Family Planning Association and the Brook Centres has largely failed to contact social classes 4 and 5—who make up the greater part of the population. Local authorities were given power under the Family Planning Act of 1968 to set up birth control clinics, but few seem to have done so. Sir John Peel comments that "gynaecologists as well as general practitioners are getting more and more incensed by the increasing demand that they should recommend and carry out abortions within the Health Service for purely social convenience and yet are prevented from prescribing the drugs and issuing the appliances necessary for effective contraceptive practice as freely within the Health Service as any other drugs and appliances, unless there exists what is called a medical indication." Is this the answer? Is there a good case for including contraceptives in the N.H.S. whatever the indication?

If it became accepted that "you get your contraceptives from your G.P." there might be less embarrassment about asking for them. Family doctors know how often mothers of problem families are advised to use contraceptives but do not: the logical solution is for the doctor himself to do something about it. He can recognize many patients at risk as they come into his surgery for routine consultations: he should be able to offer them a prescription or fit an appliance on the spot. Just as important is contraceptive advice for women in hospital for abortions or delivery of babies. Referring the patient to a clinic or another doctor too often means that nothing is done.

A major objection to free contraception is the cost—perhaps £20 million a year—described by the Secretary of State as a gratuitous waste of taxpayer's money. Some doctors may argue that people should be encouraged to take more and not less personal responsibility, and that free contraceptives would be yet another example of the molly-coddling of the Welfare State. But how many abortions and illegitimate births would need to be prevented to balance the account? The effects of such a change can only be guessed at, yet no other alternative has been put forward which seems likely to have any significant effect on the current rising trends in unwanted pregnancies. A second objection is that family doctors, already overworked, would be asked to take on yet another item of service. Again the effects can only be estimated, but there should be some reduction in the number of consultations for abortion. It would certainly help if the Review Body could be persuaded to price this service.

So this would be the bargain. On the one hand doctors should refuse to compromise their principles and should not accede to popular pressure for abortion on demand. This attitude can be supported only by an unambiguous commitment by Government, family doctors, and gynaecologists to offer contraceptive advice and appliances within the N.H.S. to all comers (with the proviso that in this field as in any other, sincerely held religious convictions must be respected, and that most doctors will do their best to dissuade teenagers from sexual experience at too early an age). Once such a service is available, unprotected sexual intercourse resulting in unplanned pregnancy can be seen for what it is in an age of effective contraception—an example of thoughtless self-indulgence.

[1] *British Medical Journal*, 1971, **2**, 478.
[2] Borrie, W. D., *The Growth and Control of World Population*. London, Weidenfeld and Nicolson, 1970.
[3] Select Committee on Science and Technology, *First Report: Population of the United Kingdom*. London, H.M.S.O., 1971.
[4] *Registrar General's Statistical Review of England and Wales for the year 1969: Supplement on Abortion*. London, H.M.S.O., 1971.
[5] *Findings of an Inquiry into the First Year's Working of the Act conducted by the Royal College of Obstetricians and Gynaecologists. British Medical Journal*, 1970, **2**, 529.
[6] Claman, A. D., Wakeford, J. R., Turner, J. M. M., and Hayden, B., *Canadian Medical Association Journal*, 1971, **105**, 35.
[7] Diggory, P. L. C., *Lancet*, 1969, **1**, 873.
[8] *British Medical Journal*, 1971, **2**, 484.

The Masai's Cholesterol

The Masai of East Africa have long held a particular fascination for the visitor as well as the scientist because of their physical and cultural characteristics and their adherence to a traditional nomadic way of life. Over the past decade there has been considerable interest in the dietary habits and physical activity patterns of the nomadic peoples of East Africa (of which the Masai are one) in relation to hypotheses about atherosclerosis and coronary heart disease. Their nomadic pastoralism entails a fair amount of physical activity, and their diet consists of meat, milk, and blood. This clearly is a natural setting for the investigation of diet, blood cholesterol levels, and atherosclerosis. The most recent report on the Masai,[1] summarizing studies which will appear in greater detail elsewhere,[2] has a bearing on the general problem of atherosclerosis in man.

