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EDITORIAL

CORONARY DISEASE

For long we have been accustomed to a steady decline in the general death rate and a steady increase in the mean duration of life, generally running parallel with the material welfare of the populations concerned. Good health appeared to go with high standards of living.

It has long been recognized, too, that from some diseases the mortality has decreased more than from others. Since the conquest of the formidable epidemic diseases it is from respiratory diseases, diarrhoeal diseases and tuberculosis that mortality has fallen to the greatest extent, while diseases of the heart and arteries, and cancer, remain as the greatest causes of death in the western world. An associated fact is that a greater reduction of mortality has taken place in infants and young children than in the middle-aged and elderly.

Circulatory diseases (and cancer) have moved in an opposite direction to the general reduction trend in mortality. In the last quarter of a century the increase in coronary heart disease has forced itself on the attention of the medical profession. In the United States the mortality from this condition in men aged 50-65 years has more than doubled in 25 years, and in other countries a mounting mortality from coronary disease is noted. In the Korean war over half the hearts of American soldiers killed in action showed developing atherosclerosis; their average age was 22. Braun [1] in his presidential address at a recent medical congress dealt with the increase of coronary disease in South Africa. From middle age onwards this upward trend threatens to neutralize the familiar downward trend in deaths from other causes.

With the growing recognition of these facts attention is to an increasing extent being focussed on coronary disease and its causation. A distinguished worker in this field, Professor Ancel Keys, of the University of Minnesota, who has pursued his researches in many countries and collaborated widely with other workers,

VAN DIE REDAKSIE

KRANSSLAGAARSIEKTE

Vir 'n geruime tyd al is ons gewoond aan 'n gestadige daling in die algemene sterftesyfer en 'n gestadige styging in die gemiddelde lewensduur wat in die reël ooreenstem met die materiële voorspoed van die betrokke volk. Gesondheid en 'n hoë lewenspeil het oënskynlik hand aan hand gegaan.

Dit is ook al lank bekend dat die sterftesyfer vir sekere siektes meer as dié vir andere gedaal het. Sedert die gedugte epidemiese siektes oormeester is, het die sterftesyfers vir asemhalingsiektes, diarree-siektes en tering die meeste gedaal, terwyl kanker en siektes van die hart en die slagare agterweë gebly het en vir die meeste sterfgevalle in die westerse lande verantwoordelik is. Dit moet ook in aanmerking geneem word dat die sterftesyfers vir babas en jong kindertjies 'n groter daling toon as dié vir middeljariges en bejaardes.

In teenstelling met die daling in die algemene sterftesyfer styg die sterftesyfer vir bloedsomloopsiektes en kanker. Die toename in kransslagaarsiekte gedurende die afgelope kwarteeu verg die aandag van die mediese beroep. In die Verenigde State het die sterftesyfer vir mans in die ouderdomsgroep 50—65 in die afgelope 25 jaar meer as verdubbel en ander lande teken 'n stygende sterftesyfer vir kransslagaarsiektes aan. Die harte van meer as helfte van die Amerikaanse soldate wat op die slagvelde van Korea gesneuwel het, het tekens van verharding en vervetting van die slagaar getoon; hul gemiddelde ouderdom was 22 jaar. Op 'n onlangse mediese kongres het Braun [1] in sy presidentsrede die toename in kransslagaarsiekte in Suid-Afrika behandel. Hierdie toename vanaf middeljarige leeftyd dreig om die daling in sterftesyfers vir ander siektes te neutraliseer.

Soos hierdie feite meer terdeë besef word, word die aandag steeds meer op kransslagaarsiekte en sy oorsaak gespits. Professor Ancel Keys van die Universiteit Minnesota, 'n beroemde navorser op hierdie gebied wat al in baie lande ondersoek ingestel het en met baie ander navorsers saamgewerk het, doen op die oomblik navorsing in Suid-Afrika. 'n Verslag van die lesing wat

is at present working in South Africa, and a lecture he recently delivered in Cape Town is reported in this issue of the *Journal* (page 332). He states that apart from syphilis and other preventible infections it is generally agreed that coronary disease 'seldom develops except on the basis of atherosclerosis'. This is associated with an invasion of the intima of the artery by deposits of cholesterol, which comes from the blood, and 'the tendency to form atheromata is related to the blood concentration of cholesterol and cholesterol-bearing lipoproteins'.

