Summary

Kwashiorkor appears to be one of the major nutritional diseases. It has been recognized by many African tribes, who have their own names for it.

In many parts of the Tropics and sub-Tropics the disease is extremely common. It attacks children at a time when their protein requirements are high.

The disease has been described not only in Africa but also in tropical America and in Asia, and may occur in some of the poorer towns of southern Europe.

Although African children grow well while fully breast-fed, many of them fail to put on weight satisfactorily, develop brown hair, and have attacks of diarrhoea in the weaning and post-weaning periods. These children are believed to suffer from mild kwashiorkor.

Tropical infections complicate but do not cause kwashiorkor.

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REFERENCES

- Adams, F. (1939). *Genuine Works of Hippocrates*, p. 24. Baltimore.
- Babiet, J., and Normet, L. (1937). *Bull. Acad. Méd., Paris*, 117, 242.
- Ballabriga, A. (1951). *Transactions of Sixth International Congress of Pediatrics, Zurich*, p. 131.
- Banks, A. L. (1951). *Lancet*, 2, 258.
- Brock, J. F., and Autret, M. (1952). World Health Organization Monograph No. 8. Rome. *Bull. Wild Hlth Org.*, 1952, 5, 1.
- Choremis, C. (1951). *Transactions of Sixth International Congress of Pediatrics, Zurich*, p. 130.
- Davies, J. N. P. (1952). *Ann. Rev. Med.* In press.
- Frontali, G. (1946). Quoted by Meiklejohn and Passmore (1951).
- Gillan, R. U. (1934-5). *E. Afr. med. J.*, 11, 88.
- Housden, L. (1950). *British Medical Journal*, 2, 456.
- Latham, D. V. (1934-5). *E. Afr. Med. J.*, 11, 358.
- Lieurade, M. (1932). *Bull. Soc. Path. exot.*, 25, 46.
- MacPherson, A. (1951). *Lancet*, 1, 53.
- Meiklejohn, A. P., and Passmore, R. (1951). *Ann. Rev. Med.*, 2, 129.
- Normet, L. (1926). *Bull. Soc. Path. exot.*, 19, 207.
- (1927). *Transactions of Seventh Congress, Far Eastern Association of Tropical Medicine, Calcutta*, 1, 109.
- (1937a). *Bull. Soc. sci. Hyg. aliment., Paris*, 25, 153.
- (1937b). *Bull. Acad. Méd., Paris*, 117, 239.
- Payne, G. C., and Payne, F. K. (1927). *Amer. J. Hyg.*, 7, 73.
- Petrides, E. P. (1948). *J. Pediatr.*, 32, 333.
- Procter, R. A. W. (1927). *Kenya med. J.*, 3, 284.
- Ribadeau-Dumas, L., Mathieu, R., and Lévy, M. M. (1927). *Bull. Soc. méd. Hôp. Paris*, 51, 1157.
- Salber, E. J., and Bradshaw, E. S. (1951). *Brit. J. soc. Med.*, 5, 113.
- Stannus, H. S. (1934). *Arch. Dis. Childh.*, 9, 115.
- (1935). *Lancet*, 2, 1207.
- (1936). *Trop. Dis. Bull.*, 33, 729, 815, 885.
- Stott, H. (1948). Clinical and Aetiological Investigations into Kwashiorkor. A thesis for the degree of M.D., Cambridge University.
- Trowell, H. C. (1937). *Arch. Dis. Childh.*, 12, 193.
- and Muwazi, E. M. K. (1945). *Ibid.*, 20, 110.
- Véghelyi, P. V. (1948). *Acta paediatr., Stockh.*, 36, 128, 483.
- Welbourn, H. F. (1951). *E. Afr. med. J.*, 28, 428.
- Williams, C. D. (1932). Gold Coast Annual Report Medical Department for 1931-2, Accra. Appendix E, p. 93.
- (1933). *Arch. Dis. Childh.*, 8, 423.
- (1935). *Lancet*, 2, 1151.
- (1938). *Ibid.*, 1, 97.
- (1940). *Trans. roy. Soc. trop. Med. Hyg.*, 34, 85.
- Woodruff, A. W. (1951). *British Medical Journal*, 2, 1415.

