Special Article

Background of the Prevention of Cardiovascular Disease

I. Nutritional, Infectious, and Alcoholic Heart Disease*

Oglesby Paul, MD

During recent years, I completed a biography of my former teacher, Dr. Paul Dudley White. As I was ending my labors, I talked with a representative from a large Boston publishing house who informed me frostily that the firm would not consider including the biography on their list unless the name of Paul White was a “household word.” The name of Paul Dudley White was indeed at one time a household word, but that time had gone. This experience reinforced my realization of how fleeting fame is; and in medicine, with its glorious history of intellectual and scientific achievements, many physicians and other scientists are ignorant of the distinguished leaders of even the recent past. This brief effort is, therefore, a reminder to the reader and a record of some of the important figures in the history of cardiovascular disease prevention, a field in which there have been many outstanding participants. Also, this review is evidence that cardiovascular disease prevention has been much more than just the prevention of coronary heart disease and hypertension. No attempt has been made in this review to be exhaustive, and most of the references here have been included because they represent early and not current milestones.

Beriberi

The following intriguing item appeared in the November 1880 issue of the Pacific Medical and Surgical Journal:

Some excitement has been lately produced among our medical men and in the community in general, by the introduction into our port [San Francisco] of a disease known in the United States hitherto only by stray scraps of history abroad.

The disease was beriberi, occurring among seamen on the Brazilian man of war Vital d’Olivera, and the “stray scraps of history” were chiefly Asiatic in origin.

Beriberi is said to have been recognized in China as early as 2697 BC, but it was unknown to physicians until AD 1645 when Jacobus Bontius described dry beriberi (i.e., neuropathy) in an essay published in Batavia (now Djakarta) in Java. The condition was identified in Japan, where it was called “kakke,” in 1717 by Tachibana Nan’ke of Kyoto. Colinus Rogers wrote about beriberi with serious effusions and edema in a report published in Edinburgh in 1808. A major prize-winning (500 rupees) essay on beriberi by John Grant Malcolmson appeared in Madras, India, in 1835, which described the dry and the wet forms of the condition and indicated that the two might be seen in the same patient. Malcolmson wrote on the basis of his Indian experience,

Of the morbid affections which occur in the course of this disease, the most important are those of the heart and pericardium, which give rise to much of the distress of the patient, are frequently the cause of sudden death, or more slowly, by inducing effusion either into the pleura or into the substance of the lungs, and lay the foundation, too often, for the most distressing class of chronic ails.

A comprehensive review by Anderson of the disease as encountered in Japan appeared in the St. Thomas’s Hospital Reports in 1876. Today, beriberi heart disease is recognized as a type of myocardiopathy in which impaired myocardial energy production occurs, associated with biventricular dilatation and hypertrophy, myocardial degeneration, and the clinical picture of heart failure with edema, responding to rest and thiamine administration.

The story of prevention starts with the work of Kanehiro Takaki of the Medical Department of the Japanese Navy. Takaki, who had received 5 years of training in London at St. Thomas’s Hospital, was concerned by the frequency of beriberi in the Navy. Between 1878 and 1880, there was in the Navy an average of 349 cases per 1,000 men, with a death rate of 8 per 1,000. Often, ships on long cruises were severely affected, and in 1882, one vessel with
a complement of 330 sailors was essentially disabled because of 195 cases of beriberi. After studying the problem intensively, which included experiments on dogs, Takaki concluded that diet must be the culprit (he decided that there was too much high carbohydrate food and not enough “nitrogenous elements”). On his recommendation, the daily allowance of food to seamen was changed to include fresh meat and fish and more vegetables and bread. Takaki found that “most of the men dislike meat as well as bread” because they had been accustomed at home to a predominance of rice, and he was forced to replace bread with barley, which received a better acceptance. This new diet, introduced on a trial basis first in 1884, was dramatically successful, and rapidly eliminated beriberi from the Japanese fleet and eventually from other navies.8

The precise constituent lacking in the diet, the absence of which resulted in beriberi, was not identified for another 40 years, but in this interval, much productive work was in progress. From 1890 on, the Dutch scientist Christian Eijkman working in Java produced experimental peripheral neuritis in fowl by feeding a diet similar to that producing beriberi in humans.9 He was slow to recognize that this represented not infection but a dietary deficiency, but for this important work, he shared the Nobel prize for medicine in 1929. Fraser and Stanton10 conducted controlled dietary studies in humans in Malaysia from 1907 to 1908, and concluded that milled white rice lacked an “essential” substance. It was Jansen and Donath11 who, using as an experimental model a rice bird (“bondol”), first isolated thiamine in pure form in 1926, describing it as “anti-beriberi”; and 10 years later in 1936, the vitamin was synthesized by Williams and Cline,12 thus permitting large-scale production.

