, because medical manpower will remain scarce, and the costs of acute medicine will be rapidly mounting. Let us, therefore, try to examine the prospects of both avenues!

**The Strategical Approach**

Studies since the time of Anitchkov relating vascular pathology to lipid biochemistry in rabbits or chickens have added much to the brickwork of understanding coronary heart disease. But humans are not rabbits and are not in cages. Disease as a "patterned response" may also be studied elsewhere, in human populations, where nature or history create their own experimental conditions. The identification of certain metabolic patterns in man leading to coronary atherosclerosis, and of other nervous reflex patterns involved in the genesis of arrhythmias, has forced the question of the phylogenetic meaning of such patterns, which once may have been of great survival value but in our present form of life may be conducive to disease and death. The "agent" of disease may be neither outside nor exclusively within; disease may develop as a result of maladaptation of an old-fashioned biological machinery exposed to the forces of quite another life situation than it was evolved to master.

What have we learned about the occurrence of myocardial infarction in our zoological ancestors and relatives? While atherosclerosis of the aorta with advancing age seems to be as frequent in many animal species as in man, coronary atherosclerosis is a rare feature observed, for example, in some wild birds such as pigeons, which may die from myocardial infarction. There may even be "epidemics" of myocardial infarction in some birds during the breeding period. But it is only in the large primates, such as gorillas and chimpanzees, that the human pattern of coronary arteries, diseased before puberty, is observed. It has been stated that records and observations on disease and death of mammals and birds in zoological gardens indicate...
that coronary artery disease, myocardial fibrosis, and infarction reflect effects of physiological and psychological responses to social stimuli of various kinds rather than the influence of age per se. Studies of primates seem to have revealed the impact on the circulation of emotional factors, which resemble those operating within ourselves. Among these are sexual frustration and restrained aggression. This may provide the missing link in our search for causes and mechanisms of human disease, for we may share with these animals enough of an emotional, or even conceptual, frame of reference to permit an experimental analysis of the ways of life that lead to disease and death. It will be of utmost interest to study their physiological parameters, such as lipids, blood sugar control, and hemostasis, under different psychological conditions, acutely, and over the course of time. For human physiology and pathophysiology must be conceived in terms of our genetic-environmental past and the meaning of diabetes in relation to starvation, of hypertension and of thrombosis in relation to fight, flight, and blood loss, and so forth. The secrets of the relative immortality of the female may be illuminated in this perspective.

The importance of the genetic element in coronary heart disease has always been emphasized by Paul White. Studies of family trees and on twins support this conviction. But there is always an interplay between genetic endowment and environmental demands. The strategy of prevention before overt disease or after an infarction is based on the concept of diverting an inherently unfavorable course into a more favorable one.

Much wishful thinking (in both directions!) has accompanied studies of the preventive effects of reduction of blood lipids, either by dieting or by the use of drugs and the preventive effects of the long-term use of anticoagulants and female sex hormones, and of giving up smoking. These endeavors, as well as those directed toward diabetic tendencies in coronary patients all belong to the strategic forces. It is my feeling that the evidence in favor of a definite effect of lowering blood lipids is getting stronger, not least through Leren’s careful 5-year study of 400 male survivors of myocardial infarction in Oslo, Norway. He found a significant reduction of the incidence of new angina pectoris and of recurrent infarction in patients under the age of 60, while sudden deaths, more frequent in patients with angina pectoris or an abnormal ECG, or both, were not affected. Giving up smoking has been shown to improve prognosis, and preliminary data concerning anti-diabetic measures in infarct patients with abnormal intravenous glucose tolerance appear promising. The effects of such interference with pathogenic mechanisms may, in principle, be of several kinds (fig. 5).