The report of this year's annual conference of the National Association for Maternity and Child Welfare is now available. The theme of the conference was "The Wastage of Child Life before and after Birth." The report contains abstracts of papers read at the conference and an account of the discussions. The principal papers were: "The Problem and the Facts" (C. Fraser Brockington); "Infertility and Abortion" (Margaret Hadley Jackson); "Neonatal Deaths" (Dugald Baird); "Infant Deaths" (Thomas McKeown); and "Prematurity" (J. Marshall Scott). Copies of the report cost 5s., and may be obtained from the National Association for Maternity and Child Welfare, Tavistock House North, Tavistock Square, London, W.C.1.

KWASHIORKOR

II. CLINICAL PICTURE, PATHOLOGY, AND DIFFERENTIAL DIAGNOSIS

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Clinical Picture

Digestion and Appetite

The transition from the mild to the severe form of kwashiorkor is signalized in many instances by the appearance of loose stools. The diarrhoea may be persistent or intermittent; rarely, it lasts for only a week or two and then subsides. Vomiting is not usually an important feature of the earlier stages.

The stools, by their character and consistency, usually show the kind of food eaten; thus, when the staple food of the child has been the cooked banana (plantain), they are bulky and semi-solid, and contain large particles of recognizable food. They may completely lack any faecal odour. Fats, and even moderate amounts of pure sugars, are poorly tolerated. Full-cream milk will therefore often produce a brisk diarrhoea, and even skimmed milk cannot be given in unlimited quantities (Dean, 1952).

Appetite varies from child to child. It may be fairly good, but tends to be fastidious, and in the later stages of all severe cases is lost entirely.

Weight, Age, and Bone Age

Children suffering from advanced kwashiorkor are always much underweight, in spite of the presence of the oedema. The weights of 59 children admitted to Mulago Hospital, Kampala, with the diagnosis of kwashiorkor, and in whom no other disease was subsequently discovered, are shown in Fig. 1. The standards for U.S. boys and girls given by Nelson (1950) and for Uganda children by Welbourn (1951) have been added. The children with kwashiorkor were all below the Uganda standard, although they were oedematous. Most of the older children were girls.

FIG. 1.—Weights of children admitted to Mulago Hospital with the diagnosis of kwashiorkor. The children were all below the Uganda standard, although they were oedematous. Most of the older children were girls.

The average weight of the oedema fluid, calculated from the weight loss during the first 7 to 10 days of successful high-protein treatment, was about 1 lb. 12 oz. (840 g.). Fig. 1 also shows that most of the older children were girls; their average age on admission was nearly 24 months, whereas that of the boys was only 20 months. This difference may indicate the greater resist-

ance of the girls to the onset of the disease; the children of both sexes were presumably weaned at the same time, but the development of a state severe enough to cause the parent to bring the child to hospital may have been a slightly longer process in the girls.

The bone ages of 42 of the children (21 of each sex) were calculated from the data of Greulich and Pyle (1950). The average stated age for the girls was 24 months and that for the boys 21 months. The average bone age for the girls was 19 months and that for the boys 18 months. The older children of both sexes were, on the whole, farther in advance of their bone ages than the younger children. Although it may be argued that American standards should not be applied to African children, the figures suggest that there may have been some retardation of bony development.

Oedema

Oedema is a constant feature of all advanced cases. It usually starts in the feet, but the face is soon involved. Eventually it may become very severe, and the scalp, the hands, and the genitalia (especially the penis) may swell. Ascites is, however, very rare, and effusions into joints have never been detected. The oedema tends to accumulate in the dependent parts, and it is common to see that side of the face on which the child has lain more swollen than the other. There are no signs of congestive failure or of peripheral neuritis. The urine often contains a little albumin, but red blood cells, leucocytes, and casts are uncommon. The blood pressure is never raised; as with the pulse and the temperature, any deviation from the normal for the age is in the direction of decrease rather than increase. The very slow pulse often accompanying hunger oedema in adults is not found in kwashiorkor.

Subcutaneous Fat

During life, and at necropsy, it is often possible to demonstrate, even in advanced cases of kwashiorkor, a fair amount of subcutaneous fat. The muscles tend to be weak and thin, but their condition is partly concealed by the fat and by the oedema. In children who survive many weeks, especially in those who have much diarrhoea, all subcutaneous fat may be lost.

The Skin and Mucosa

The dermatosis which is present in nearly all severe cases of kwashiorkor is most distinctive. It is doubtful if anything exactly the same occurs in any other disease. Because of the appearance at one stage, it has been called "the crazy-pavement dermatosis"; but this term describes only a transient state. The dermatosis almost always appears first in the inguinal region; later the whole of the napkin area and the axillae are invaded, and then other sites of pressure and irritation, such as the thighs, chest, and shoulders. The lesions are less frequent on the face, neck, arms, and lower part of the legs, and the feet.