The clinical cardiac aspects were only tardily investigated. In 1928, Wenckebach13 of Vienna with the essential assistance of Aalsmeer, who had lived in Java, reviewed the subject in a St. Cyres lecture in London, and showed that the administration of vitamin B (he did not specify thiamine) to patients with beriberi heart disease and congestive heart failure was an “immediate and usually complete success.” The following year, Aalsmeer and Wenckebach published a major report on the subject.14 Weiss and Wilkins15,16 of the Boston City Hospital authored in 1936 and 1937 a major report of cardiovascular disturbances in patients with vitamin deficiency states, particularly with deficiency of vitamin B complex. They observed the benefits of proper diet, vitamin B complex, and crystalline vitamin B1. They commented that

in this part of the United States, development of vitamin B deficiencies is apt to occur mainly in alcoholics, and less frequently in pregnant women, in persons without work and hence in poverty, in “food cranks,” and finally in patients with diabetes or with certain types of gastrointestinal disease. They thus identified the populations most at risk.

This combination of Oriental and Occidental biochemical, clinical, and epidemiologic study provided the necessary ingredients for the effective recognition and prevention of beriberi heart disease. Gradually, cereals were milled to lessen thiamine loss, and enrichment of these and other foodstuffs with thiamine was widely adopted in the Western world, although there was considerable resistance to this change in some countries in Asia and Africa that needed it most. Many patients with acute and most with chronic illnesses were given vitamin B supplements on a routine basis. Today, full-blown beriberi heart disease is rare in the experience of most physicians, although a subclinical thiamine deficiency state probably exists in special situations of poor nutrition or excessive thiamine excretion, including some of those conditions mentioned by Weiss and Wilkins.

Diphtheria

I recall a sad episode in the summer of 1942 during my internship in medicine. I had been assigned to spend a few weeks away from the Massachusetts General Hospital at the old Haynes Memorial Hospital, a Boston area center for contagious diseases. A 12-year-old girl, whose mother had refused to let her receive diphtheria toxoid for routine immunization, was admitted with typical acute diphtheria involving the tonsils and pharynx. In the course of a few days, and despite antitoxin and intubation, she went on to die with acute myocarditis. Five years later, I saw a young girl with complete heart block and severe congestive heart failure, the sequel of acute diphtheria that occurred several months earlier.

This dread disease was best described by the great French physician, Pierre Fidèle Bretonneau of Tours, who separated diphtheria (which he named diphthérîte) from other throat inflammations and croup in a series of publications beginning in 1821. From 1818 on, Bretonneau had seen many cases of diphtheria occurring in Tours in infants and in troops quartered in the city, and he developed a life-long interest in the disease. His classic work—a book of 540 pages with three elegant anatomic drawings—appeared in 1826 in which “by unsurpassed powers of clinical observation and generalization based on the accurate records and post-mortem studies of a large number of cases he formulated his doctrine of ‘diphthérîte’ as a specific disease.”17,18 He also described epidemics occurring at La Ferrière and Chenusson. He must have been a courageous person because in 1825 he dared to perform for the first time a life-saving tracheotomy on a young girl with laryngeal diphtheria. Bretonneau did not describe the cardiac complications, which were detailed much later by Bouchut and Labadie-Lagrange of Paris in 187219 and 187320 and by Rosenbach21 of Göttingen in 1877. My former chief at the Haynes Memorial Hospital, Dr.
Conrad Wesselhoeft,22 wrote a summary article on the subject of diphtheritic heart disease for the *New England Journal of Medicine* in 1940. In this review, he compiled benchmark statistics from three large contagious disease departments indicating at that time (which was just before the introduction of antibiotics) a mortality from diphtheria of 11%. Notable was the fact that 59% of these fatal cases were associated with myocarditis. The magnitude of the problem early in this century is shown by the 1920 data of the U.S. Public Health Service revealing that the rate of diphtheria cases that year was approximately 150 per 100,000 population, yielding a national annual prevalence of more than 150,000 cases.23 The deaths in the category “diphtheria and group” for that year amounted to 13,395.

