CORONARIES/ CHOLESTEROL/ CHLORINE

JOSEPH M. PRICE, M.D.

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It would seem that if cholesterol is the primary cause of atherosclerosis one could avoid heart attacks and strokes by avoiding foods high in cholesterol. Is this a valid statement? It is true that this point of view has many influential proponents. The most widely accepted theory holds that it is the excessive consumption of foods containing cholesterol that predisposes to atherosclerosis and its sequelae. As a result of the widespread acceptance of this cholesterol theory there has been an informal nation-wide campaign on for the last few years to discourage the "excessive" consumption of foods high in fats and cholesterol. As a corollary of this, researchers have discovered that certain types of fat, called "polyunsaturated" and found primarily in vegetable oils, have a vague antagonistic effect in the body on the "saturated" animal fats which are high in cholesterol. Witness the commercialization of this finding in ads and on TV—"So-and-so margarine, highest in polyunsaturated fats!"

Many experts in the field believe that if by diet, drugs or by other means one can lower his blood cholesterol levels he has significantly reduced his chances of developing a heart attack or stroke. Recently medical researchers have been experimenting with certain new drugs which lower the level of blood lipids (fatty substances) including cholesterol in the hope of delaying or eliminating heart attacks and strokes.

CHAPTER 6

IS THE CHOLESTEROL THEORY VALID?

"Well-organized ignorance often passes, unfortunately, for wisdom."

-ANON.-

In spite of the remarks made in the previous chapter, this belief in the possible avoidance of atherosclerosis by dietary manipulation is by no means universal. It has been emphasized by a minority of researchers that *no* direct evidence exists to prove that lowering of blood cholesterol by any method possible will decrease the risk of coronary heart disease or stroke, or even affect the underlying atherosclerosis. In fact, the U.S. Food and Drug Administration has taken this stand in its regulation of advertising to physicians of the new serum lipid (cholesterol and triglyceride) lowering drugs.

Although a causal relationship between cholesterol and heart attacks and strokes (the cholesterol theory of atherosclerosis) has not been

cient proportions to affect statistical tables about then. This would tend to imply that some environmental consideration of supreme importance in the etiology (causation) of atherosclerosis began to affect men in the early part of this century. (Remember that 10-20 years of increasing atherosclerosis precedes clinical manifestations.) This could not be cholesterol because cholesterol has been with man as long as there has been man.

I have just introduced the critically important concept that coronary heart disease and other manifestations of atherosclerosis were essentially unknown before the present century, and therefore these disease processes must have an underlying causal agent of rather modern origin. Are there any other historical facts which support this contention? Angina pectoris, the intermittent chest pains which usually imply narrowing of the coronary (heart) arteries by atherosclerosis and often-times precede full-blown heart attacks, was first described only as recently as 1768. And not only was it an exceedingly rare disease for the century and a half following this description but the cases described during this period were not necessarily due to an underlying atherosclerotic process—even today it is an indisputable fact that angina pectoris may rarely be the result of some other disease process such as syphilitic involvement of the base of the aorta or anemia of any origin. These non-atherosclerotic instances of angina pectoris are exceedingly rare, but then so was the syndrome of angina pectoris itself until some years into this century.

Now let us consider the heart attack itself. The coronary heart attack, which is almost exclusively the end result of atherosclerosis of the coronary (heart) arteries, was completely unknown until early in this century. Hard to believe? Yes, it sure is. I'll be the first to admit that most present-day physicians will immediately challenge this statement. But the pure and simple facts are that the first clinical description of coronary thrombosis (another term essentially synonymous with "heart attack") was made as recently as 1912; the great Canadian-American physician Sir William Osler did not mention the existence of the entity in lectures on heart diseases in 1910; and most amazingly the world famous heart specialist Dr. Paul Dudley White who treated President Eisenhower for his heart attack in the early 1950's did not see his *first* case of myocardial infarction (once again another term essentially synonymous with "heart attack") until after 1920!

Now that I have presented you with historical facts (which may be verified in any good medical library) debunking the concept that atherosclerosis and its clinical manifestations such as the heart attack and stroke are as old as mankind, what is there to say about the role of cholesterol?

