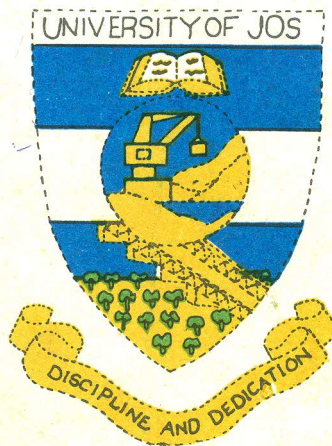


UNIVERSITY OF JOS

**THE END OF A MYTH:
THE EVOLUTION OF
CARDIOLOGY IN AFRICA**



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INAUGURAL LECTURE

by

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An Inaugural Address delivered at the
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INTRODUCTION

MR. VICE-CHANCELLOR, eminent guests, Deans and Colleagues in the various Faculties of this august University and from other Universities, distinguished ladies and gentlemen; the saying is attributed to Aristotle that: "The Philosopher must begin with Medicine and the Physician must end with Philosophy." An inaugural lecture presents the new incumbent of a Chair in the academic community the opportunity to examine in public those aspects of his beliefs which have guided his academic career thus far; and since it is an inauguration, prepares him for continuing academic activity in the exalted position which he would now hold among his academic colleagues. In a personal sense this would not be an appropriate description of an inaugural address; however, the Faculty of Medical Sciences of this University today, at the end of its sixth year, celebrates its coming of age and the gradation of its first set of students. It is in this sense that an inaugural address from the Chair of Medicine in this University is opportune at this time and I consider it a unique honour to have had the opportunity to present this year's address.

The major difficulty of an address of this sort to a mixed audience, is to achieve the correct blend of erudition and lucidity. If during this address therefore one becomes obscure, it will be in the hope that the major theme would not be lost on my audience. On the other hand, the field of Medicine is not one in which it is easy to achieve widespread understanding without becoming anecdotal and if this happens today, it would be because the topic is such as cannot be presented in the time available without some over simplification.

THE PHYSICIAN MUST END WITH PHILOSOPHY

It is accurate that the physician must end with philosophy. Philosophy in this sense represents the guiding belief, the motive principle in one's continuing search for knowledge; a syn-

thesis of all previous working experience into principles and ideas for continuing study. This state of affairs is not achieved like St. Paul in a blinding flash except for some fortunate few; more usually it is arrived at after years of painstaking activity, some of them leading to blind alleys, but always in general returning one to the main theme of activity and the gradual evolution of a working philosophy.

For this speaker, a philosophical position has been reached which recognises that the study of cardiovascular disease in our environment must emerge from the study of the manifestations of disease in the individual to their manifestations in whole populations. There are several methods of achieving understanding of the nature of disease. In this century, the oldest method, that of the study of its manifestation in the individual, has become so technical and costly for most developing countries, that it is often not possible to undertake more than superficial study of even the most common disorders in this way. Study of disease manifestations in populations on the other hand often yields considerable insight into the nature of disease and even in developed countries has provided valuable information relating to problems of disease management. Today we shall discuss some insights into the problem of cardiovascular disease in tropical Africa which have arisen in part from our studies and also provided further instigation for such studies.

The study of diseases of the heart and the circulation is as old as medicine itself. About 400 BC, Hippocrates, the Father of Medicine in his Aphorisms made clear allusions to disease of the heart and the circulation. In the fourth century B.C. Aristotle described the anatomy of the heart, although he believed then that the human heart had three chambers. In the second century A.D. Galen described the value of the study of the pulse at the wrist in the clinical diagnosis of cardiovascular disease and thereby established a practice that is still with

us today, nearly two thousand years later. But modern cardiology cannot be said to have started until the publication in 1847 by Harvey of his anatomical exercises on the motion of the heart and blood in animals. I will quote you part of his dedication of this work to Charles I of the United Kingdom: "The heart of animals is the foundation of their life, the sovereign of everything within them, the sun of their microcosm, that upon which all growth depends, from which all power proceeds. The King in like manner is the foundation of his Kingdom." The task of the scientific publisher was not simple in those days; but in likening the importance of the study of the heart and the circulation to the role of the monarch, I believe Harvey spoke of a truism for all time. The contribution of Harvey to modern medicine and to the English tradition in Physic is today recognised in the annual oration in his honour given to the Royal College of Physicians of London.

Modern medicine was introduced into Africa by men trained in this tradition; men trained in the study and understanding of disease as manifested in the individual and among whom therefore techniques of the study of disease behaviour in populations were either unknown or ill understood. Although at the turn of the century some understanding existed of the ecology of transmissible disorders, this was not often widely known or believed; epidemiology as a medical discipline involving also as it does now, the study of non-transmissible disorders is a relatively recent science. Early medical practitioners in Africa therefore found themselves requiring to make medical generalisations relating to the incidence of disease; and to making inter-country or inter-ethnic comparisons, all of which were the province of epidemiology, without having had the training to make these judgements and without having had the training to make these judgements and without understanding the principles involved in making such judgements. Many of the earliest assertions about cardiovascular disease in tropical Africa

arose from observations and judgements made by these early practitioners.