The story as presented is as follows. The Masai have a staple diet of milk, meat, and blood. The milk contains a fairly high proportion of total lipid, phospholipids, and cholesterol. The average daily calorie intake is about 3,000, with 66% of calories derived from fat. The average daily cholesterol intake is 500 to 2,000 mg, which is comparable to the average in the U.S.A. Despite their customary high-fat diet the aortas and coronary arteries of the Masai show little atherosclerosis. Serum cholesterol levels are low, with no increase in level with increasing age. The serum levels of beta-lipoprotein are low and of pre-beta-lipoprotein low or absent. Pregnant Masai women show no increase in serum cholesterol level in the first and second trimesters of gestation, but in the third trimester there is a 50% increase over the non-pregnant level.

An experiment was designed to investigate "the basic mechanisms that protect the Masai from hypercholesterolaemia on a diet rich in cholesterol and animal fat during their entire lifetime". Masai students, aged 18 to 24, at an agricultural school were divided into two groups and given a basic diet low in saturated fats and free of cholesterol. One group received 2 g crystalline cholesterol daily with a trace dose of cholesterol-4-^{14}C mixed in the basic diet, and the other group received only the trace dose of cholesterol-4-^{14}C in the diet. The trace dose was discontinued after eight weeks, and the decreasing curve of tracer activity was followed for another six months. The amounts of cholesterol absorbed, synthesized, and excreted per day were calculated from weekly blood and stool samples, and turnover times and pool sizes of the total body exchangeable cholesterol were determined.

Despite the daily dose of 2 g cholesterol to the experimental group the results showed no significant difference between the two groups in serum cholesterol, phospholipid, triglyceride levels, and lipoprotein patterns. The size of the exchangeable body cholesterol pools of the two groups remained constant, and the turnover times and turnover rates of body cholesterol were the same. But endogenous synthesis of cholesterol in the experimental group was about half that in the control subjects. The investigators concluded that this suppression of synthesis of cholesterol by

the feeding of cholesterol compensated perfectly for the influx of dietary cholesterol absorbed from the intestine, and that this was the only homoeostatic mechanism which protects the Masai from the development of dietary-induced hypercholesterolaemia.

The study also showed unusual serum protein patterns in the Masai, with levels of IgA almost twice as high as in American white persons, and reached very early in life. In studies of gall-bladder bile the Masai showed patterns very different from many white groups (U.S.A., New Zealand, Finland), with a high ratio of phospholipid to cholesterol and of bile acid to cholesterol. The authors concluded that the gall-bladder bile of the Masai has an enormous reserve capacity to dissolve cholesterol and that this system protects them from cholesterol-gallstone formation.

All the characteristics described above are regarded as reflecting a long-term biological adaptation, and the investigators regard them as genetically transmissible traits unique to the Masai. Ethnic groups may well differ in their biological mechanisms, but before accepting the new hypothesis we must be reasonably sure that the findings cannot be explained by the Masai's mode of life.

An assumption made in these and in previous studies[3] is that the Masai consume a diet rich in cholesterol and animal fat *during their entire lifetime*. This, however, is not true, and indeed, the body build of these people makes it unlikely that they live on more than a subsistence level of intake. Seasonal variation in the availability of food is great, and famine and famine-relief are unfortunately familiar features of their life pattern. The fat content of their cow's milk has a considerable seasonal variation, and the fermentation procedure the Masai use can considerably reduce the total lipid, cholesterol, and phospholipid. The amount and type of fatty acids in meat depends largely on the food supply and grazing habits of the animals, and M. A. Crawford has drawn attention to the striking differences between domesticated and wild animals.[4] There are no available data expressing in quantitative terms the food intake of the Masai or of any of the other nomadic East African groups over a long period of time. The low levels of serum cholesterol and beta-lipoprotein are those seen in communities on low intakes of saturated fat. We know that it requires a long continued intake of a diet high in saturated fats to maintain high blood cholesterol levels, but we know little if anything about the effects on cholesterol metabolism of an irregular high-fat diet interspersed with periods of want. We know that the blood cholesterol levels of young Samburu warriors (a related Masai-speaking group from northern Kenya) can fall considerably on a change of diet when entering the Army,[5] and it is possible that this phenomenon and the levels reported in the present and previous Masai studies reflect the substitution of carbohydrate for protein and fat. We also know from studies on the camel-herding Rendile nomads of northern Kenya that a similar way of life to the Masai and Samburu can be associated with blood cholesterol levels similar to those seen in affluent societies.[6]