Let me make the statement that while cholesterol may be one of many contributing factors to the development of atherosclerosis and its complications, it certainly is not sufficient alone—it is not *the* factor. The evidence for this point of view is substantial. We should first consider a finding closely related to the facts just described: through careful investigation of the literature it has been shown that in England at the end of the 19th century almost one-third of the population consumed dietary fats in amounts which must be considered excessive by present-day standards, and yet heart attacks and other evidences of atherosclerosis were non-existent! Another most impressive finding is that atherosclerotic heart disease has always been unknown in China—700,000,000 people and no heart attacks! And don't let anyone explain this away by saying that it proves that cholesterol *is* all-important because the Chinese consume very little fat. While the peasants have always lived on basically vegetarian diets, many of the traditional dishes of the higher social classes who have been able to afford them are nauseatingly fatty. Yet atherosclerosis has been as non-existent among the well-to-do classes as with the Chinese peasants.

While atherosclerosis and its consequences are practically non-existent among most primitive peoples of the world, there is no better example of high dietary fat intake coupled with the absence of atherosclerosis than seen among the Eskimos. The dietary fat intake of the Eskimos is simply hard to believe—a single adult may eat several pounds of blubber (about as saturated a fat as exists) at a sitting. This fantastic dietary pattern is followed for a lifetime—and yet no heart attacks or strokes from atherosclerosis. If there were no other evidence than this, any thinking man would still question the cholesterol (dietary fat) theory. Nevertheless, there is still considerably more evidence to raise doubts in our minds.

For instance, if you want something a little nearer to home we have that too. A few years ago there was an article in a popular magazine about a small town called Roseto in the hills of Pennsylvania. The people in this town, of Italian descent, tended to be obese and ate a diet abnormally high in animal fats and yet seemed to be immune to heart attacks as long as they did not move out of the community. A little more food for thought . . .

For those with a more classical medical research bent, one should mention the existence of the gerbil, a tiny Mongolian rodent who despite a fat-rich diet and high levels of blood cholesterol shows no tendency toward atherosclerosis.

A number of other similar findings exist; and several more of these will be mentioned when the explanation for all this is elucidated.

at all. Cholesterol is one of *many* contributing factors influencing atherogenesis and therefore dietary change *may* affect the disease process to at least a small degree, just as other factors like cigarette smoking, physical exercise, etc. may also have some influence on the matter. I *am* implying that even if a person changes his diet to one low in cholesterol and "saturated" fats he is not guaranteeing himself immunity against heart attack and stroke. Persons who do not smoke get heart attacks and strokes (although admittedly less frequently than those who do). The same statement is true for those who exercise regularly. And I am saying that the same is true for persons who would change their dietary habits.

By now I should imagine that you, the reader, have become a little uneasy. I have told you how heart attacks and strokes are the end result of a disease process called atherosclerosis. I went on to present the classical cholesterol theory of atherogenesis including the possibility of retarding the disease process by changing one's diet. But then I proceeded to produce some rather convincing evidence which would tend not only to discredit the theory but also destroy the one ray of hope with regards to prevention of the disease process by dietary manipulation. You are probably wondering where do we go from here.

Fortunately, this book was not written merely

to give the *coup de grace* to the untenable cholesterol theory. Have I come up with some original theory that all the best medical minds in the entire world over a period of many decades have overlooked?

Indeed I have.

And there are irrefutable scientific *proofs*, reproducible by anyone of average intelligence or more, to back me up.

In retrospect it is amazing how many separate observations point to the definitive solution of the problem and yet have been ignored. It is truly a tragedy that modern medical science in desperation has grasped onto the cholesterol theory (even to the point of ignoring and denying proven facts which would tend to discredit it) to the exclusion of all else. It is just inconceivable to most medical men that such incredibly important, widespread and basically untreatable diseases as heart attack and common stroke could have been essentially unknown less than 75 years ago. If this is true (as it is) it would mean that something has changed in the last 6-7 decades of human history but medical science has failed to see the light. In all fairness let me say that the reason for this shortsightedness will seem much more justified when it is realized exactly what the culprit is.