I was trained in this tradition and as a young medical officer lived in the milieu in which these assumptions were made and held. One such major assumption was that cardiovascular disease was rare in the African and text-books of Medicine as well as medical journals carried this information. I remember that similar assumptions were made of other disorders such as poliomyelitis, pulmonary embolism, appendicitis, etc. As one grew older, one's experience began to challenge these assumptions and this address relates to the contribution of the study of cardiology in Africa to destroying this myth as it relates to cardiovascular disease.

The problem that any cardiologist interested in the epidemiology of cardiovascular disease in Africa has to resolve, and one about which this address attempts to offer an opinion, is whether in fact beliefs about the rarity of cardiovascular disease in Africa were true at the time they were held.

The question often arises as to whether cardiovascular disease is being recognised more frequently now as medical services improve or whether in fact the incidence of cardiovascular disease is increasing in our countries so that our current experience is not simply the result of improved diagnostic facilities. This is not merely an academic exercise as its clarification would resolve the important issue of the time trends of cardiovascular disease prevalence in Africa; and this has a bearing both on predictions as to future disease behaviour in our populations and also on our understanding of their aetiology.

In 1965⁴² I analysed data on cardiovascular disease as seen in the cardiac clinic at the UCH Ibadan and from autopsy material over a period of three years. Both these sets of data indicated that hypertension and hypertensive

heart disease, the cardiomyopathies and rheumatic heart disease, accounted for more than 75% of all cardiovascular disease seen at this centre. By this time of course it was clear that many cardiovascular disorders which had been thought to be non-existent or extremely rare in the African did in fact exist. To examine in this address the historical evolution of the African experience of cardiovascular disease, it would be best to deal with individual disorders and to examine not the entire problem, but the few conditions which constitute the bulk of the cardiovascular disease burden among our populations.

HYPERTENSION

In 1929, Donnison²⁷ in Kenya claimed that in a two year period and after 1800 African patients he had not seen a single individual with hypertension. For many years these views provided the definitive opinion on the importance of hypertension in Africa. But the picture was not simple; for although a spate of publications emerged from East Africa concerning tribal groups among whom the blood pressure did not rise with age and hypertension did not occur,^{62, 92, 99, 101} the occasional publication^{12, 74, 22} claimed that hypertension did occur in other population groups in Africa. The belief however persisted that in general the blood pressure was lower in the African than in the Caucasian and that hypertension was uncommon; this, despite extensive data from the United States where scientists seemingly obsessed with possible genetic differences between the negro and white populations of America, were amassing data which showed that the U.S. negro had higher average blood pressure than whites and a greater prevalence of, and morbidity from hypertensive disease^{3, 23}.

The rationale for this apparent disregard of slowly accumulating evidence would seem to derive from the common concept at the time, of the genesis of high blood pressure, which regarded psychological variables as being para-

mount in the development of the disorder. This concept has been put crudely in the form of an analogy to a closed container being heated. "If steam (hostility) is not allowed to escape (expressed) the pressure (blood pressure) inside the container (person) may rise excessively (become high blood pressure) and produce an explosion (apoplexy)!"⁷⁵ The implication of this analogy was that suppressed aggression arising from psychological and emotional stress contributed to the development and establishment of hypertension. Since these observations on the possible relation of psychological factors to the development of hypertension were made among urban U.S. communities where individuals readily admitted to stress, it was not too difficult to believe, given this hypothesis, that the apparently slow life of primitive communities did not produce stress and therefore hypertension; nor was it too difficult to extrapolate this belief to the entire African continent.

X Emotion does in fact influence the blood pressure. The classical observations on the effects of routine daily activity and emotion on the blood pressure were made by Pickering and co-workers in 1964⁸¹ through continuous recording of the blood pressure. They showed that the blood pressure was constantly changing and that there was no single or fixed level of blood pressure for the individual. They observed also, that the blood pressure was low during sleep, the lowest figure being recorded at about 2.00 a.m. in normal sleep. The blood pressure recorded during the day was variable; high pressures being recorded in circumstances which might be regarded as stressful: while conducting a vehicle through traffic, or when speaking at an important meeting.

Last year during an inaugural lecture³¹, examples were given of stress in fish. All mammals respond to perturbations of the environment with what has been termed the alarm (defence) reaction. This response originates in the

cerebral cortex and is mediated by the hypothalamus; and provides the link between our consciousness of our surroundings and the cardiovascular system. The reaction is not only displayed in times of manifest threat, but is involved in small adjustments of the individual to all biological challenges, which have been loosely termed as stress – be they amusing or life threatening. There are neural and hormonal components to the defence reaction; in the short run, neural mechanisms lead to an increase in adrenaline secretion; this increases the heart rate and so the cardiac output; alters the calibre of resistance blood vessels and as a consequence, the blood pressure rises. Changes in adrenal cortical hormone output in the long run, produce more complex responses particularly relating to sodium balance which it is thought lead to a sustained elevation of the blood pressure or at least prevent a full return of the blood pressure to normal, if the stressful situation persists. It is inconceivable that the African does not have stress or that he responds to stress differently from other mammals.