However, it remains to be seen whether the results will be substantial or only of marginal value, as appears increasingly to be true of long-term use of anticoagulants. Gofman and co-workers recently struck a sad note in this respect. Their feeling is that blood lipid levels are of little prognostic value in already manifest coronary heart disease in persons over 50 years of age, and that moderate reductions of blood lipids will have no influence on life expectancy beyond 55 years of age. Diet changes in order to have effect would have to begin early and be drastic. The lower the dividends become with age, the more imperative will it be to establish the predictive value of biological indices in young individuals and to act selectively on young candidates. This action may take the forms outlined in table 2. Although most preventive measures might be accomplished by natural remedies, it is probably more in line with our present technology to expect that artificial means will take the place of natural ones, and the use of drugs will be considered more acceptable than the cultivation of healthy habits.

The Tactical Approach

The remarkable and rapid developments of intensive cardiac care, culminating in the modern coronary care unit, have created in
**Preventive Measures**

<table>
<thead>
<tr>
<th>Natural</th>
<th>Artificial</th>
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<tbody>
<tr>
<td>1. Restore man's primitive functions:</td>
<td>Physical exercise</td>
</tr>
<tr>
<td>2. Reduce demands on metabolism:</td>
<td>Diet</td>
</tr>
<tr>
<td>3. Reduce demands on nervous system:</td>
<td>Equanimity and sleep</td>
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<tr>
<td>4. Counteract inappropriate natural responses:</td>
<td>?</td>
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<tr>
<td>5. Refrain from dangerous habits:</td>
<td>Quit cigarette smoking</td>
</tr>
</tbody>
</table>

Table 2

A short time an almost revolutionary situation with regard to medical tactics. Nevertheless, Oliver and co-workers in Edinburgh have recently warned against making too optimistic predictions of the results of such treatment in the front line of the infarct battle. High fatality rates still prevail in such places. Their own short-term gains, so far, in 400 patients were judged to be 5% of all patients, or 21% of the potential deaths.

A recent report from the London hospital stated that of 150 patients treated in a coronary care unit, 21 died and 129 survived, 10 of whom had been resuscitated from otherwise fatal arrhythmias. These figures are said to represent a reduction in mortality by 50%. However, other estimates, for example those from Grendahl's study from Oslo are much lower.

We do not know as yet whether the immediate gain will be maintained, or lost, during the following years (fig. 6).

This should probably become evident rather soon in view of the fact that myocardial infarcts recur most frequently within the first year or two after the preceding infarct (fig. 7). Such follow-up studies will determine the medical economy of coronary care units. Once established, however, they have probably come to stay, regardless of economy.

**Health Unmasked—or What?**

We have now completed two thirds of the twentieth century. During the last decade man has broken the genetic code, computerized a considerable part of management, industry, and science, conquered outer space, and landed digging television cameras on the moon. What has been accomplished with regard to coronary heart disease and myocardial infarction?

Although we have known for 10 years what should be done and what could be done, both

**Figure 5**

*Alternative effects of preventive modifying patho-genetic mechanisms in prevention of myocardial infarction (MI). (A) No effect. (B) Delayed infarction, little or no effect on longevity if patient survives. (C) Delayed infarction and correspondingly delayed death. (D) Delayed infarction and also more than correspondingly delayed death.*

**Figure 6**

*Estimates of survival after myocardial infarction.*

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with regard to strategy and to tactics—as is obvious to anyone ransacking "progressive" medical articles from 10 years ago—our moves have been cautious, hesitant, and slow. It is to your credit that they have been less slow here in the United States than elsewhere. More patients than ever are dying from myocardial infarction in most countries. Are we, then, attempting an impossible job, fighting the inescapable?

Some years ago, Maurice Campbell took the trouble to examine the apparent increase in death rates from heart disease in England between 1876 and 1959. He arrived at the conclusion that the very palpable increase—including deaths from coronary heart disease—could be accounted for by assuming that one quarter of younger persons whose lives had been saved from death by tuberculosis and other infectious diseases had ultimately died instead from heart disease at about the time other people die from such diseases. He also predicted that the increase in cardiac deaths would continue rapidly until 1967, more slowly to 1977, and finally would settle around a steady figure about 1990. This opinion has not remained unchallenged, but the test of the hypothesis will come with time. It cannot easily explain the different trends for men and women unless additional hypotheses are included. However, I think it is essential that Campbell's concept be carefully considered by statistical experts. It has been pointed out that the trends in young people as against those in old people are important for the validity of Campbell's claims. For if Campbell is right in principle, then the increase of coronary heart disease may be due to an unmasking of a fundamental biological tendency in man which presents itself in the very moment humanity (or part of it) is liberated from infectious diseases and other primitive challenges (such as diseases of infancy, nutritional disease, and starvation), and not necessarily because of exposure to the dangers of affluence. Is coronary disease
or myocardial infarction an expression of the true biology of privileged man, or is it a new pathological entity in man exposed to increasingly unbiological social challenges?