As implied in the above paragraph, the great stumbling block which has inhibited rational,

author believes it may be of some future interest, the original observation which led to this new theory of atherogenesis about to be expounded will be singled out and mentioned first. The absolute validity of similar future *in vitro* experiments as regards to extrapolation to living systems is of little importance. What was important is that it provided the nidus for a whole new stream of thought.

This was the seemingly insignificant and widely known fact that in the dairy industry very tenacious, yellowish deposits build up on milk utensils washed in certain kinds of germicidal solutions. It was evident that some chemical in the *water* used to wash the utensils reacted with milk or some component of milk to produce a deposit. The biological analogy should be obvious.

Have there been any experiments reported in the medical literature relating water and elements contained in it to coronary heart disease? It has been shown most conclusively that the harder (higher concentrations of ionized minerals) the drinking water the *less* the incidence of coronary heart disease; and that hardness of drinking water is related to no other known diseases *except* heart and vessel disease!

By now I assume that the reader will have taken the not-too-subtle hints and come to the conclusion that there must be something in *drinking water* that is the culprit. In one short,

succinct sentence the cause of atherosclerosis and resulting heart attacks and strokes is none other than the ubiquitous *CHLORINE* in our drinking water!

It is, in my opinion, certainly one of the greatest paradoxes of recorded history that one of the very same public health measures which have been primarily responsible for the great increase in statistical life expectancy in the Western world should also unsuspectedly be responsible for many of the chronic disorders of later life.

It should now be manifestly apparent why no medical scientist has ever even for a fleeting moment entertained the truth. Chlorine is a classical "sacred cow" of modern medical science. Is it conceivable that something of such obvious and wonderful utility, so widely used, and with no apparent acute side effects could be responsible for heart attacks and strokes? Is it not incredibly difficult to believe that millions upon millions of dollars and incalculable hours of time have been poured down the drain by thousands of medical research workers busily and eruditely engaged in finding answers to the wrong questions? And although the two chemicals are totally unrelated, anyone opposing the use of chlorine would be immediately disregarded as an anti-fluoridation-type "nut". What medical scientist would dare risk tarnishing his reputation and grant-collecting future by espousing a

theory which would automatically be tainted in the popular mind (due to the popular misconception nearly equating chlor*ine* with fluo*ride* and the smear-campaigns against anti-fluoridationists)?

This is, it may be recalled, not the first time in modern medical history that a presumably innocuous chemical material has been indicted as an agent of serious disease. Some years ago blindness caused by a disease known as retrolental fibroplasia was distressingly common among children, especially those born prematurely. Only some years after this form of blindness had become prevalent was it discovered that high concentrations of life-supporting oxygen in incubators caused the disease. Today new cases of this form of blindness are totally unknown; unfortunately for the victims of that era of ignorance the blindness is irreversible.

You will, of course, now want to know on what basis can I make the sweeping statement that *chlorine* in drinking water is the greatest crippler and killer of modern times, i.e. the prime causative agent of atherosclerosis and its end results, the heart attack and stroke.

Let us retrace our steps and see if this new chlorine theory will explain satisfactorily the same facts used in tearing apart the classical cholesterol theory.

Great emphasis was put previously on the concept that the heart attack was essentially an

unknown entity before about 1920 and did not become of sufficient significance to affect mortality statistics until about 1930. How does this correlate with the use of chlorine in drinking water? Experimental use of chlorine to "purify" water supplies began in the late 1890's. Chlorination gained relatively wide acceptance in the second decade of this century and in the third decade (1920's) it was found that satisfactory killing of organisms was dependent upon a *residual* of chlorine in the water above the amount necessary to react with organic impurities. When it is remembered that evidence of clinical disease from atherosclerosis takes 10-20 years to develop, it becomes evident that there is a correlation between the introduction and widespread application of chlorination of water supplies and the origin and increasing incidence of heart attacks that is exceedingly difficult to explain away.