The earliest population blood pressure studies in Nigeria were by Abrahams and co-workers¹, who showed in 1960 that the blood pressure levels of the Yoruba of Western Nigeria were similar to those found in Caucasians and that hypertension was common. These observations were supported by later studies by Akinkugbe^{5,6}. By this time, of course, work in East Africa apart from confirming that there were populations there among whom mean blood pressure did not rise with age and who exhibited little or no hypertension^{88,89}, also showed that there were those, as was the case in European and American communities, who had an augmentation of the blood pressure with age and manifest hypertension^{87,73}.

Personal studies among sub-urban populations have indicated a prevalence of hypertension in an East African community 45 years and over of 34%⁴⁶ and in a similar sub-urban

group in Ghana aged 15-64 years of 11%; rising from 6.3% in the age group 15 to 44 years to 33% (or one person in three) in the age group 45 to 64 years⁵⁷. Widespread clinical experience now places hypertensive disease as one of the commonest, if not the most common cardiovascular disease in the African⁷⁸. Epstein and Eckoff³² have defined several blood pressure age trends and showed that the steeper the rise of the mean population blood pressure with age, the greater the prevalence of hypertension in that community. Most populations studied in East and West Africa belong along with American Negroes to the group with the steepest age trend⁶¹.

There can be no certainty as to the historical progression of hypertension as a population disease in Africa. Two possibilities which are not mutually exclusive are likely. The first is that the prevalence of hypertension has been changing as African populations have changed. The second is that inaccurate conclusions were drawn based on studies of unrepresentative populations. With respect to this second possibility it has just been stated that there are several diminishing populations both in Africa and elsewhere in the world, among whom blood pressure does not rise with age and the prevalence of hypertension is low. Such populations usually consist of small primitive groups; and the earliest studies about blood pressure levels and hypertension from East Africa, appear to have been from among such groups. Several studies have shown that when such groups become sedentary the mean population blood pressure tends to rise and the prevalence of hypertension increases^{89,8}. With these observations in mind it is probable that mean blood pressure among African populations has been rising in the last six or seven decades and that the prevalence of hypertension has also been increasing. However, Africa has had sedentary populations for many centuries; but it is possible that there have been changes in population habits in these sedentary groups which might have contributed to an increase in the

prevalence of hypertension in the last century or so.

One important difference between populations that exhibit hypertension and those that do not, relates to the intake of dietary salt. Shaper in East Africa⁸⁹ was able to demonstrate an increase in mean blood pressure associated with a five-fold increase in salt intake among nomads who have changed to a sedentary life. There is probably a relationship between the amount of salt consumed in the diet and the ability to recognise a salty taste, the less the ability the more salt is consumed. We have shown that African hypertensives have a lower salt taste threshold than normal individuals and when we compared out salt taste thresholds with those obtained from Caucasians; normal Africans appeared to have a higher salt taste threshold than Caucasians⁵⁵. Without going into current debates on the relationships of sodium to the occurrence of hypertension, there is ample evidence that mean population blood pressure levels is constantly and positively related to dietary sodium intake among such populations⁶⁶. It is probable that an increase in the prevalence of hypertension of African communities may be found to have paralleled an increase in the general availability of dietary sodium on the continent.

Osuntokun⁷⁶ has shown that cerebrovascular accidents in the Nigerian most commonly occur in association with elevation of the blood pressure; while Mordi and Okuwobi⁶⁹ have shown that sudden deaths in Nigerians are most commonly due to cerebrovascular accidents. Ikeme et al. studied the blood pressures of children⁵⁴ and compared some characteristics of the parents and grand-parents of the 10% of children with the lowest blood pressures with those of the 10% of children with the highest blood pressures in our study. Apart from the finding that the parents of children with the highest blood pressures tended to have higher pressures than those of children with the lowest blood pressures; we found that

sudden deaths which we believed to be due to cerebro-vascular accidents occurred significantly more frequently in the grand-parents of children with the highest blood pressures. Although this study is of minor historical importance and was designed to examine the predictive value of childhood blood pressure and not the problem of the historical progression of hypertension in Africa, it is of interest that at least two generations ago at a time many African populations were thought not to have hypertension, sub-groups of such populations existed among whom deaths from strokes appeared to be occurring.

There seems no doubt that hypertension existed in Africa at the turn of the century and that its prevalence may be increasing, but whether this is due to changes in population habits or to increasing recognition of the disease will remain a matter for conjecture. The facts now are that hypertension is widely prevalent in Africa, that in the United States in general the prevalence of hypertension among the Negro population who are persons of West african descent is higher than among those of Caucasian descent, that hospital observations place hypertension as the commonest cardiovascular disorder and that this pre-eminence of hypertension among the cardiovascular disorders, is confirmed by studies among populations.

RHEUMATIC HEART DISEASE AND ENDOMYOCARDIAL FIBROSIS

We now turn our attention to rheumatic heart disease. Some twenty years ago, Abrahams and Brigden wrote: "rheumatic mitral valve disease has been studied intensively during the past 25 years. Mitral Stenosis is rare in Nigerians..."² Earlier reports tended to support this picture of the rarity of rheumatic heart disease in Africans; examples were reports by Davies²⁶ from East Africa in 1948 and by Edington²⁹ from West Africa in 1954; both reports were based on pathological stu-

dies. However, during this period, as in the early days of reports on hypertension, there were other publications of contrary findings on the importance of rheumatic heart disease in Africans^{11, 36}.