The extraordinary achievements in conquering this tragic condition began when the causative bacillus was identified in 1883 by Klebs,24 who at the time was studying and teaching in Zurich. Then in 1884, Loeffler,25 working in Berlin under Koch, reported mucous studies in which he had cultured the bacillus from the throats of patients with diphtheria, had grown it on special media, had showed its pathogenicity for animals, and had even suggested that the organism may cause death by the elaboration of a toxin. Roux and Yersin26 showed in 1888 that in experimental animals this bacillus did indeed form a soluble exotoxin capable of producing fatal effects similar to those in human infection. Two years later in 1890, an antitoxin that could neutralize the exotoxin was discovered by Behring27 of Berlin, and it was first given to a human, an ill child, by Geissler on Christmas night of 1891. Commercial production of antitoxin soon followed, and the serum was given to many thousands of sick children and had life-saving effects, providing it was administered in the early phase of the infection. Active immunization with a toxin-antitoxin mixture resulted from the work of Theobald Smith28 of Boston in 1907, Behring29 in 1913, and others. However, it was the acceptability and the availability of diphtheria toxoid, developed through formalin treatment of toxin in 1923 by Ramon,30 which made possible mass immunization. As a consequence of these successive, important discoveries extending over 40 years, heart disease from diphtheria soon ceased to exist as a problem in the Western World, although foci of diphtheritic infection, chiefly cutaneous, lingered in a few areas in both developed and underdeveloped countries. In the years from 1980 to 1984 in the United States, no more than five noncutaneous cases of diphtheria were reported annually for the entire country, and there were no deaths.31

**Syphilis**

Syphilis has an ancient but uncertain history. It possibly existed in Europe in the Middle Ages and was confused with leprosy because some cases of that disease were highly contagious, were associated with sexual contact, had hereditary features, and may have shown some response to treatment with mercury. Also, it may have been present during the same period in the New World, which has been suggested by findings in skeletons of pre-Columbian South American Indians. What is unequivocal is that shortly after Christopher Columbus returned in 1493 to Portugal and Spain from his first voyage to the Bahamas, Cuba, and Haiti, syphilis became epidemic in Europe. Many writers have assumed that syphilis was brought back from the New World by infected members of Columbus’s crew. A large number of cases occurred, especially in the army of King Charles VIII of France in an expedition he undertook between 1494 and 1495 to conquer the Kingdom of Naples.32,33 In 1530, the Italian physician and writer, Girolamo Fracastora,34 wrote an epic poem titled *Syphilis Sive Morbus Gallicus* in which he described the appearance of this new disease in graphic terms:

I sing of that terrible disease, unknown to past centuries, which attacked all Europe in one day, and spread itself over a part of Africa and of Asia.

I will tell what concourse of influences, what occult germs have caused it, how it arose in Latium [Italy] at the time that the French armies rendered desolate that unhappy country, what reason caused it to be called the French disease.

Fracastora gave the disease the name of “syphilis,” after a mythical shepherd, Syphilus, who offended the gods, and as a consequence was struck down with a terrible illness. During the next 400 years, an extensive literature documented the clinical features of this common and serious malady.

Indication of the potential impact of syphilis on the cardiovascular system is chiefly to be found in the long-term study of patients by Caesar Boeck at the Municipal Hospital, Oslo, in the years from 1891 to 1910. Clark and Danbolt35 who have described this material wrote, “Nowhere in the world is there a more unique opportunity to learn what happens when early syphilis goes untreated.” The approximately 2,000 patients in the original series all exhibited primary or secondary syphilis and were essentially untreated because at the time there was no satisfactory therapy and because Boeck stressed the importance of natural acquired immunity. (Subsequent to the primary and secondary phases, a few patients did receive irregular treatment.) In 1925, Bruusgaard reported on a follow-up of 473 members of this population, and a further review of 953 from the original cohort was made by Gjestland beginning in 1948. Eight hundred and eighty-seven members of this latter group of 953, aged 15 years and over when first seen, were observed for over 40 years. During this period, 10.4% had developed cardiovascular syphilis. The manifestations included 4.6% with aortitis with aortic insufficiency, 3.0% with uncomplicated aortitis, 2.3% with saccular aneurysms, and 0.5% with coronary ostial stenosis. Some estimate of the pres-
ence of cardiovascular syphilis in the later era from 1910 to 1929 in the United States and United Kingdom is also provided by statistics compiled by Coombs\textsuperscript{36} from six large autopsy series, showing that cardiovascular syphilitic lesions were noted in 2–6\% of all postmortem examinations. Overall, the data indicated that for every 100 patients with untreated syphilis, 25–35\% would undergo a spontaneous cure, 25–35\% would remain latent, 10–15\% may have benign tertiary lesions of the skin, mucous membranes, and bones, 10–15\% would develop cardiovascular involvement, and 1–2\% would have central nervous system disease.\textsuperscript{37} Cardiovascular syphilis thus ranked as a major and not a minor sequel of the illness.