In light of the chlorine theory just presented we can now understand why there were no heart attacks in England during the last century despite a significant portion of the population consuming diets high in fat. We can understand why the Eskimos, whose diet is composed in the main of highly saturated animal fats, are immune to coronary heart attacks and other manifestations of atherosclerosis. Why heart attacks have been totally unknown among the Chinese

and most all primitive peoples. Why the inhabitants of Roseto, Pennsylvania have no heart attacks unless they move to another community. And we can understand why the gerbil shows no tendency to atherosclerosis.

There was no chlorinated drinking water in England during the last century. Eskimos may consume huge quantities of dietary fats, but their drinking water is pure melted snow. Chlorine in the drinking water is unknown among primitive peoples. The Chinese are basically a poor ignorant race who spread their sewage on the ground and get worms in their guts from drinking contaminated water and eating filthy food. We in the Western world are more civilized —we take our sewage and dump it into our rivers. We then drain it into our water supply, strain it and inject chlorine into it. We don't get worms in our guts but we sure do get something else! The inhabitants of Roseto drink water straight from flowing mountain springs, but when they move to the big city and drink chlorinated water like all the other city dwellers they are subject to the same retribution.

Although not mentioned before: the Japanese who normally have a very low rate of heart attacks are no different from other people when they move to Hawaii—and drink chlorinated water; the Masai tribesmen of Kenya have almost no heart disease although they eat at least as much cholesterol as most Americans—but

drink no chlorinated water; coronary heart disease is unknown among a group of 500 poor Irish farm workers studied by famed Dr. Paul Dudley White while being widespread among their chlorine-drinking brothers in the United States. And contrary to popular belief, high level business executives (supposedly under much stress) have a statistically lower incidence of heart attacks than their subordinates. In America when an executive reaches the highest echelons not only does he receive a key to his own "washroom", but while at the office drinks non-chlorinated bottled water.

The lowly gerbil drinks no water at all, instead manufacturing all the water it needs from the dry food it eats, and therefore escapes the end results of chronic chlorine poisoning.

We can understand even the intriguing, apparently unrelated and sometimes apparently facetious facts and correlations presented before. The documented lower incidence of coronary heart disease in areas with hard water could possibly be explained by postulating chemical reactions between free chlorine, an extremely active chemical, and the ions which cause hardness of water resulting in biologically innocuous chlorides. (This is not to exclude the possibility of some other, more complex biological mode of action of the hard water ions.) Even the strange and apparently facetious correlation between the number of telephones per unit of population

and the cardiac death rate can be explained quite logically and seriously on the basis that chlorinated drinking water and telephones are both products of modern civilization, both became widespread in the early decades of this century and both are most prevalent in urbanized areas.

There is one other finding that we should not forget to mention. During the Korean War autopsies were performed on otherwise apparently healthy soldiers killed in battle. In an article in the Journal of the American Medical Association it was reported that among the soldiers whose average age was 22.1 years over 75% showed some gross evidence of coronary arteriosclerosis. These results have been widely discussed with the usual conclusion being that coronary artery disease is far more common and extensive than previously suspected, especially in young men. In light of my new theory I most strongly question this conclusion. If you ask any man who served in that war he will tell you that the water in Korea for our soldiers was so heavily chlorinated for sanitary reasons that it was almost undrinkable. Incidentally, discussions with Korean physicians reveal that heart attacks are almost totally unknown among their fellow countrymen. Koreans drink un-"purified" water. (Recent similar postmortem studies performed on U.S. casualties in Vietnam have shown an even higher incidence of coronary disease. Being

a miserably hot country the average GI cannot avoid drinking large quantities of incredibly highly chlorinated water. The water made available for soldiers is required to contain a *minimum* of 5 parts per million of chlorine residue. But because only a minimum standard is required by regulations oftentimes the water is chlorinated at a level several *times* that, levels quite comparable to that in the animal experiments to be described. ("Results" on the soldiers seem to be the same as in my experiments.) Apparently there is a direct *causal* correlation between the amount of chlorine ingested and the speed and degree of development of atherosclerosis!)