Rheumatic heart disease is a disease of poverty; streptococcal infection of the throat being common in conditions of poverty and overcrowding; often reaching epidemic proportions under these conditions. However, only a small fraction (0.5% to 2%) of individuals who have streptococcal sore throat develop rheumatic fever; of these a variable proportion will develop chronic rheumatic heart disease – resulting from damage to the heart valves³⁵.

In the United Kingdom, deaths from valvular disease of the heart – not entirely chronic rheumatic heart disease, but made up largely of this condition and therefore providing a reasonable estimate of the prevalence of this disease; rose from an average of 388 per million living persons in the years 1891 to 1910 to a peak of 658 million during the years 1921 to 1931 and then fell; so that by the years 1949-1959 it was 200 per million¹⁹. These changes were not synchronous with but followed by some decades the period of great urbanisation in Britain resulting from the industrial revolution and subsequently followed improvements in living standards. The experience in developed countries is that of a gradual reduction in the prevalence of rheumatic heart disease over the last five or more decades; so that in many such countries it is no longer regarded as a public health problem.

In Africa the documentation of the historical trends of rheumatic heart disease has not been as extensive as has been the case with hypertensive disease. This is probably due to the difficulty of making assumptions particularly from hospital material, about a disease with a relatively low population prevalence. Halim and Jaques⁴⁰ reviewing the problem in the Sudan in 1961 found rheumatic heart disease to consti-

tute 25% of all cardiac disease in their clinic. They proceeded to compare their series of cases with earlier studies in the Sudan which showed an incidence of 14% in 1945 and an even smaller fraction in 1937. In East Africa, D'Arbela and co-workers²⁴ reported an incidence of 26% in 1962/63 from the cardiac clinic at Mulago Hospital, Kampala. From the same hospital in 1960, Sharper and Williams⁸⁴ found rheumatic heart disease to be 13% of all cardiac admissions. In 1954, Williams et al.¹⁰² found rheumatic heart disease in 8.7% of an autopsy series of 231 patients dying from heart disease; while Davies²⁶ a few years earlier, had reportedly found no rheumatic heart disease. It is tempting to suggest that these figures represent an increase in the incidence of rheumatic heart disease in African populations over the last three or four decades. However, a major pitfall in making such assumptions is to ignore the lack of comparability of the data base and of the techniques of assessment. Clearly, the conditions exist in Africa for an increase in the prevalence of rheumatic heart disease: rapid urbanisation with its attendant increase in urban squalor. In many other developing countries, presumptive epidemiological evidence exists that the incidence of rheumatic heart disease is increasing.

Whatever may have been the case in the past, there is no doubt today of the importance of rheumatic heart disease in African populations. Ikeme et al. found the prevalence of chronic rheumatic heart disease in a Ghanaian population aged 15-64 years to be 3.5 per thousand⁵⁷. Ogunbi⁷¹ found a carrier rate for the streptococci of 10% among school children in Lagos, and elevated anti-streptolysin titres in 14% of children under 5 years of age. In Kampala⁹³, significant anti-streptolysin titres have been found as early as the first six months of life. The bacteriological and immunological evidence just cited suggest that rheumatic heart disease in tropical Africa can no longer be regarded as a rare disorder; but that

the basis for this disease exists; and is widespread as early as the first year of life. It is now generally recognised from several clinical studies that rheumatic heart disease in tropical Africa "is a rapidly destructive disease, producing severe disability at a young age..."⁹⁰; that valvular destruction appears to be more extensive than in Europe; that valvular damage is found in an age group much younger than in Europe and that the incidence of the disease is probably rising in most parts of tropical Africa.

Cardiologists have probably been slow in reaching this state of knowledge because rheumatic heart disease in Africa unlike in Europe, commonly produces a mitral valve regurgitation. In fact Abrahams and Brigden in 1961¹¹, described mitral valve incompetence with myocarditis and pulmonary hypertension in the belief that they were describing a new cardiovascular syndrome. With hind-sight we can now recognise that these authors described two common conditions which both produce mitral regurgitation: rheumatic heart disease and endomyocardial fibrosis. The first description of this latter condition is attributed to Bedford and Konstam in 1946¹³, who presented to the British Cardiac Society observations on West African troops serving in India and Burma during the Second World War. Again it is certain now that this description included more than one disease entity. It was not until the definitive descriptions of the pathology of this condition by Davies²⁵ that the syndrome of endomyocardial fibrosis was clearly delineated. These two conditions, endomyocardial fibrosis and rheumatic heart disease, present diagnostic difficulties since they both give rise to mitral regurgitation although I have attempted in a number of publications to show that clinical distinction between the two was possible in most cases¹⁸, 43, 44.