The dermatosis has a definite evolution. It begins with dusky patches that look as though the skin has been stained with a purple dye. The patches rapidly darken, and their edges seem to deepen, giving the appearance of irregular paving. The skin loosens as though fluid were collecting underneath, then breaks and is lost, revealing weeping paler areas. These enlarge and coalesce, so that in the inguinal region, for instance, all the skin for several centimetres on either side of the fold is changed. In one child who was under constant supervision all the stages described were passed through in less than a week.

The degree of the desquamation depends to some extent on the severity of the general condition. The fully developed dermatosis is a serious sign, occurring only in children who are within a few days, or weeks, of death. It does not occur in moderate kwashiorkor, which may show no skin changes at all.

There are other skin changes which often accompany severe kwashiorkor. There is a tendency towards pallor of the skin, which may be generalized over all the body, or

show particularly in the face. It may sometimes take the form of scattered small patches. Deep bleeding fissures may appear in any of the flexures and in sites such as the juncture of the pinna and the scalp; fingers that are habitually sucked may become sodden and white and lose all their superficial skin; deep, circular ulcers, with a grey slough, and an edge that shows the minimum of inflammatory reaction, may occur on the dorsum of the foot and elsewhere. A curious feature of all the skin lesions is that they do not seem to be painful.

The mucosae do not often show any notable change except that the tongue may be rather brightly red; sometimes it is remarkably smooth all round its forward edge. A fissure is sometimes seen in the middle of the lower lip, and mucocutaneous junctions, especially those at the corners of the mouth, are also fissured occasionally. There is often some loss of pigment along the buccal border of the lips.

Thorax and Abdomen

At Mulago there is seldom any gross enlargement of the liver, although the edge is usually palpable below the costal margin. The spleen is enlarged only if there has been much malaria. Although the child usually has a cough, which seems to be unproductive, there is little to be found on examination of the chest by percussion or auscultation, except a few moist sounds at the lung bases. As the chest wall is thin, the normal puerile breathing is heard unusually clearly. X-ray examination of the chest also reveals very little, although in some children there are areas of the lung fields which show a curious increase in the density of the markings. This may indicate the presence of an exudative condition analogous to the collection of fluid elsewhere in the body, but in no case has an actual pleural effusion been found.

The Hair

The hair is almost always affected. It is pale in colour and may be almost flaxen; sometimes, however, it may be reddish. It is thin, straight, and soft; it loses its sheen and is lifeless. The thicker hairs in other parts of the body, such as those of the eyebrows and eyelids, are resistant to this colour change, but eventually it affects them too. The fine downy body hairs redden and are seen easily against the dark skin. There appears to be little correlation between the degree of the dyspigmentation and the severity of the kwashiorkor; in a very few of the children who are acutely ill crisp black hair is retained.

Demeanour

Mental apathy is profound, and occurs consistently in severe kwashiorkor; it is most unusual for a child to retain any semblance of alertness. The children lie in their beds or in their mothers' arms, taking no interest in their surroundings. They dislike any interference, and resist, feebly and petulantly, being undressed or examined. They cannot be induced to play or to smile. Their motor activity is reduced to the minimum, and perhaps for this reason, or possibly because of the presence of the oedema fluid, their limbs often feel remarkably cold. They present a picture of extreme misery (cf. Clark, 1951).

The Blood Picture

Some degree of anaemia is usually found in kwashiorkor even when the possibility of a tropical infection can be excluded. Naturally, if a blood-destroying disease such as malaria or hookworm is also present the degree of anaemia becomes more severe. It is possible by using a concentration technique to detect almost all cases of infestation by helminths: previous attacks of malaria and a low and fluctuating malarial parasitaemia are more difficult to exclude. In 44 children admitted to Mulago Hospital in whom no infection or infestation could be detected, the averages for haemoglobin and erythrocytes on the day of admission or on the next day were 8.46 g. per 100 ml. and 2,850,000 per c.mm. respectively. These figures indicate slightly more anaemia than that reported by Altmann and

Murray (1948) in Johannesburg (mean Hb 10.2 g. per 100 ml. and mean red-cell count 3,600,000 per c.mm.) and Walt, Wills, and Nightingale (1950) in Durban (mean Hb 10.1 g. per 100 ml. and mean red-cell count 3,270,000 per c.mm.). The figures of Van der Sar (1951) obtained in Curaçao, if those for one extremely anaemic child are omitted, averaged 9.3 g. Hb and 3,230,000 red cells. Johannesburg, Durban, and Curaçao are all places in which tropical blood-destroying diseases are virtually absent.