The amount of cholesterol in the blood is shown to be mainly determined by the diet. At first it was supposed that the cholesterol in the food was the determining factor; but this was disproved and it was found that the concentration of blood cholesterol depended mainly on the amount of fat (vegetable as well as animal) in the diet. The explanation of this lies in the fact that fat, which is insoluble in the watery media of the body, is transported in the blood in the form of water-soluble lipoproteins, of which cholesterol is an important constituent. Keys' researches have applied to populations rather than individuals, and on this scale he has investigated the correlation between fatty diet, blood-cholesterol, and coronary disease. The fat in the average diet (expressed as the percentage of total calories that are supplied by fat) varies from 8% in Japan and 15% in the South African Bantu to 41% in the United States. In all the countries investigated, including also England, Sweden, Italy, Spain and others, it was found that a close correlation existed between diet-fat percentages, blood-cholesterol concentration, and the prevalence of coronary disease.

In certain countries the same correlation was found when working-class groups with low diet-fat were compared with the well-to-do classes; and it was also found in countries where as the result of the last world-war the fat in the national diet was temporarily reduced that the coronary mortality fell to a corresponding extent and increased again when diet-fat returned to normal.

Keys states that when the calorie-percentage of fat in the diet 'is less than 20% atherosclerosis is slight and coronary disease is rare; at 30-35% . . . . coronary heart disease tends to become the first cause of death for all ages over 40; and at 40% or more, coronary heart disease tends to become a veritable plague'.

It appears that obesity *per se* is of only secondary importance in relation to atherosclerosis. In the Italian population, where dietary fat, blood cholesterol and coronary disease are all low as compared with American and English populations, the people are as fat as the Americans and fatter than the English.

We are accustomed to the conception of poor diet as a cause of disease; now we are faced with a widespread disease that is the result of richness of diet. Further research into the problem is needed; for instance, the disparity between the sexes in regard to coronary disease has still to be explained. But the striking results that research has already provided give good reason to hope that in dietary control methods may be found of reducing the serious menace of coronary disease.

1. Braun, L. I. (1953): S. Afr. Med. J., **27**, 477.

professor Keys onlangs in Kaapstad gegee het, verskyn in hierdie uitgawe van die Tydskrif (bladsy 332). Hy verklaar dat afgesien van sifilis en ander voorkombare infeksies, dit algemeen aanvaar word dat kransslagaarsiekte '*seldom develops except on the basis of atherosclerosis*'. Dit gaan gepaard met die skending van die slagaarbinnevlies deur 'n neerslag van cholesterol wat van die bloed afkomstig is, en '*the tendency to form atheromata is related to the blood concentration of cholesterol and cholesterol-bearing lipoproteins*'.

Dit is bewys dat die hoeveelheid cholesterol wat in die bloed aanwesig is hoofsaaklik deur die dieet bepaal word. Die mening was eers dat die cholesterol in voedsel die bepalende faktor was; hierdie opvatting is egter weerlê en die konsentrasie bloedcholesterol hang hoofsaaklik af van die hoeveelheid vet (plant- of dier-) in die dieet. Die verklaring hiervoor is dat vet, wat nie in die waterige media van die liggaam oplosbaar is nie deur die bloed vervoer word in die vorm van lipoproteïene (wat in water oplosbaar is) waarvan cholesterol 'n belangrike bestanddeel uitmaak. Keys se navorsing het volke eerder as individuele geraak, en op hierdie grondslag het hy die verband tussen 'n vetterige dieet, bloedcholesterol en kransslagaarsiekte bestudeer. Die vet in die gemiddelde dieet (uitgedruk as dié persentasie van die totale kalorieë wat deur die vet voorsien word) wissel van 8% in Japan en 15% vir die Bantoe in Suid-Afrika tot 41% in die Verenigde State. In al die lande waar ondersoek ingestel is, o.a. Engeland, Swede, Italië en Spanje, bestaan daar 'n noue verband tussen dieetvet-persentasies, bloedcholesterolkonsentrasies en die voorval van kransslagaarsiekte.

In sekere lande bestaan daar dieselfde verband tussen die arbeiderklas wie se dieet min vet bevat en die meer gegoede klasse; in lande waar die vet in die volksdieet—as gevolg van die tweede wêreldoorlog—tydelik verminder was, het kransslagaarsiekte tot dieselfde mate afgeneem, en toe die vet in die dieet herstel was, het kransslagaarsiekte weer toegeneem.

Keyes konstateer: '*when the calorie-percentage of fat in the diet is less than* 20%, *atherosclerosis is slight and coronary disease is rare; at* 30—35% . . . . *coronary heart diesease tends to become the first cause of death for all ages over* 40; *and at* 40% *or more, coronary heart disease tends to become a veritable plague*'.

Sover dit verharding en vervetting van die kransslagaar aangaan, blyk dit dat vetsug *per se* nie van primêre belang is nie. In Italië waar die vet in die dieet, die bloedcholesterol en kransslagaarsiekte laag is, in vergelyking met Amerika en Engeland, is die mense net so geset soos die Amerikaners en meer geset as die Engelse.