It is very interesting that clinical material (specimens obtained at operation or autopsy) has shown atherosclerosis in synthetic vascular grafts (artificial Dacron fabric arteries) in humans. One cannot help but think of the analogy between this and the deposition of milkstone on smooth rubber or metal surfaces. In both we have the flow of fat and cholesterol-containing fluids over surfaces in the presence of chlorine, with resultant surface deposits. If this analogy holds true, it would be powerful evidence in favor of the so-called "encrustation" theory of the mechanics of atheroma formation, the main differences seen in the artery lining being due to the body's reaction to the deposit. One should note that the encrustation theory of-

fered no explanation for the basic etiology of atherosclerosis. All it did was to try to explain where the deposit came from, not *why* it formed.

Some of the previous examples suggest strongly that dietary consumption of fats bears little relationship to the development of atherosclerosis. This is *not* to say, however, that there is no connection whatsoever between dietary fats, hypercholesterolemia (high blood cholesterol levels) and atherogenesis. To avoid later misinterpretation let me emphasize that atherogenesis involves a system of multiple etiology. This is to say that such factors as diet, exercise, smoking habits, etc. *are* of some significance under appropriate circumstances.

In any system of multiple etiology the primary agent (in atherogenesis the primary agent is chlorine) is only one cause. But it must be an *essential* cause. For example, it would be inconceivable for tuberculosis to occur in the absence of the tubercle bacillus. Nevertheless it is generally recognized that there are multiple other causes than the primary agent (the tubercle bacillus) in the production of clinical tuberculosis. Thus the primary agent must be an essential cause (tuberculosis cannot occur under any circumstances whatsoever in the absence of the bacillus) though not necessarily a sufficient cause (clinical tuberculosis may not occur even in the presence of the bacillus, e.g. tuberculin positive

skin reaction in a clinically healthy person). Likewise in the process of atherogenesis chlorine is the essential agent (atherosclerosis cannot occur to a clinically significant degree in the absence of chlorine regardless of diet and other contributing factors) though it is not necessarily a sufficient cause (e.g. normal premenopausal women do not develop atherosclerosis even if they are exposed to chlorine and the other factors).

on human subjects, at least in a society where we do give lip-service to the inherent worth of human beings and human dignity. Thus we must turn to animal experimentation.

Significant atherosclerosis is almost totally unknown among wild animals, just as it is among primitive peoples. Under special conditions, however, animals of certain species have been made to develop at least some of the early plaques of atherosclerosis in their arteries. With rabbits it is necessary to give the bunnies diets high in added cholesterol (quite obviously a grossly abnormal situation for a normally vegetarian animal). To use the dog a diet high in cholesterol will not do, by itself. In order to induce atherosclerosis in the canine species it is necessary first to destroy the animals' thyroid glands either by surgical removal or by administration of radioactive iodine.

The one other animal species extensively used in the experimental production of atherosclerosis is the lowly chicken. Some researchers maintain that the chicken is prone to the *spontaneous* development of lesions of atherosclerosis, i.e. even when given an apparently normal diet. As will soon be shown this is not true at all, but nevertheless the chicken has been shown to be an excellent experimental species for the purpose at hand. Indeed, it has been widely accepted that the chicken is as good as or better than any other animal for research in atherosclerosis in

procedures designed to help analyze pathogenesis and/or therapeutic value of different regimens and substances.

With this knowledge in mind the author set up a controlled experimental situation using the chicken as the experimental species.

There were two general phases to the proof of the chlorine theory. In the first, 100 day-old cockerels were divided into two groups of 50 each. With the understanding in mind that what was to be proven was that chlorine is the essential cause of atherosclerosis though not necessarily a sufficient cause, the two groups were set up with known contributory causes acting on both, the only difference being the presence of chlorine—the presumed essential cause—in the drinking water and mash (food) of the experimental group and absent from the food and water of the controls.

The male of the species was chosen in the knowledge that just like human beings it is the male which is primarily susceptible to the development of atherosclerosis. All other experiments on atherosclerosis using the chicken which have been reported in the medical literature have used cockerels for the same reason.