There is agreement that the anaemia responds but slowly to treatment by a high-protein diet. At Mulago, after 21 to 28 days, the 44 children had improved on an average by only 0.84 g. of Hb and 430,000 red cells, and none of the investigators in Johannesburg or Durban could report better results. There is fairly general agreement that the anaemia does not respond to folic acid, vitamin B₁₂, or liver extracts (Wills, 1951, personal communication). Sometimes, however, it responds to iron. The anaemia does not present clear or constant morphological features. It is usually normocytic or slightly macrocytic, and is often slightly hypochromic. The bone marrow is seldom, if ever, megaloblastic. The anaemia has many features in common with that seen in men and pregnant women who are also suffering from protein deficiency (Trowell, 1951; Woodruff, 1951).

The aetiology of the anaemia is almost certainly not exactly the same in all places. It may have different characters if it is mild or if it is severe, and it seems that treatment may bring about changes in the kind of anaemia

FIG. 2.—Relation between red-cell count and colour index in 56 of the children whose weights are shown in Fig. 1. There was an inverse relationship between the cell count and the index.

by accelerating at different rates the production of haemoglobin and erythrocytes.

Some of these tendencies are illustrated by findings at Mulago. In Fig. 2 the red-cell count on admission of 56 children diagnosed as suffering from kwashiorkor has been plotted against the colour

index, and it seems that the index was higher as the cell count was lower. (A similar result was not obtained by plotting haemoglobin against colour index.) The average colour index was about 1.05. As the result of treatment, the index fell, and when the children were discharged was approximately 1. The corresponding indices of the Durban children (calculated from the average figures given, and therefore not entirely accurate) are 1.07 and 1.01. It seems reasonable to conclude from these two sets of figures that, as the anaemia improved, the rate of haemoglobin formation did not keep pace with the increase in the number of red cells.

Mode of Death in Kwashiorkor

Kwashiorkor, once it has reached the severe stage with anorexia, oedema, digestive failure, and gross dermatoses, is usually fatal, and even in hospital recovery has until quite recently been exceptional. In spite of this, little has been written about the mode of death, possibly because that is not fully understood.

Pneumonia occurring in lungs full of exudate, or the exudative condition itself, probably accounts for most of the deaths in Mulago. Many deaths are sudden. A child who has been seen and thought to be in no special danger, who may or may not have some diarrhoea and a mild degree of dehydration, and who may have no discoverable infection, may die within a few hours without warning, having shortly before taken a substantial feed. In such children, necropsy, although it may disclose the

expected state of the liver and pancreas (see below), usually fails to reveal any cause of death. The real cause is presumably to be found only in changes in tissue and serum chemistry, but, so far, the nature of those changes has not been determined. If a study is undertaken it should take into account the possibility of an acute hypoglycaemia, caused perhaps by a failure in the mechanisms for the utilization of carbohydrate. For such a failure either the damaged liver or an abnormal protein-carbohydrate conversion might be found to be responsible.

Some children whose history is typical of kwashiorkor present an appearance of emaciation and dehydration when they are brought to hospital, and occasionally a child who was oedematous when first admitted has been seen to drift into this state. The dehydration does not respond readily to increased fluids given orally. There may be no pyrexia, no vomiting, and little diarrhoea, and feeds containing amounts of fluid which should be adequate to cover all the observed losses are taken readily. Some of these children appear to die from the effects of dehydration. It seems impossible for the loss of water to have occurred except through the kidneys, but the losses by that route, and the changes in electrolytes, have not yet been investigated, and the true cause of death in these cases remains a mystery.

Pathology of Kwashiorkor

A disease caused largely by a deficiency of dietary protein might be expected to affect every organ of the body, and particularly those organs which are concerned with the elaboration of proteins and protein-containing substances such as enzymes. The histological changes have been investigated by a number of workers, including Waterlow (1948), Davies (1948), Véghelyi (1948), Hartz (1949), Manson-Bahr (1951), and Van der Sar (1951). The pancreas and the liver have shown the most consistent changes.