The development of a successful therapeutic and preventive program took a brief 42 years after the many centuries of observation in which therapy had been limited to mercury and, after 1836, potassium iodide. All but the last of the critically important contributions in this 42-year span came from European laboratories. First came the discovery of the phenomenon of complement fixation by Bordet and Gengou\textsuperscript{38} of the Pasteur Institute in Paris in 1901, leading in 1906 to the laboratory diagnosis of syphilis by Wasserman, Neisser, and Bruck\textsuperscript{39} of Berlin and Breslau. Next was the elegant demonstration in 1905 by Schaudinn and Hoffmann,\textsuperscript{40} also of Berlin, of the Treponema pallidum as the causative agent of the disease. Then came the first effective therapy—Salvarsan (number 606 in the series of arsenical compounds tested)—developed by Paul Ehrlich\textsuperscript{41} of Frankfurt in 1907 and used clinically for the first time by Ehrlich and Hata in 1909. The creator of this compound, Paul Ehrlich, was an extraordinarily gifted investigator of whom it could be said without hyperbole that “among medical scientists of his generation Ehrlich was probably the most original, stimulating and successful.”\textsuperscript{42} Finally, in 1943, Mahoney, Arnold and Morris\textsuperscript{43} of the U.S. Public Health Service showed the great effectiveness of penicillin in the treatment of syphilis.

The foundation was thus laid in a relatively few years for satisfactory diagnosis and treatment and with it prevention of major tissue complications. As early as 1938, Vonderlehr and Usilton\textsuperscript{44} could report that among syphilitic patients observed and treated by the Cooperative Clinical Group for 10–20 years, there were no instances of definite cardiovascular involvement. Of course, syphilis is still very much with us; in 1980, the U.S. Public Health Service statistics showed that 68,832 cases of syphilis had been reported, including 27,204 primary and secondary types.\textsuperscript{45} However, the experience of the last 50 years has shown that although we cannot prevent the acquisition of syphilis, we can prevent almost all of the major complications. Today, many physicians have never seen a patient with syphilitic cardiac or vascular disease.

### Rheumatic Fever

The term “rheumatism” was first used by Guillaume de Baillou in his Liber de Rheumatismo written in Paris in 1616, the year of his death. During the 18th century, both David Pitcairn of St. Bartholomew’s Hospital, London, in 1788, and Edward Jenner (of vaccination against smallpox fame) of Berkeley, Gloucestershire, in 1789, associated rheumatism with heart disease. Haygarth, in 1805, was evidently the first to use the designation “rheumatic fever.”\textsuperscript{46} During the 19th century, accurate delineation of rheumatic myocarditis, endocarditis, and pericarditis was gradually made, leading to the famous phrase of Professor Charles Lasègue of Paris, written probably in 1883: “acute rheumatism licks the joints, the pleura, even the meninges, but it bites the heart.”\textsuperscript{47} Cheadle gave three comprehensive lectures on rheumatic fever before the Harveian Society of London in 1889, describing the clinical features including cardiac involvement in detail and alluding to its causes only in one sentence that referred to “the rheumatic virus, whatever its exact nature.”\textsuperscript{48} That rheumatic fever did indeed frequently bite the heart was described by John R. Paul,\textsuperscript{49} who estimated in 1930 that there were then 840,000 patients with rheumatic heart disease in the United States; and of those who when first seen showed definite signs of rheumatic heart disease, 50\% would be dead on or before the age of 40 years. A diagnosis of rheumatic fever was thus bad news, and such was the respect it received in the United States that special institutions like the House of the Good Samaritan in Boston, Irvington House in New York, and La Rabida Jackson Park Sanitarium in Chicago were established to study, diagnose, and treat the disease.