More intriguing is the possible relationship of this condition to rheumatic heart disease. Firstly the age incidence of rheumatic heart di-

sease and endomyocardial fibrosis is almost identical⁴⁵. Secondly while rheumatic heart disease is a world-wide problem, endomyocardial fibrosis remains a disease of hot, wet tropics. Lastly, a similar reciprocal difference in regional or ethnic incidence between endomyocardial fibrosis and rheumatic heart disease has been observed among the Baganda and Rwandans of East Africa⁸⁶; as in Ibadan, between the population around Ibadan among whom endomyocardial fibrosis is comparatively uncommon and rheumatic heart disease common; and the population of the Ijebu province among whom the reverse is true⁶¹. Shaper⁸⁵ on the basis of his observations in this respect proposed that endomyocardial fibrosis was a form of rheumatic heart disease modified by as yet poorly defined immunological mechanisms set off by malaria infection. So far, this has remained speculative and the problems associated with the diagnosis and aetiology of endomyocardial fibrosis have been presented in this address only in order to emphasise the extent of the problem of understanding the historical trend of rheumatic heart disease in tropical Africa because of the confusion produced by the presence of a closely related disorder, and to draw attention to the emergence of this cardiovascular disease syndrome at about the time rheumatic heart disease became recognised as an important problem in the African.

HEART DISEASE OF UNKNOWN AETIOLOGY

The evolution of cardiology in Africa has not consisted solely in discovering that disorders prevalent in Europe and thought to be absent in Africa did in fact occur in Africa. Perhaps the most important aspect of our understanding of the nature of cardiac disease and of the evolution of knowledge about the importance of cardiovascular disease in Africa, has been in the recognition of the cardiomyopathies; and in modest but systematic efforts to determine if not their aetiologies, at least their pathogeneses. The term 'cardiomyopathy' has been de-

efined by Goodwin³⁹ as applying to any "acute, subacute or chronic disorder of the heart muscle of unknown or obscure aetiology, often with associated endocardial or sometimes pericardial involvement but not arteriosclerotic in origin." In many ways the cardiomyopathies are difficult to understand. For the cautious clinician they often represent diagnostic traps; and for the negligent a diagnostic waste paper basket. They include endomyocardial fibrosis which has just been described, peripartal cardiomyopathy and idiopathic cardiomegaly. It is this last condition with which this section of this address will be concerned.

Early descriptions of the cardiomyopathies include those of Bedford and Konstam in 1946¹³; Davies in 1948²⁵; Gillanders from South Africa in 1951³⁸ and Becker and co-workers also from South Africa, in 1953¹⁰. Initially, all these authors believed they were describing one and the same disease entity. It was not until a seminar in South Africa in 1957⁹⁵, that the two conditions: endomyocardial fibrosis and idiopathic cardiomegaly were recognised as separate disease entities. It was then acknowledged that the East Africans had been describing the former and the South Africans the latter conditions. The term idiopathic cardiomegaly embraces all "those subacute or chronic disorders of the myocardium associated with cardiomegaly and congestive cardiac failure occurring in the absence of valvular disease, hypertension coronary disease or other observable cause and presenting at autopsy with variable cardiac dilation and hypertrophy and with inconstant endocardial lesions"⁵³. Simply this means disease of heart muscle with heart failure, of unknown cause – but this particular definition excludes endomyocardial fibrosis.

My first contact with international cardiology was at a World Health Organisation seminar in 1965 which defined the clinical features of some cardiomyopathies and identified the various clinical reports which the panel believed

represented isolated descriptions of idiopathic cardiomegaly⁴¹. Despite the fact that this WHO study group indicated some areas for research in the cardiomyopathies, interest in idiopathic cardiomegaly remained sporadic. The most sustained activity in this regard in the 1960's was from the West Indies. This group followed their initial reports⁹⁶ in 1963, with longitudinal studies of the Lawrence Tavern population (a hilly village near Kingston) and were able to show that enlargement of the heart in the absence of any apparent abnormality occurred in this population and that subsequently, myocardial failure occurred in these subjects and that this was associated with a high mortality⁹⁷. Ikeme and various associates have been able to show that such enlargement of the heart without any obvious cause occurs both in East and West Africa^{47, 48, 52}.

The arguments as to the pathogenesis of idiopathic cardiomegaly are too complex to be related here and I will concern myself with those aspects of the understanding of the problem of its pathogenesis and of its importance in our populations to which our own studies have contributed. With respect to its pathogenesis, I⁶⁰ have analysed some characteristics of these individuals with cardiomegaly observed at field studies and have shown: firstly that the aorta on chest radiographs in these persons tend to be as wide as those of hypertensive individuals suggesting that hypertension may have contributed to cardiac enlargement; secondly that women with cardiomegaly have a higher mean number of children at each age than those without cardiomegaly suggesting that pregnancy and parturition contribute to cardiac enlargement; thirdly that when the prevalence of cardiac enlargement is examined by social class, a bi-modal curve is obtained with the higher peak in the low socio-economic group, indicating a possible multiple aetiology and the likelihood that the associations of poverty contribute to the pathogenesis of the disease in the poorest section of the community;

and finally that persons with cardiomegaly have a significantly higher body size than those without cardiomegaly. I have suggested that this relation may be causal and described the mechanism through which obesity might be responsible for producing cardiomegaly in otherwise normal persons⁶⁰