Ons is gewoond aan die stelling dat 'n swak dieet siekte veroorsaak; nou staar die feit ons in die oë dat ryk kos die oorsaak van 'n alombekende siekte is. Hierdie probleem vereis verdere navorsing; die verskil tussen die manlike en vroulike geslag wat hierdie siekte betref, wag nog op verduideliking. Die treffende feite wat navorsing alreeds aan die lig gebring het, gee ons goeie rede om te glo dat die ernstige bedreiging van kransslagaarsiekte deur geskikte dieetkontrole gestuit kan word.

1. Braun, L. I. (1953): S.-Afr. T. Geneesk., **27**, 477.

THE DURBAN OUTBREAK

It is a far cry from frigid Iceland to Addington Hospital on Durban's sunny South Beach. This association is evoked, however, by the outbreak among the nursing staff of the Addington Hospital of a disease which according to reports received apparently attacks the nervous system and bears a striking resemblance to a number of other outbreaks, the first of which occurred in Akureyri, in Iceland, in 1948.[1]

The Akureyri episode ('Icelandic disease' as[2]) was explosive as the Addington outbreak is reported to have been, and affected 465 persons or 6.7% of the total local population. Comparable outbreaks have been reported from Adelaide, South Australia,[3] from New York State[2], from the Whitley Hospital, Coventry [4] and from the Middlesex Hospital, London.[5] The Coventry outbreak and that at the Middlesex are of particular interest as they were also confined to members of the nursing staff. The Adelaide and Coventry outbreaks, like that in Durban, followed in the wakes of poliomyelitis epidemics.

In these outbreaks there is a variable prodromal stage with malaise, headache, sore throat or gastro-intestinal disorder and mild fever. After about a week the headache often increases and a stiff back and neck develop, without true rigidity. The patients complain of a heavy feeling in one or more muscle groups and often of difficulty in sitting up. Muscle pain is common and was a striking feature in the Middlesex outbreak. Paraesthesiae may occur in the affected limbs but, although muscle tenderness is marked, there are only slight changes in cutaneous sensibility. There is little more than mild paresis of the affected muscle groups, and tendon reflexes may or may not be diminished. Occasionally pyramidal-tract signs are seen. Cranial-nerve involvement is not marked; nystagmus, however, may occur and there was a high incidence of diplopia amongst the Middlesex nurses. Sphincteric disturbances are limited to difficulty in the initiation of micturition during the acute phase of the illness.

Laboratory investigations are not informative. No haematological abnormality has been found and, although a few cases from Akureyri and Adelaide did show mild pleocytosis or increased protein, most cases have normal cerebrospinal fluids.

The course of the disease is generally benign and no deaths have been reported. There is rapid initial improvement and most of the acute symptoms, including the paresis, subside within a few weeks. Further convalescence, however, is tardy; relapses may occur and lassitude, irritability and impaired concentration are persistent sequelae.

In each of the overseas outbreaks the early cases were thought to have 'atypical poliomyelitis'. Poliomyelitis or rather polio-encephalitis has been known to produce pyramidal-tract changes, cranial-nerve involvement and sensory or sphincteric disturbances.[6, 7, 8] A normal cerebrospinal fluid may also be found in the occasional case.[9] The very high attack-rate, however, and the very high incidence of these unusual features made this diagnosis most improbable. Careful investigation of the stools in the different outbreaks did not yield poliomyelitis virus nor was there any serological evidence of recent poliomyelitis infection.

Investigations for other known neurotropic viruses have been made but is has not been possible in these cases to demonstrate infection with lymphocytic-choriomeningitis virus nor with the viruses of St. Louis, Japanese B, Australian X, equine, lethargic or mumps encephalitis. Coxsackie virus produces meningitic rather than encephalomyelitic manifestations [10] but was suspected because of the striking muscle tenderness. Its presence, however, could not be demonstrated by the Icelandic, New York or Middlesex Hospital workers.

The cause of these outbreaks, therefore, remains obscure. The remote observer, however, may care to indulge in a speculative exercise and a number of theories are offered for consideration:

The character of the outbreaks makes a toxic cause most unlikely and some sort of infection must be sought. The clinical picture suggests a virus infection and it is probable that such an aetiological agent will be found here. Two of the overseas epidemics and the Durban epidemic have been closely associated with poliomyelitis outbreaks, and a causal connection is feasible. Is a mutational variant of the poliomyelitis virus responsible for the atypical syndrome? Or is the clinical picture modified by partial immunity in the host? May the disease be not directly due to poliomyelitis infection, but represent a nervous-tissue hypersensitivity to poliomyelitis antigen? Or is this indeed a new disease due to an as yet unidentified organism? The medical profession in this country and elsewhere will be most interested to hear the report of their colleagues, who have observed the Durban outbreak, which is published in this issue (p. 344). It is a summary of the clinical findings and does not include the detailed investigations that are in progress. In due course a detailed report will be published.

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