Both groups were placed on a cooked mash consisting of about a 1:1 mixture of corn and oat meals with about 5% low-priced oleomargarine added. Pure distilled water was used exclusively. Chlorine was added to the drinking water and

THE CHARLES E. BLACK CLINICAL LABORATORY
112 WEST ALLEGAN STREET
LANSING 8, MICHIGAN
TELEPHONE IV 9-8044

CHARLES E. BLACK, M.D.
DIRECTOR

PATHOLOGY
AND
DIAGNOSTIC SERVICES

Chicken Aorta

Number: OA-167-67

Date: July 28, 1967

GROSS EXAMINATION:

The previously opened aorta submitted shows a typical raised yellow atheromatous plaque of the aorta. The entire specimen is submitted for microscopic examination.

MICROSCOPIC EXAMINATION:

The sections show a large typical appearing atheromatous plaque of the aorta. The plaque involves the entire wall of the aorta. Areas of liquefaction necrosis and degenerative change are noted in one area of this plaque. No calcium deposits are noted. The adventitia of the aorta shows active inflammatory changes, chiefly lymphocytes.

DIAGNOSIS:

1. Atherosclerosis of the aorta of the chicken with typical atheroma formation. An intimal plaque is characterized by the focal deposition of lipids and fibrosis in the subendothelial connective tissue of the intima. Early, the atheroma is rich in lipid and filled with a soft pink staining grumous material.
The plaque may become fibrotic or calcified. It may ulcerate into the lumen. The intact, or more often the ulcerated plaque predisposes to mural thrombosis. Atherosclerosis, then, causes marked deformity, narrowing and occlusion of arteries. It is responsible for ischemia, atrophy and infarction of dependent structures. As focal endothelial injuries, the atheromas weaken arterial walls and potentiate rupture or aneurysmal dilatation.

2. Atherosclerosis is the most common varient of arteriosclerosis. On this basis, unless the other forms of arteriosclerosis are specifically designated, the term arteriosclerosis is used as synonymous with atherosclerosis. As mentioned above, atherosclerosis is characterized by the formation of atheromas, "focal intimal lipid deposits particularly cholesterol." One theory involves the concept that injury to the endothelial lining of arteries underly the abnormal accumulations of lipid within the intima. It has been shown experimentally that a variety of macro-molecular substances may injure the endothelium and increase its permeability to lipo-proteins. Atheromas tend to occur in arteries at points of stress. Atheromas likewise tend to occur around the mouths of vascular branches and bifurcations including other points of stress. It is believed that atherogenesis is related to the diet of man and particularly to the level of lipid or cholesterol intact. The plaques contain granular acidophilic, lipid-rich debris and crystalized needle-like spicules of cholesterol. A low grade inflammatory reaction is accompanied by the accumulation of a scant number of lymphocytes, such as is observed in this specimen.

3. Active aortitis involving mainly the adventitial outer coat.

Charles E. Black M.D.
Charles E. Black, M.D.

CEB/dlk

mash of the experimental group in the form of chlorine bleach (disinfectant), about one-third teaspoonful per quart of water. This highly chlorinated water was first given to the experimental group at twelve weeks of age.

The results were nothing short of spectacular! Within three weeks there were grossly observable effects on both appearance and behavior. The experimental group became lethargic, huddling in corners except at feeding time. Their feathers became frayed and dirty and the cockerels walked around with their wings hunched up, their feathers fluffed up as if they were always cold (the experiment was performed in an unheated barn in winter), their pale combs drooping. This appearance is most suggestive of symptoms resulting from clogging up of the micro-circulation.

Meanwhile the control group was the epitome of vigorous health. They were much larger in size than the experimental group, active, quarrelsome, vigorous appearing with smooth, clean, shiny feathers and bright combs held up erectly.

No less remarkable was the gross appearance of the aortas. The abdominal aorta (the place where atherosclerosis is known to occur in chickens) of all of the cockerels dying after four months were carefully examined. In more than 95% of the experimental group grossly visible thick yellow plaques of atherosclerosis protruding into the lumens were discovered! These

chickens were noted to have an extremely high apparently spontaneous death rate and common findings on examination of the carcasses were hemorrhage into the lungs and enlarged hearts. Although no blood pressure readings were taken, these findings are suggestive of gross arterial hypertension.

At seven months there were so few experimental chickens remaining alive that the survivors were sacrificed, with identical findings. At the same time one-third of the apparently healthy control group was also sacrificed with not one abnormal aorta found!