The conquest of rheumatic fever began before a proper understanding of its causes and certainly before any specific preventive programs were underway. J. Alison Glover\textsuperscript{50} in his Milroy Lectures of 1930 delivered before the Royal College of Physicians in London pointed out the “cheering fact” that the mortality figures for rheumatic fever for England, Wales, and the United States had shown a substantial and steady decline from 1901 to 1928. He stressed the roles of droplet infection, crowding in housing, defective diet, and dampness, and he stated that “No disease has a clearer cut ‘social incidence’ than acute rheumatism, which falls perhaps thirty times as frequently upon the poorer children of the industrial town, as upon the children of the well-to-do.” He added the vivid line: “Every slum destroyed, every overcrowded tenement cleared, every unhygienic school building improved, every playing field provided, is a step forward.”

As Glover stated, the initial decline in the incidence of rheumatic fever would appear to have been in part, at least, attributable to improved social and economic conditions during the first half of this century, conditions that helped to reduce the
spread of respiratory infections particularly within the family or school group. The next step toward the goal of prevention came with better understanding of the causes of the disease. Throughout the years, various investigators believed that they had isolated bacteria responsible for acute rheumatic fever, including a *Micrococcus rheumaticus*, but such claims could not be verified.51 The splendid Hektoen lecture given before the Institute of Medicine in Chicago by Homer F. Swift in 1929 reviewed the possible place of streptococci in rheumatic fever and included the statement: “Practically all investigators in this field have been forced at one time or another to a consideration of the role of the streptococci in this disease.”52 The “allergic theory” linking streptococcal infection and subsequent tissue reaction was offered by Swift as a “reasonable explanation” of “divergent observations.” Two years later (1931), Alvin F. Coburn, while still a resident at the Presbyterian Hospital in New York City, published a monograph on *The Factor of Infection in the Rheumatic State*.53 He wrote,

Among immigrants from Ireland working in the hospital, the development of tonsillitis with *Streptococcus hemolyticus* was followed by rheumatic manifestations. This was likewise true in a number of instances of sore throats among the student nurses. A few rheumatic subjects on the wards while under observation developed pharyngitis with hemolytic streptococcus predominant on culture, and these were followed by severe recrudescences. In many of the acutely ill rheumatic patients admitted to the hospital wards during this study, throat cultures frequently demonstrated as hemolytic streptococci in large numbers.

He concluded that “the findings in this study justify the conception that *Streptococcus hemolyticus* is an important factor in the rheumatic state.”54

Soon, the etiologic role of the group A hemolytic streptococcus in the genesis of rheumatic fever was accepted, and there was prompt progress in prevention. It was the same Alvin F. Coburn who with Lucile V. Moore54 reported in 1939 that the administration of sulfanilamide to 80 young rheumatic patients during one winter season appeared to protect 79 of them from recurrent hemolytic streptococcal infection and recurrent rheumatic fever. The extraordinary value of sulfa drugs in the secondary prevention of rheumatic fever was rapidly confirmed. The group from the House of the Good Samaritan in Boston headed by Jones and Massell not long afterward (1947, 1948) found that penicillin administered by injection and also by mouth was similarly highly effective in prophylaxis.55,56 Meanwhile, valuable information had been obtained through careful investigations of epidemics of group A hemolytic streptococcal infection and rheumatic fever seen in young war-time populations. In 1953, the Council on Rheumatic Fever and Congenital Heart Disease of the American Heart Association promulgated its first set of recommendations for the prevention of rheumatic fever, including not only long-term administration of penicillin or sulfa drugs in patients with a history of rheumatic fever for secondary prevention but also treatment with penicillin of all acute hemolytic streptococcal pharyngeal infections, with the goal of primary prevention.57

From these beginnings came many successful community-based prevention programs in the United States and other industrialized countries, funded both from governmental and private sources. The result has been a remarkable and gratifying reduction in the incidence of acute rheumatic fever in the developed areas of the world (for example, a survey of the city of Baltimore for the period of 1977 to 1981 revealed only five patients had been admitted to hospitals with that diagnosis).58 Nevertheless, there have continued to be numerous cases of active rheumatic fever and newly acquired rheumatic heart disease in under-developed areas with poorer socioeconomic conditions and where similar preventive efforts are lacking. High prevalence rates have been found in the Philippines, India, Taiwan, and Thailand.59 Sporadic limited outbreaks have also been reported from time to time in the United States. But as Edward Bland60 has written,

Thus, as one contemplates the future and ponders why this remarkable demise of rheumatic fever has occurred in our work, there is hope for those in less favored regions where it is estimated that 15 to 20 million new cases per year are to be expected.