This recent study of the Masai is part of a long continued debate about a fundamental aspect of cholesterol metabolism. C. B. Taylor and R. G. Gould[7] had in 1950 reported compensatory suppression of newly synthesized cholesterol in the liver and plasma of dogs ingesting a high cholesterol diet, and others subsequently found a similar homoeostatic mechanism in rats.[8] In 1951 Gould[9] proposed a hypothesis of quantitative human cholesterol metabolism, from which there arose two general concepts: firstly, that the human liver probably synthesizes 1 to 1·5 g of cholesterol per day; and secondly, that the human liver can suppress endogenous synthesis of cholesterol and thereby compensate for about 1·5 g of dietary cholesterol per day. This hypothesis has been criticized by Taylor and his colleagues.[10 11] Some extrahepatic tissue, which is unaffected by dietary cholesterol, synthesizes and delivers a large portion of plasma cholesterol at a constant rate; this may be the small intestine. Thus in Western man at any rate dietary cholesterol is additive to an essentially quantitatively unaltered endogenous supply of plasma cholesterol. This failure of ingested cholesterol to suppress endogenous synthesis is in part responsible for the rise of plasma cholesterol when cholesterol-containing foods are added to the diet.

Yet now the Masai are reported[1] to have a very efficient feedback mechanism for the suppression of endogenous cholesterol synthesis. In contrast to white people in the U.S.A., who have a limited maximal absorption capacity of 0·3 g cholesterol, the Masai can absorb more than 0·65 g cholesterol. Compared with the 25% suppression of synthesis found in U.S. white people, the Masai can suppress 50% of their endogenous cholesterol synthesis. The evidence clearly requires a re-evaluation of previously held beliefs or a new hypothesis, and the investigators have come down firmly on the side of the Masai having unique biological characteristics which are genetically determined.

In affluent societies, where degenerative diseases of many kinds are endemic or epidemic, it becomes increasingly difficult to recognize what is biologically normal—that is, related to and conducive to good health. The mean blood cholesterol level rises progressively with age and serves remarkably well as an index for susceptibility to atherosclerosis and its complications. The mean blood pressure rises progressively with age, and the levels are associated with morbidity and mortality. Are these "normal", or should we be looking beyond our restricted environment for help in defining normality?

[1] Biss, K., Ho, K-J., Mikkelson, N., Lewis, L., and Taylor, C. B., *New England Journal of Medicine*, 1971, **284**, 694.
[2] Biss, K., *et al.*, *Archives of Pathology*, 1971, May.
[3] Mann, G. V., Shaffer, R. D., Anderson, R. S., and Sandstead, H. H., *Journal of Atherosclerosis Research*, 1964, **4**, 289.
[4] Crawford, M. A., *Lancet*, 1968, **1**, 1329.
[5] Shaper, A. G., Leonard, P. J., Jones, K. W., and Jones, M., *East African Medical Journal*, 1969, **46**, 282.
[6] Shaper, A. G., and Jones, K. W., *Lancet*, 1962, **2**, 1305.
[7] Taylor, C. B., and Gould, R. G., *Circulation*, 1950, **2**, 467.
[8] Frantz, I. D., jr., Schneider, H. S., and Hinkelman, B. T., *Journal of Biological Chemistry*, 1954, **206**, 465.
[9] Gould, R. G., *American Journal of Medicine*, 1951, **11**, 2091.
[10] Taylor, C. B., Mikkelson, B., Anderson, J. A., and Forman, D. T., *Archives of Pathology*, 1966, **81**, 213.
[11] Taylor, C. B., and Ho, K-J., *Archives of Pathology*, 1967, **84**, 3.

Emotional Stress in Hypertension

People with essential hypertension usually show an abnormally large rise of pressure when emotionally or physically stressed. But the possible causes of it (indeed, the existence of the phenomenon itself) have been argued about for several decades.[1] Most evidence suggests that the rise of blood pressure is not only higher absolutely but also as a percentage of the initial value. P. J. Nestel[2] attempted to correlate this with catecholamine secretion, and reported that patients with labile hypertension secreted more catecholamines in response to emotional stress than did control subjects.

Such observations are possibly relevant to the aetiology