Although these results seemed conclusive, it was decided to repeat the procedures by taking the healthy control animals of the first experiment and dividing them also into an experimental group receiving chlorine and a control group. Once again the roosters receiving chlorine showed fantastic gross changes in appearance and behavior within three weeks. The first change noted was a remarkable paleness of the combs. Instead of bright fiery red the combs became nearly orange in color and soon began to droop. These changes were shortly followed by the ones described for the original experimental group and as expected gross atheromas of the aortas were found on examination within three months.

To summarize, in both experimental groups gross changes in appearance and behavior, most

likely explicable by postulating obstruction of the micro-circulation, were evident within a few weeks in the fowls receiving chlorine; followed by gross atherosclerotic lesions of the aortas evident within a couple of months. Control groups treated in an identical manner except for the absence of chlorine remained healthy and vigorous, grew well, and showed no evidence of either atherosclerosis of the aorta or symptoms of possible obstruction of the micro-circulation.

As a side comment, it should be mentioned that the so-called "spontaneous avian (chicken) atherosclerosis" reported in the literature was not spontaneous at all. These experiments, as well as most others on dogs and rabbits, were performed in urban university centers using city water which inadvertently contained chlorine. It has been noted that animals in our zoos are starting to show evidence of atherosclerosis. Once again the culprit is the ubiquitous chlorinated city water.

CHAPTER 10

PRACTICAL SUGGESTIONS

About now the reader of this book is getting more than a little concerned about the water he has been drinking and will drink in the future. Go to the water tap and get yourself a big glass of water. Now hold it up and look at it. What does it mean to you? Perhaps you are a little angry—it is the insidious poison contained in that very glass of water which has been responsible for so much suffering and death of modern times. More men died of coronary heart attacks alone in the last two years in the United States than have been killed in our many wars since our country was founded. Have you ever seen an active, vitally alive human being reduced to a state little more than that of an inanimate vegetable by a stroke?

But rather than being upset, you should be over-joyed. Soon the coronary heart attack and

stroke should be no more / mere uneasy memories of a blind and ignorant past. Ever since the conquering of infectious diseases earlier in this century by the use of public health measures (primarily sanitation) and in more recent years the use of antibiotics, the chronic diseases of the aging have come to the fore. But now two of the important "degenerative" diseases have been conquered. Now our citizens may truly enjoy the joys of "life in the years".

What are you going to do about that glass of water? Let me re-emphasize that the development of atherosclerosis is a slow process under ordinary circumstances (e.g. ordinary city water; the heavily chlorinated water given to our combat troops fighting in the stinkholes of the world is another and much more serious matter). There is no need to panic—for most persons the drinking of chlorinated city water for a few more weeks or months will be of little significance.

But what *should* you do now? You must insist, to all persons in positions of power and by all means possible, that chlorination of all water supplies be stopped permanently as soon as is humanly possible.

What can be used to replace chlorination as a means of "purification" of water? While there are several distinct possibilities, e.g. other chemicals (but would these show some terrible side effects in 20 years?), the process most likely to re-

place chlorination is the passing of thin sheets of water under ultra-violet lights. It has long been known that water flowing through streams and rivers is purified by nature using a combination of friendly bacteria and sunlight, which contains ultra-violet rays. In a similar manner it has been found that pathogenic bacteria in water passed under strong ultra-violet lights are killed, most probably by minute amounts of free oxygen released from the water by the light rays. In addition, water treated in such a manner will kill or impede bacteria added subsequent to the treatment.

Now, a most vitally important point must be considered. Is the use of ultra-violet light systems a practical possibility from an economic standpoint? Does it cost too much to be feasible? Most wonderfully, recent developments in the field of purification of water by means of ultra-violet light have brought down the cost of treatment to no more and potentially less than by means of the deadly chlorine. In addition, with the use of ultra-violet light there is no chemical taste or odor to the water, no storage or mixing of dangerous chemicals, no long storage period required and a new treatment unit is not any more expensive to install than many other types of units. The ultra-violet treatment unit is reasonably compact, is of high capacity and requires a minimum of maintenance.