**Rubella**

It is extraordinary that a problem arising when two common conditions—pregnancy and rubella—coexist in the same person should not have been recognized until 1941, but such was the case. It is all the more extraordinary because the problem was often a very serious one. Rubella was gradually distinguished from measles and scarlet fever during the 19th century, finally achieving a respectable and separate identity when it was recognized by the International Congress of Medicine meeting in London in 1881. On that occasion, Dr. William Squire declared,

A century was required to complete the separation of measles from smallpox. Another century passed from Sydenham to Withering before scarlet fever was finally distinguished from measles... The century is fulfilled that should give autonomy to rubella.61

However, rubella did not subsequently receive much attention because it was regarded as a benign self-limited infection of little significance.

It was an astute ophthalmologist, Norman McAlster Grege62 of Sydney, Australia, who first reported to the annual meeting of the Ophthalmological Society of Australia in October 1941 on a series of 78 children with congenital cataracts (13 in his own practice). A definite history of rubella during pregnancy was obtained from 68 of the mothers. Forty-
four of the children were also considered to have congenital cardiac lesions. Soon thereafter, under the aegis of the National Health and Medical Research Council of Australia, a cooperative study headed by Charles Swan extended and confirmed this pioneer observation, in particular adding deafness and mental retardation to the defects seen in the syndrome and noting that 90% of the mothers who gave birth to infants thus categorized had exhibited rubella during the first 3 months of pregnancy. If ever any confirmation of these findings was needed, this need was met in 1964 during a major epidemic of rubella in the United States, when 448,000 cases of infection were reported that, with an infection requiring only optional notification, constitutes a gross underestimate of the true numbers. That year, it is stated that 4% of pregnancies were complicated by rubella, associated with 6,250 fetal deaths, 5,000 therapeutic abortions, 2,100 neonatal deaths, and the birth of more than 20,000 children with the congenital rubella syndrome.

A recent (1982) report from Great Britain of 407 women who contracted rubella during pregnancy and who did not have the pregnancy interrupted found that congenital defects were present in 80% of the infants whose mothers exhibited rubella during the first 12 weeks of pregnancy. The general experience has been that the most common congenital defect has been deafness, then congenital heart disease (especially patent ductus arteriosus and ventricular septal defect), and then cataracts.

The current situation is greatly improved. Rubella is no longer dismissed as an unimportant disease. It can be prevented. Several serologic assays have been developed to test for the presence of rubella antibodies as an indication of immunity. Live rubella vaccine is available, and it is general public health policy now to administer the vaccine to all persons 12 months of age or older unless there is serologic evidence of immunity. The vaccine-induced immunity is expected to be lifelong.

Statistics from the Centers for Disease Control in the United States reveal only 551 cases of rubella reported in 1986, with only 12 cases of the congenital rubella syndrome. Although these figures are incomplete, neonatologists tell me that the syndrome described by Gregg in 1941 happily is now rarely encountered.

**Alcohol**

The story of alcohol and the heart is complicated, largely because the early clinical interest was directed toward the thiamine deficiency that was seen in chronic heavy drinkers and that now appears less common. However, the early 20th century literature on heart disease included brief references to the impression of bad effects of an excess of alcohol on the heart muscle leading even to heart failure. Steel in 1906 spoke of the cardiac muscle failure of beer drinkers; Hirshfelder in 1910 wrote of heart muscle weakness from alcohol; Vaquez in 1924 referred to right heart failure from “alcoholic myocarditis”; and White in 1931 stated that large amounts of alcohol injured the myocardium. However, Leary, on the basis of his observations, suggested that alcohol may actually prevent arteriosclerosis. When Weiss and Wilkins in 1936 wrote their classic paper on cardiovascular disturbances in vitamin deficiency states, they cautiously stated, “The role of alcohol in the development of cardiovascular manifestations requires further investigation.” And in 1937, they stated, “There is valid evidence indicating that alcohol per se cannot be primarily responsible for the manifestations observed.”