The pancreas is affected early in kwashiorkor. There is at first a reduction in the zymogen granules, and later total atrophy of the cytoplasm. The nuclei are, however, well preserved, and the islets of Langerhans appear always to be intact. In parallel with the changes in the cells there is a fall in enzyme production, and the duodenal juice has less amylase, lipase, and trypsin (Thompson and Trowell, 1952). In these findings lies the most probable explanation of the fact that large quantities of undigested food are passed in the stools.

Observation of children who have been treated with high-protein diets, but who have died, suggests that with good treatment the histological appearance of the pancreas may rapidly revert to normal. In many children not so treated, however, the pancreatic atrophy persists, and a fibrosis of greater or less degree may occasionally develop. At Mulago no child has been found who showed changes resembling those of cystic fibrosis of the pancreas. The lung changes shown by x-ray examination (see above) do not include any condition similar to that often found to accompany cystic fibrosis in European and American children.

The parotid glands, the other salivary glands, and the gland cells of the gut are all affected in kwashiorkor, but the changes in the pancreas are probably more persistent than those elsewhere.

The fatty infiltration of the liver that is found in kwashiorkor was one of the first lesions to be described as characteristic. It begins in the peripheral cells of the liver lobule. The fat globules are initially small, but they increase in size and gradually encroach on the central areas, until in severe cases the whole lobule may be filled with fat. As the fat increases, lymphocytes, and sometimes polymorphonuclear leucocytes and eosinophil cells, accumulate in the portal triads. A fine perilobular fibrosis also develops, with strands reaching out from the triads and tending to delimit the lobules. The fibrosis increases by the collagenization of the reticulin fibres and the peripheral liver cells are strangulated. Adequate treatment may restore the liver completely to a normal appearance, but it seems

likely that if the fat remains long in the liver there is an increase in the stellate fibrosis, which in older children produces an appearance resembling that of a mild Laennec cirrhosis.

The function of the liver is known to be affected (Waterlow, 1948; Van der Sar, 1951), and the liver damage must surely be an important factor in the production of the alterations in serum chemistry which are found in kwashiorkor. These have been described by Dean and Schwartz (1952).

Differential Diagnosis

The recognition of the fully developed picture of kwashiorkor should not occasion much difficulty. The dietetic history of the child, and his physique, should go far towards establishing the diagnosis, and the characteristic dermatosis probably occurs in no other condition. Chronic dysenteries and typhoid should usually be distinguishable from kwashiorkor by pyrexia and the presence of pathogens or cellular exudates in the stools. Abdominal tuberculosis may cause more difficulty, but glandular enlargement, a positive Mantoux reaction, and a response to streptomycin should be of assistance. Coeliac disease and fibrocystic disease of the pancreas have not been found at Mulago, and seem to be at least rare in other places where kwashiorkor is common. In fibrocystic disease, as in kwashiorkor, the duodenal contents possess very little enzymatic activity, but in kwashiorkor, under proper treatment, the activity is restored.

General undernutrition, in which the chief deficiency is of calories, may be associated with oedema, but the association is rare in children. The undernutrition does not cause alterations in the colour of the hair and skin, and is always accompanied by much loss of subcutaneous fat. In a severe anaemia, due, for instance, to hookworms, the skin may become pale, but does not assume the curious bronze colour found in kwashiorkor—a distinction which can be appreciated readily with experience.

Another condition which sometimes resembles kwashiorkor, but does not cause dyspigmentation, is nephritis. In the Tropics this disease has some special features, but the urine will usually contain red cells and casts, and often the amount of protein in the urine will be greater than that found in kwashiorkor. The oedema does not usually respond so rapidly and completely to treatment.

It is clear that secondary effects, due particularly to failure of absorption, are likely to complicate kwashiorkor. It is in fact uncertain to what extent some of the signs described, such as the alterations in the tongue, and the cheilosis, are due to vitamin deficiencies which are secondary to malabsorption or to altered or suppressed synthesis in the gut. Similarly deficiencies of fat-soluble vitamins, of iron, or of other minerals may occur, especially if the home diet has been poor in those substances.

The similarity of the dermatosis of kwashiorkor to that of pellagra gave rise in the past to some confusion, and the term "infantile pellagra" is still used by some authors as a synonym for kwashiorkor. In kwashiorkor, however, the lesions do not develop so particularly in areas exposed to sunlight, and they follow their well-defined evolutionary pattern. There may be a superficial resemblance to the eruption seen in acute pellagra, and Gillman and Gillman (1951) believe that the histological appearances of both conditions are essentially the same.

