IT IS attractive to contemplate that when and if the prevention and reversal of atherosclerosis is achieved, most cerebrovascular disease will fade from the medical scene together with coronary and peripheral arterial disease. Unfortunately we have little to be complacent about in this regard. Most of the so-called epidemiologic studies dealing with the relationship of diet to atherosclerosis are inadequate and at best only suggestive. For example, the studies showing that death from atherosclerotic heart disease is rare in the Bantu natives have been largely based on autopsy findings and hospital records. The results at first thought appear valid, but to be valid they must be accurate and meaningful. Certain little-known factors may, however, render their interpretation doubtful. For example, transportation for natives in Bantu country is often difficult and the distances are great. This could mean that many patients suffering sudden death, of which cardiovascular diseases are the commonest cause, fail to reach the hospitals, whereas those suffering from diseases that do not kill suddenly do reach the hospitals. Such a situation may well affect the hospital and autopsy statistics. The errors are compounded on a national level by the fact that the cause of death of a Bantu dying of natural causes need not be established or recorded under the law. Recently Laurie and Woods\(^4\) published autopsy data that cast doubt on the conclusion that atherosclerosis is rare in the Bantu. On the basis of just under 2,000 autopsies they conclude that atherosclerosis is common in the aorta and that it is fairly common in the cerebral vessels. The significance, in terms of the development of atherosclerosis, of the fact that these natives exist on a diet low in saturated fats is thus open to question. If atherosclerosis is in reality not rare in the Bantu natives, then what is the significance of the reported low serum cholesterol readings obtained from this group? It has been claimed that the Japanese have a low incidence of deaths from heart disease, but a fairly high incidence of cerebral vascular disease. This illogical paradox may well be explained by the long-standing custom among the Japanese of recording the cause of sudden deaths as apoplexy. Some national populations arranged according to the amount of intake of saturated fats may show a parallel relation between this factor and death from heart disease. However, as Yerushalmy and Hilleboe\(^2\) have pointed out, 22 countries, selected at random, fail to confirm this relationship; indeed they found the relationship of the intake of protein to deaths from heart disease was closer. Because of the weakness in such statistics, it behooves us to maintain a healthy scepticism and to lean more heavily on solid statistical material such as that emanating from the Framingham Study of the U. S. Public Health Service.\(^3\) In most studies the great emphasis has been on atherosclerosis of the coronary arteries and the aorta. It is to be hoped that before this study is terminated, the details of the cerebral vascular and other peripheral vascular pathology will be as complete as those relating to the coronary arteries and the aorta.
It has now been shown as a result of the work of Ahrens and his co-workers8 and Maluros9,10 that the serum cholesterol level of subjects can be reduced by sharply reducing the intake of saturated fats and substituting for these unsaturated fats with high-iodine indices and linoleic acid content. Whether this will have any effect on the further progression of atherosclerosis, or, more specifically, the incidence of strokes, is unknown. Many patients have been indulging themselves with their usual fat intake and taking in addition doses of safflower oil in the illusion that this will in some way reduce their cholesterol level.11 Double-blind studies in our laboratory with 24 young men who continued on their regular diet but took the maximum dosage of safflower oil recommended, and in most cases tolerated, (15 ml. 65 per cent safflower oil emulsion 5 times daily—total 75 ml. a day), failed to show any decrease in the serum cholesterol levels as compared with controls using an inert placebo preparation.

Another aspect of fat ingestion that is pertinent to our present problem is the question whether or not a large fat meal will increase the clotting tendency in the blood, and, by inference, the risk of thrombosis. Waldron and Duncan12,13 and Buzina and Keys14 have obtained results which they think justify the position that such a relation exists. Two teams of workers in our own laboratory have failed to confirm this finding.15 Sheehy and Eichelberger16 have recently published results with a more objective method of thrombelastography that show no significant increase in clotting tendency following the ingestion of a high-fat meal. Doubtless the last has not been heard on this subject.

Although hypertension is a very important factor in the production of strokes due to hemorrhage, and although we now have potent drugs for the reduction of blood pressure, the situation is still not ideal. After a massive cerebral hemorrhage it is too late to accomplish much by lowering the blood pressure. Therefore, our attention must be directed at trying to prevent such a critical development. However, this should be undertaken with great caution. Too rapid or profound lowering of the blood pressure may result in decrease or cessation of blood flow through narrowed atherosclerotic arteries with the subsequent development of an ischemic pattern and signs of a stroke. This may be transient, or it may be irreversible and thus, once more, the physician is placed on the horns of a dilemma. Renal dysfunction on the same basis may further complicate this picture.

