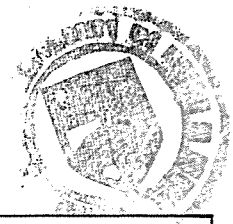
An Overview of Arteriosclerosis and Surgical Considerations of Coronary Arteriosclerosis.



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In the western civilisation arteriosclerosis has become the major cause of death, accounting for approximately 65% of all deaths. In recent years it has increased in almost epidemic proportions.

Studies have shown that the disease process is initiated early in teenage life and progresses insidiously thereafter to produce extensive morbidity and death in later life due to ischaemic damage to the myocardium, brain, lower limbs and viscera.

The aetiology is multifocal and has strong social implications. The financially richer a society becomes the greater is the development of arteriosclerosis.

I. Diet

There can be little doubt that the major cause is dietary due to increasing intake of animal lipoprotein products such as cheese, cream and butter. It is interesting that all these foods are judged to be highly nutritious and desirable by poorer societies and are avidly sought after when income increases.

The offending constituents are probably saturated fats (found in all these foods) and to be more specific perhaps the low density lipoprotein fraction.

Not surprisingly the incidence of arteriosclerosis is markedly reduced in societies eating *poorer* diets of high carbohydrate and low animal protein content, eg. Japanese fisherfolk and Central African tribes. In populations that have suddenly acquired wealth, eg. certain Arab populations, and who have within one generation changed their dietary intake from low to high animal lipoprotein intake the incidence of arteriosclerotic disease has exhibited a dramatic step-like increase.

It is interesting to note that the *normal* range of blood lipids in high animal protein intake populations, eg. British, is higher than the *normal* range in low animal protein intake populations, eg. Japanese fisherfolk.

In England the Milk Marketing Board has for many years extolled us to "drink a pint of milk a day" from childhood to adulthood. I know of no better way of priming a population for arteriosclerosis!

There are other important aetiological factors:-

II. Inadequate Physical Exercise

Lack of regular physical exercise results in lack of episodes of vasodilated high blood velocity circulation. It is perhaps not simplistic to view periods of rapid blood circulation as a flushing mechanism of the vascular tree, not unlike how episodes of fast river flow stop silting. It would appear that significant physical exertion needs to be taken at least three times per week to be of benefit, and the level of physical exercise must make one at least breathe heavily.

III. Smoking

There is no doubt that smoking is an aggravating factor. A person smoking twenty cigarettes per day is eight times more likely to suffer symptomatic arteriosclerotic disease. Nicotine increases vascular endothelial permeability and it can be surmised that this allows greater passage of saturated fats into the sub-endothelial area where it cannot be cleared quickly enough to stop deposition which is the beginning of arteriosclerosis.

IV. Certain Other Diseases

These increase propensity to arteriosclerosis. Among these are:

(a) Familial Hyperlipidaemias:-
Again it is easy to surmise that the high blood fat levels in these families increases the tendency to sub-endothelial deposition. Many of these families give a history of generations of early death in the fourth and fifth decades of life from arteriosclerosis.

(b) Diabetes Mellitus:-
Again associated with increased blood fat levels.

(c) Systemic Hypertension:-
If the hydrodynamic pressure on the arteries is abnormally raised the blood fats will be increasingly pushed into the arterial wall with the same result as in Familial Hyperlipidaemia and Diabetes Mellitus.

When considering the pathology of arteriosclerosis there is initially a deposition of saturated fats in the vascular sub-endothelium of the major arteries. This is often more marked in the posterior wall, perhaps because of the significant proportion of one's life spent in the horizontal position sleeping.

The lesions that cause reducing blood flow and subsequent ischaemic damage are however localised accentuation of the arteriosclerotic process. These stenoses characteristically occur at points of arterial branching or occasionally where arteries are relatively fixed and immobile e.g. Adductor opening in the lower thigh. Experimental studies in the Angiology Department of Guys Medical School and elsewhere have revealed that at these arterial anatomical points a minor reduction in arterial lumen by early fat deposition causes haemodynamic mismatching which produces a retrograde reflection pressure wave from each pulse. In turn this retrograde reflection pressure wave quickly meets the next pulse and opposing forces produce a stationary standing pressure wave. This standing pressure wave is an expression of kinetic energy (in contrast to the potential energy) and exerts a damaging effect on the local arterial wall which in turn locally accelerates the arteriosclerotic process to produce localised progressive stenosis, often with ulceration. Further progress in this vicious cycle ends by producing local total arterial occlusion.

Blood flow down an artery begins to reduce when its cross sectional area is diminished by 75% i.e. quite late in the disease process. It is only then that tissue hypoxia begins to develop, producing symptoms such as angina and claudication. When occlusion occurs, and if there are inadequate collateral vessels and flow stops, then tissue death occurs causing myocardial infarction, strokes or gangrene of lower extremities or bowel.

It is only because of this mechanism of localised accentuation of the disease that it is possible for operative vascular reconstruction to be successful in relieving symptoms and avoiding tissue death. This surgical principle applies to all arteriosclerotic disease in the body where the offending stenosis or occlusion can be technically bypassed or removed.

Considering Coronary Artery Disease.

Modern investigating equipment and medical expertise allows studies of the afflicted heart by such methods as Thallium scan, Gated red cell studies and cardiac catheterisation with coronary arteriography to accurately detail the extent of any myocardial damage, extent of myocardial hypoxia and exact localisation of coronary stenosis or occlusions.

From these studies it is possible to accurately assess the operative possibilities, risks and prognosis of bypassing the coronary lesions. When the risk is low and potential benefits high, operation is advised.

The operation involves cardiopulmonary bypass, high technical expertise and teamwork including high quality post-operative intensive care. The long saphenous vein is removed and appropriate lengths are used to bypass the coronary lesions by micro-vascular surgical techniques.

Today the operative risk is usually only 1-2% and the patient is usually fit to leave hopsital on the seventh post-operative day. The number of bypass grafts varies, as indicated, from one to seven with most patients having three or four.

The operation is advised if the patient is crippled with angina despite full medical therapy. There is an 87% success rate in relieving the angina. More importantly coronary artery grafting is indicated if there are certain specific patterns of coronary artery disease, to increase life expectancy:

(i) Left main stem stenosis
(ii) Three separate lesions affecting major coronary artery branches - known as *triple vessel disease*
(iii) Stenosis at beginning of left anterior descending artery - known as the *widow maker*

A prospective controlled trial, the *European Coronary Control Trial,* involving over 750 patients in which my unit at Guys participated, has established that operating on the above groups of patients not only usually relieves angina but also significantly prolongs life.

It is because of the good surgical results in these types of patients that the operation of coronary grafting has become a major industry in Western Civilisation. In the U.S.A. 350 - 400 people per million population are being grafted *each year.*

Other operations on hearts afflicted by myocardial infarction can be life saving e.g. excision of left ventricular aneurysm, closure of ruptured ventricular septum and replacement of severely regurgitant mitral valves because of papillary muscle infarction.

There can be no doubt that primary prevention of this disease by population education with appropriate dietary and social changes is all important. This is proving to be a difficult problem to overcome but perhaps the forecasted world wide shortage of food will automatically produce the desired effect!

Meanwhile as long as arteriosclerosis exists in such major proportion surgery plays a very important part in alleviating hypoxic problems and avoiding ischaemic damage in the heart. There are also, of course, vascular operations to relieve the same problems with regard of blood flow to the brain, viscera and lower limbs.