Because of the belief of some authors that idiopathic cardiomegaly was simply hypertensive cardiac failure^{17, 70}, Ikeme et al. undertook clinical studies of this condition and observed that of 25 patients with idiopathic cardiomegaly who presented in heart failure with a normal blood pressure and were followed for from three months to two years, hypertension infact became evident in seven of these (28%) after heart failure had regressed^{49, 58}. On the other hand, 90% of those whose blood pressure remained normal, also remained in heart failure or had an enlarged heart during the follow-up period. These observations have now been supported by others³⁴. We concluded in the patients we studied that hypertension was contributory to heart failure; but since heart failure due to high blood pressure could not be expected to occur with a very elevated blood pressure in one individual and in another with low or normal blood pressure; the latter group of patients were clearly persons in heart failure with hypertension in association with some other cause of heart failure. Since blood pressure is a function of the cardiac output and the peripheral resistance, it is easy to see why the blood pressure might be normal in severe myocardial failure despite the very raised peripheral resistance associated with this condition. We went on to propose a hypothesis of the pathogenesis of this condition⁵¹ and later expanded it to describe a working concept of idiopathic cardiomegaly in Africa⁵³. Briefly, idiopathic cardiomegaly is a disorder with multiple pathogenetic factors. The factors leading to heart failure might include hypertension, pregnancy and parturition, chronic malnutrition, the late effects of viral infection and possibly the con-

sequences of obesity. The combination of factors causing heart failure in any person would be unique for that individual. One may add that evidence for each of these factors operating in idiopathic cardiomegaly has been described severally; in each case sufficient support exists for the view that the factor could not be the sole cause of heart failure. I believe also that a similar clinical condition seen in infants and children probably has more immediate causes and should be excluded from the designation idiopathic cardiomegaly.

In clinical practice in Nigeria, idiopathic cardiomegaly varies from 15% to 44% of all cardiac admissions^{20, 72, 77}. Parry and Gordon⁷⁵ reviewed in 1968, published data on hospital admissions for various cardiovascular disorders; admissions for idiopathic cardiomegaly varied from 4% in Mombassa, Kenya to 34% among the Bantu in Durban and from Bathurst in Gambia. This wide variation reflects the difficulties of clinical diagnosis and nomenclature, rather than a true inter-country variation in incidence. In population studies, the prevalence of enlarged hearts without any obvious cause varies in various published reports depending on the criteria used in selecting such cases. Geser and Thorup³⁷ using a cardiothoracic ratio greater than 62% found a prevalence of obscure cardiomegaly of 9 per 1000 from over 16000 chest radiographs taken in 8 countries south of the Sahara. This certainly represents a serious underestimation of the problem. In two major population groups we have studied; using a cardiothoracic ratio greater than 55% in the absence of hypertension, murmurs or anaemia, the prevalence of cardiomegaly was 7% in the Ugandan study in individuals aged over 45 years; and 6.9% in the Ghanaian community aged 15 to 64 years. In this latter population, the prevalence of cardiomegaly in the age group 45 to 64 years was 15% or twice as much as in Uganda. Even when the lowest prevalence figures are used, this still represents a high abnormality rate. Doubts

still exist as to whether these rates might not represent an overestimation of the problem. If only a small fraction of these enlarged hearts follow the Jamaican pattern, and progress into heart failure, it would still represent a huge burden of obscure heart failure.

There is no doubt that idiopathic cardiomegaly constitutes an important problem in tropical Africa. It is a disorder that affects the rich and the poor, although one's impression is that this may represent different aetiological factors. The disorder is world-wide but the highest prevalences have been reported from tropical developing countries. There is no indication that the prevalence of this disorder is increasing in Africa; but idiopathic cardiomegaly is a major cause of morbidity and death which up till now is still ill-understood and a few decades ago was not even documented.

CORONARY HEART DISEASE

Coronary heart disease is one more condition which we should examine. In the last 50 years or so a modern epidemic has emerged and ravaged developed countries; coronary heart disease has emerged from an insignificant problem in the Western world to become the commonest cause of death among men in Europe and America⁷⁹. Despite this pre-eminence of coronary heart disease in the Western world, the incidence of the disease is still rising in many countries; while in a few such as the United States of America, the incidence is now beginning to fall.

Coronary heart disease results from intimal damage (atherosclerosis) leading ultimately to occlusion of the coronary arteries of the heart. The immediate mechanism of this damage is ill-understood, but the major contributory factors have been recognised as high blood levels of low density lipo-protein (cholesterol), the smoking habit and elevation of the blood pressure. Other contributory risk factors are physical inactivity, high body weight and dia-

betes. It would seem that mean levels of serum cholesterol in a population is a measure of the extent of atherosclerosis in that population and the amount of coronary atherosclerosis is an index of the community susceptibility to coronary disease^{79, 91}.

All reports from tropical Africa emphasize the rarity of coronary heart disease in the African. The earliest comment on the rarity of coronary artery disease was by Becker⁹ on the South African Bantu. Schrire⁸² reviewed 4167 electrocardiograms of patients attending Groote Schuur Hospital in Capetown during 1956. Of 336 electrocardiograms accepted as indicative of myocardial infarction, 250 (74%) were Caucasians and 1 (0.3%) was in a Bantu patient. This rarity of coronary heart disease has been confirmed from autopsy studies by Davies in Uganda²⁶ and by Edington in Ghana²⁹. In a personal series of 465 cases registered in the cardiac clinic at UCH Ibadan over a 15 month period in 1964/65, 3 (0.6%) were cases of myocardial infarction; while from an autopsy series, myocardial infarction constituted 1.5% of deaths from cardiovascular disease⁴². In more recent years, reports from Nigeria^{3, 33, 100} and other parts of West Africa^{14, 63} have continued to confirm the rarity of coronary heart disease in the African in comparison with Caucasians. Pathological studies have also shown that in comparison to Caucasian populations atherosclerotic patches are as much as ten times less frequent in the blood vessels of Nigerians than in Caucasians^{80, 94}.