The diagnosis of the location, size, and exact pathologic status of lesions producing the clinical manifestations of strokes is admittedly difficult, but often it is clear enough to proceed with suitable therapy. The excellent descriptions of carotid and basilar artery thromboses by Millikan and co-workers17-19 and by Fisher20 have helped to clarify this picture. Occlusion of the cerebral arteries and other vessels penetrating the brain present a greater challenge. A persistent problem, now more important than ever before, is the degree of hemorrhage that may be present in an infarcted area due to a thrombosis or embolism. In the past, therapy for strokes presented no problem. There was none. Today, however, with the advent of new therapeutic agents, notably anticoagulants, enzymes such as plasmin, and new surgical techinie, the clinician can no longer treat his patient with "skilful neglect," but is forced to undertake most careful study and analysis to determine whether the new therapeutic approaches may be helpful or even life-saving. These advances have resulted in much greater interest and improved general care, but have they improved the outlook for the patient? Even at this early stage, for certain individual cases, the answer can be given in the affirmative. For example, there is substantial evidence that the anticoagulant drugs are effective in decreasing the risk that transient episodes due to partial occlusion of the carotid or basilar arteries will progress to massive and permanent neurologic deficit. Accumulating evidence also indicates that after the first stroke due to either thrombosis or embolism the risk of additional strokes and other thromboembol-
ic complications will be markedly diminished by the long-term use of anticoagulants.

The value and the risk of these drugs during the acute phase of a stroke due to occlusion of the cerebral vessels and other branches penetrating the brain tissue are presently under intensive study in several institutions. As implied above, the amount of bleeding into the infarcted areas is difficult to evaluate. This aspect of the problem requires further study. The use of enzymes to hasten the dissolution of thrombi, such as fibrinolysin (plasmin), has opened a new approach that is worthy of intensive study. Used together with anticoagulants, they may offer a potentially important forward step.

Surgery is also on the march. Striking results have occurred by tapping subdural or extrameningeal hemorrhages, but so far the results of surgery for massive brain hemorrhage have been disappointing. However, with the use of hypothermia, this approach has, on a few occasions, been encouraging. Surgery for intracranial aneurysms and arteriovenous anastomoses has occasionally been successful, but like surgery for similar conditions elsewhere, the long-range results are often unsatisfactory. Among the most encouraging new steps are the use of synthetic prostheses to replace segments of carotid arteries occluded in sharply localized sites, and thromboendarterectomy of these same vessels. When one recalls the status of heart surgery 15 years ago, great advances in vascular surgery of the brain seem clearly visible on the horizon.

After the acute phase of the stroke, 2 main objectives constitute the responsibility of the physician: (1) Rehabilitation from the damage already suffered. This goal is being widely and intensively pursued, but carefully controlled evaluation of the results has, as yet, not been carried out. The question to be answered is whether or not the expense and work involved in such a program really increase the rate and degree of improvement, or whether this is controlled by the motivation of the patient himself, which, in turn, may be dependent on the home and work life that he has to face in the future. A controlled study to answer this difficult but important question is in progress. (2) Prevention of future strokes that often produce more serious and permanent or fatal damage. The judicious use of anticoagulants and antihypertensive agents, serious efforts to control cardiac rhythm, the avoidance of obesity and excessive physical and emotional stress, all contribute to such a program. Formerly the physician could rest his ears after the initial crisis had subsided. Now his responsibility continues during the subsequent history of the patient. With these and other technics opening up new vistas, we can anticipate advances which, however, are likely to require ever more diligence and skill by the physician.

Irving S. Wright

REFERENCES


Letter of Jenner to Heberden

"The importance of the coronaries, and how much the heart must suffer from their not being able duly to perform their functions (we can not be surprised at the painful spasms), is a subject I need not enlarge upon, therefore shall just remark that it is possible that all the symptoms may arise from this one circumstance.

"As I frequently write to Mr. H. I have been some time in hesitation respecting the propriety of communicating the matter to him, and should be exceedingly thankful to you, sir, for your advice upon the subject. Should it be admitted that this is the cause of the disease, I fear the medical world may seek in vain for a remedy, and I am fearful (if Mr. Hunter should admit this to be the cause of the disease) that it may deprive him of the hopes of a recovery."—William Osler, M.D. Lectures on Angina Pectoris and Allied States, 1897.
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IRVING S. WRIGHT

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