Since then, Bridgen (1957), Evans (1959), Bridgen and Robinson (1964), and others have identified alcoholic cardiomyopathy as a frequent form of cardiac disease attributable indeed to the effect of alcohol per se on the myocardium. The impressions of the older clinicians have proved correct. Direct depression of cardiac output and myocardial wasting with variable observed tissue defects have been repeatedly reported in humans as the consequence of alcohol ingestion. Just how common this type of heart disease is remains uncertain, but it certainly is more widespread than is generally recognized. It is not limited to “Madison Street bums.” Alexander has stated that 2.5–3.0% of cases of “symptomatic heart disease” seen in a large metropolitan Veterans Administration hospital was the result of alcoholism. What Bridgen and Robinson wrote in 1964 probably remains true today: “We believe that an excessive consumption of alcohol is a causal factor in many patients with cardiomyopathy but we are unable to make an overall assessment of the frequency of the condition.”

Most of the patients seen have been men, the presentation is that of a dilated cardiomyopathy with varying degrees of congestive heart failure (although in some patients, arrhythmias are the presenting and dominant manifestation), and the prognosis varies from good in the earliest phases when total abstinence of alcohol is adopted to hopeless among chronic drinkers who persist with their fatal habit. It is important to note that among 50 patients with alcoholic heart disease seen at the National Heart Hospital in London between 1952 and 1963, there were 25 deaths—a mortality of 50%. Of course, these were serious hospitalized cases, but such are not rare. Mention should also be made of the less well-known fact that women who drink heavily during pregnancy may give birth to infants whose abnormalities may include atrial and ventricular septal defects, anomalies of the great vessels, and the tetralogy of Fallot.

Weisner has recently written, the modern treatment system for alcohol problems in the United States found its inception in a social movement that, beginning in the 1940s successfully pressed home the program that alcoholism is a disease, that the alcoholic can be helped and
deserves help, and that the provision of help was a public health responsibility for society.

Preventive efforts thus have involved medical and public health research, mass lay and profession education, group support through Alcoholics Anonymous (which began in 1935), special clinics including some industry-based, more widespread availability of rehabilitation facilities, legal penalties for the sale of alcoholic beverages to minors and for driving an automobile while under the influence of alcohol, political pressure from Mothers Against Drunk Driving, etc. It is clear that in the last 30 years the diagnosis of alcoholic cardiomyopathy has no longer been mysterious and that by its very identification assists in prevention. The astute doctor and the patient both can know what is there and what should be done to treat and prevent it. Regrettably, not all physicians recognize the condition.

That these and other efforts have had some impact on the total drinking problem is becoming apparent. Long-term alcohol consumption trends in the United States, after a steady rise through the 1960s and 1970s, have shown a slight downward deviation beginning in 1982. The 1984 per capita consumption of 2.65 gallons was the lowest since 1977. What has happened to the numbers of individuals developing or dying from alcoholic heart disease is unknown. There is no reason for true optimism yet because the whole drinking problem continues to be enormous—enormous medically, socially, and economically.

The alcohol-heart story includes one striking example of a totally successful prevention of serious myocardial disease due to a highly toxic element associated with one form of alcohol. This is an intriguing tale, involving the manufacture and sale during 1965 and early 1966 of beer containing cobalt in three areas: the Province of Quebec, Nebraska, and Belgium. The cobalt was added by the local breweries in the form of cobaltous chloride to prevent the beer from gushing and to stabilize and improve the appearance of the foam. Shortly after this change in the brewing process, indeed within 1 month, individuals who were heavy beer drinkers began to be admitted to hospitals in the three localities with an unusual type of cardiomyopathy. Twenty of 48 patients hospitalized in Quebec City with this condition died, as did 32 of 64 cases in Omaha, Nebraska. Prompt and intensive medical and public health investigation, particularly in Quebec by Morin and Daniel, pointed to the possibility of cobalt toxicity to the myocardium. The addition of cobalt to beer was promptly ended, and the epidemic ceased. This episode is a frightening instance of a new cardiac disease produced by a change in industrial technology and successfully prevented through prompt recognition of the cause of the disease. One can only wonder if there may be other substances in our food or drink also capable of damaging the myocardium but in a less florid and, as yet, unrecognizized form because of the subtlety of manifestation and difficulty of correlation of medical, public health, and industrial or agricultural aspects.

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O Paul

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