This low incidence of both atherosclerosis and coronary heart disease despite the high prevalence of hypertension in many African populations, is now believed to be due to the observed lower mean age adjusted serum cholesterol levels in African populations^{8, 16, 30}, and a lower prevalence of the smoking habit⁵⁷. There is evidence in the African of increased fibrinolytic activity⁶⁵ and a reduced platelet stickiness⁹¹ – both of which might result from

the lower serum lipid levels observed in Africans and which reduce the tendency to thrombus formation in blood vessels. Thus low cholesterol and smoking levels probably ensure the absence of significant damage of the intima of blood vessels in Africans and the reduced platelet stickiness and increased fibrinolytic activity reduce the chances of intravascular obstructive clots forming.

Despite this massive accumulation of data on the low incidence of coronary heart disease in tropical Africa, there is increasing evidence that the incidence of coronary heart disease is in fact rising in Africa as well as in many other developing countries. Chia²¹ in a keynote address at the 1980 ASEAN Congress of Cardiology points out that while there has been a fall in death rates for hypertensive heart disease and rheumatic heart disease in Singapore, in the last 30 years the death rate for coronary heart disease has risen alarmingly.

In a recent document I reviewed the problem as seen in Africa⁵⁸ and drew attention to the documented experience in Abidjan¹⁵ and Dakar¹⁰³. In Dakar there seems to be an exponential growth of the number of cases seen at the Centre Hospitalier de Dakar over a 15 year period, with a doubling time of probably less than 10 years. In the Mamprobi Survey, the only epidemiological study of the three major risk factors for coronary heart disease in tropical Africa. In this Ghanaian urban population, apart from hypertension, the levels of other major risk factors: hypercholesterolaemia and the smoking habit, were considerably lower than in Caucasian populations^{57, 59}. However, when the prevalence of each major risk factor is examined in relation to socio-economic status there was either a strong linear correlation of increased risk with socio-economic status, or a statistically significant higher level of risk in the higher than in the lower socio-economic stratum⁵⁹. This was true both of individual risk factors and of combinations of risk factors. It

was concluded then that: "The high socio-economic group in Africa has a higher risk for coronary heart disease than the low having as it does values for risk approximating those in high incidence countries... It is to be expected that economic development would in the future, increase the wealth of a great proportion of the population. Experience in developed countries would suggest that in one or two decades, affluence with consequent changes in life style particularly with regards to saturated fat intake and smoking would radically increase the prevalence of coronary heart disease in many presently low incidence populations"⁵⁹. Thus far, the story of coronary heart disease.

EX AFRICA SEMPER ALIQUID NOVI

In the past five decades, many population studies of cardiovascular disease have been conducted in Africa. Unfortunately most of these studies have been of discrete disorders and often in unrepresentative populations. While providing time specific information about these individual disorders, these studies have had two major inadequacies: firstly; although cross-sectional studies provide information about disease prevalence, i.e. the amount of disease present at the particular time; they do not provide information about the rate of occurrence of new disease – its incidence. What is more, cross-sectional surveys have the additional disadvantage that the amount of some disorders will be underestimated. For example, diseases that are rapidly fatal would be underestimated; so would those that are associated with disability, if for example subjects have to report to a central study area. Secondly, in order to meet the technical requirements for making valid conclusions, the study population has to be accurately circumscribed. When this is done, except for a few well planned studies in developed countries, the extrapolation of the data obtained from such surveys to the entire population – often proves misleading and inaccurate. It is with these deficiencies relating

to the type of population surveys that have been done so far in tropical Africa in mind, that we should view the achievements in tropical cardiology in Africa over the last four or five decades.

There is still a great deal to learn with regard to cardiovascular disease in Africa. In the last five decades cardiology in Africa has moved from affirming that diseases which occur in Europe also occur in Africa; to recognising the specific nature of the problem of cardiovascular disease in Africa and that inter-racial comparisons are only of value in so far as the answers as to why these differences occur provide new insights into the understanding of disease aetiology. More importantly, one must accept that the social importance of cardiovascular disease in Africa is no longer in doubt. In reporting the Mamprobi findings we wrote: "Despite repeated reports of hospital cases of various cardiovascular disorders, the general impression has remained that cardiovascular disease was not a major public health problem in tropical Africa, because of the sparse nature of the prevalence data available. This study, however, indicts that the prevalence of abnormal cardiovascular findings in an urban population aged 15 to 64 years is 25%. The major contributions to this high prevalence are hypertension and unexplained cardiomegaly. Valvular disease and electrocardiographic abnormality contributed little to this high prevalence"⁵⁶. One should add that as far back as 1962, Miller and co-workers⁶⁸ wrote of Gabon: "a large amount of unexplained cardiac disease exists, manifested mainly by 'organic' systolic murmurs, cardiomegaly or abnormal ECG's". It would seem therefore, despite the sparseness of truly scientific assessments of global cardiovascular disease manifestation in African populations, that the evidence although still scanty, already exists and is compelling; that cardiovascular disease is an important public health problem.