**What Are “Realistic Goals?”**

There is no doubt that a great many infarcts, and more or less sudden deaths are “premature.” But at what age does death cease to be premature?

A long revered expression, often emphasized by Paul White, is that our task is not only to add years to life, but life to years. This is true, too.

Addressing your Commission on Heart Disease, Cancer and Stroke, 3 years ago, President Johnson envisaged the need to adjust yourselves and your economy “to a life span and a work span for the average man or woman of 100 years.” Survival to a hundred years of age—how come?

The curves in figure 8 depict 200 years of gains in saving lives in my country: great advances in younger years, less in old age. It will take great strides to move the curve to the hypothetical position (of LBJ) on the right. But even greater may be the impact on society of caring for the tremendous, aged population that would result from it—a society with four, or even five generations living side

**Figure 8**

Two hundred years of gains in saving lives in Sweden compared with L.B.J.’s tentative aim for life.

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by side—at a time when automation reduces the need for human labor in our societies and “the population explosion” in other parts of the world is considered to be humanity’s greatest peril. As pointed out by two of the leading medical statesmen of our time, Irvine Page\textsuperscript{32} and George Pickering,\textsuperscript{53} we must accept death as a biological law: “It is the diseases which kill (that) make way for the new life. Without them none of us would be living as we are to-day.” This is a somewhat more diplomatic way of stating what one of Pickering’s predecessors as Regius professor at Oxford, William Osler, once expressed in a remarkable valedictorian address about the uselessness of men above 60 and comfortable ways to get rid of them.\textsuperscript{54}

Are there any means by which the numbers of premature myocardial infarcts and deaths could be reduced, without unduly delaying by those very means the long day’s journey into night? Nature rarely gives something for nothing: What other conditions will we, then, encounter on our passage from here to Eternity?

These are questions for which I have no answers. Perhaps such questions should not be asked.

And yet while we are striving with our low-fat diets and our automated defibrillators, in years to come the artificial heart and the transplanted heart may present us with problems not yet surmounted in the history of medicine and ethics. For whom and from whom? For one who has been involved in the problems of kidney transplantation, which is in principle a simpler matter than transplantation of a heart, the words of Jean Hamburger\textsuperscript{55} sound in the ear: “As a result of medical progress, our technical decisions may become easier, but moral problems, on the contrary, will be increasingly significant.” Medical science fiction has repeatedly shown itself to come true far earlier than expected. Sooner or later, transplantation of hearts may be here, and like all transplantation surgery, it will be expensive in its demands, of all kinds. The triumph of the surviving individual may have to be paid for by a new biological solidarity among men, a sacrifice, akin to that of the Aztecs, who performed the world’s most heroic cardiac surgery, but more meaningful—at least so we may hope.

In our civilization and our time coronary heart disease is probably the most important single determinant of the limits of human life, mediated through sudden death or through myocardial infarction. The biology of myocardial infarction is, therefore, part of the biology of human life, of body and mind.

Medical science is constantly pressing forward to conquer new grounds, to test new possibilities. But medical technology is not an aim in itself. It has to be handled to serve a true biological purpose, which also means a humanitarian one. For this we are responsible to society.

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The Grand Design

Science is the century-old endeavor to bring together by means of systematic thought the perceptible phenomena of this world into as thorough-going an association as possible. To put it boldly, it is the attempt at the posterior reconstruction of existence by the process of conceptualization.—Albert Einstein.
The Biology of Myocardial Infarction
GUNNAR BIÖRCK

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