Despite the sketchy information, disease pattern studies^{28, 64, 72, 77, 83} do also indicate clearly the relative importance of cardiovascular disease. *There is no doubt that quantitatively, infectious disorders still present an overwhelming health problem; but deaths from cardiovascular disease occur most frequently in the 5th and 6th decades. I consider these decades to be economically the most important age groups to this country.* Mial et al.⁶⁷ in a longitudinal survey of a small Jamaican population aged 35 to 64 years, the Lawrence Tavern study – found that in this population, 21 out of 36 deaths (58%) occurring in males during a 5 year follow-up period were due to cardiac and cerebro-vascular causes; while among the female population, similar deaths occurred in 14 out of 28 deaths (50%). They commented: "It is apparent that cardiovascular disease, particularly heart disease, is the main cause of death in these rural Jamaican adults..." Notwithstanding these alarming but scattered and seemingly ignored data, there is clear evidence that the incidence of a number of cardiovascular diseases is increasing, in association with social change and economic development. Hypertension has always been with us but in the sense that populations with low average blood pressures and a low incidence of hypertension are disappearing either by aculturation or extinction; the prevalence of hypertension may be said to be increasing. Even if it were not, the hypertension prevalence rates in most populations studied in Africa would seem to be among the world's highest. Although coronary heart disease is still relatively rare, the stage is being set in Africa for the development of this disease in similar epidemic proportions as has been the case in developed countries. The incidence of rheumatic heart disease is also probably increasing. Idiopathic cardiomegaly – whatever its cause might be, affects in considerable numbers the economically most important among our populations.

It is extremely likely that the whole story has not yet been told; and if I may, I shall quote Sir George Pickering who felt the need to use an old Roman saying in his preface to the first ever symposium on Blood Pressure and Hypertension in Africa: "Africa with its extraordinary differences in the behaviour of peoples, offers one of the great opportunities for the epidemiologist. In this, as in other fields, EX AFRICA SEMPER ALIQUID NOVI." But is there reason any longer for the cardiologist in Africa to be taken by surprise? In developed countries, data on the magnitude of cardiovascular disease have not been obtained from cross-sectional surveys, but from national vital statistics. Despite the accepted difficulties in respect of the diagnostic validity of death certificates, they provide reasonably accurate information on a much wider scale and in a more meaningful way than does cross-sectional surveys. UCH Ibadan over a 12 year period recorded 472 cases of cerebro-vascular accidents, on admission rate of just over 3 per month. However, in 1972 a stroke register was set up in the same hospital under the auspices of the World Health Organisation – covering Ibadan and Epe; a much smaller population than the normal catchment area of this hospital. During the first eight months of this longitudinal study, the register had collected 130 cases or just over 16 cases per month – a five fold increase in the amount of recorded cases⁷. This more than anything emphasises the probability that cross-sectional surveys have so far underestimated the magnitude of our cardiovascular disease problem and emphasises also the urgent need for national vital statistics.

Mr. Vice-Chancellor, it is not only fables that must end with a moral. This has been a tale – though not a fable, that ends with a moral; in fact it has been a tale with several morals. The first has been that no matter how alarming the available data on cardiovascular disease in Africa may seem, the likelihood is that this information underestimates the problem. The se-

cond is that it is clear that while infections and communicable disorders take their high toll among infants and children, cardiovascular disease exacts its own toll among the age group which constitutes the parents of these children. The consequent deterioration in the economic status of afflicted families may well contribute to the high death rates from preventable disorders. No doubt they ever develops without planning; and none ever planned without providing itself with adequate data beforehand. There is need in our countries for national vital statistics and this last hour has been spent in spelling out their importance in one area of the field of non-communicable diseases. The third moral is that whatever difference may exist in disease prevalence or disease pattern between us and the developed West – these differences are not fixed; they should not leave us with satisfaction in their observation, but should prod us into studying these differences as a basis for understanding the nature of disease; its cause and its prevention. The last few decades have shown us that these differences disappear; it should therefore make us show concern for the future. Lastly, I shall end by quoting freely from my recent paper on cardiovascular epidemiology in Nigeria⁶:

In 1963 Maurice Campbell¹⁹ observed that deaths from disease of the circulatory system including vascular lesions of the central nervous system, increased little in the United Kingdom from 1876 to 1920. Using this period as baseline, he observed that by 1959, deaths from all heart disease had risen 2.6 times, vascular lesions of the central nervous system 2.2 times and coronary heart disease 7.0 times. During the period 1920 to 1959, deaths from communicable diseases were falling, expectation of life was increasing and social affluence rising... It is to be noted that this state of affairs: changes in communicable disease rates and in expectation of life – is not dissimilar to that now

prevailing in Nigeria. In Ghana we studied the relation of risk factors to coronary heart disease, to socio-economic status. In each case there is a strong positive correlation with socio-economic status. While socio-economic advancement will not be expected to affect the prevalence of the various cardiovascular disorders the same way and infact some probably not at all; the evidence at present points to the expectation that our social burden of cardiovascular disease will increase, unless measures are taken now to arrest changes which we know have occurred in other developed countries. This really is the task before the cardiovascular epidemiologist in tropical Africa today.

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