The term "crazy pavement," which has been applied to the characteristic skin lesion of kwashiorkor, is in some ways unfortunate because it suggests two other conditions that are probably of quite different aetiology. It is extremely common to find patches of shiny skin on the shins of African schoolchildren who appear to be well nourished. There has also been described, notably by Platt (1945), a "crackling" of the skin, often at the same site. The first condition is the result of the slight, often-repeated trauma of weather or grasses; the second is said to occur in any state of general undernutrition. In both conditions the skin

appears glazed, and shows what seems to be an exaggeration in depth of normal skin lines, marking off small irregular rhomboid areas. In the true "crackled" skin friction may cause some detachment of flakes of the altered skin, but this is usually only from the most superficial layers. The pigment changes of kwashiorkor, and the complete desquamation, do not occur in either of these conditions.

Summary

The clinical picture of severe kwashiorkor is described. The transition from the mild condition is usually marked by a period of gastro-intestinal upset.

In spite of oedema, which is almost always present in advanced cases, the children are underweight, and bone development may be retarded. A fair amount of subcutaneous fat may be retained.

As a late manifestation, a unique dermatosis appears, and evolves in a characteristic way. The dermatosis has a different distribution from that of pellagra, but there may be superficial and histological resemblances. The hair usually becomes reddish or pale. In the absence of any tropical infection, anaemia is seldom severe. Apathy and misery are constant features.

Notable pathological lesions include the disappearance of zymogen granules from the pancreatic acini and the accumulation of fat in the periphery of the hepatic lobule.

The differential diagnosis from nutritional oedema, hookworm disease, nephritis, coeliac disease, and fibrocystic disease of the pancreas is discussed.

REFERENCES

- Altmann, A., and Murray, J. F. (1948). *S. Afr. J. med. Sci.*, 13, 91.
 Clark, M. (1951). *E. Afr. med. J.*, 28, 229.
 Davies, J. N. P. (1948). *Lancet*, 1, 317.
 Dean, R. F. A. (1952). *E. Afr. med. J.*, 29, 1.
 — and Schwartz, R. (1952). *Brit. J. Nutr.* In press.
 Gillman, J., and Gillman, T. (1951). *Perspectives in Human Malnutrition*, p. 89. New York.
 Greulich, W. W., and Pyle, S. I. (1950). *Radiographic Atlas of Skeletal Development of the Hand and Wrist*. Stanford, California.
 Hartz, H. (1949). *Docum. neerl. indones. Morb. trop.*, 1, 41.
 Manson-Bahr, P. E. C. (1951). *Trans. roy. Soc. trop. Med. Hyg.*, 44, 555.
 Nelson, W. E. (1950). *Mitchell-Nelson Textbook of Pediatrics*, p. 60. Philadelphia.
 Platt, B. S. (1945). *Brit. med. Bull.*, 3, 179.
 Thompson, M. D., and Trowell, H. C. (1952). *Lancet*, 1, 1031.
 Trowell, H. C. (1951). *Lancet*, 2, 761.
 Van der Sar, A. (1951). *Docum. neerl. indones. Morb. trop.*, 3, 25.
 Véghelyi, P. V. (1948). *Acta paediatr. Stockh.*, 36, 128, 143.
 Walt, F., Wills, L., and Nightingale, R. P. (1950). *S. Afr. med. J.*, 24, 920.
 Waterlow, J. C. (1948). *Spec. Rep. Ser. med. Res. Coun., Lond.*, No. 263.
 Welbourn, H. F. (1951). *E. Afr. med. J.*, 28, 428.
 Woodruff, A. W. (1951). *British Medical Journal*, 2, 1415.

The subject of punishment in schools is apt to arouse heated controversy. Recognizing this, the L.C.C. has recently issued a pamphlet (Staples Press, Ltd.) which is intended "to stimulate thought and calm discussion of a problem that must be very much in the minds of all concerned with the upbringing of children in these difficult times." The pamphlet points out that the employment of harsh methods by teachers tends to encourage the adoption of harsh standards by pupils when they leave school. It poses the question: If other methods have failed, is corporal punishment likely to succeed? It has been abandoned altogether in some schools, and the pamphlet suggests that a worth-while experiment is to abolish it from more. Sarcasm is condemned as a fear-inspiring and dangerous weapon for a teacher to use; children are particularly defenceless against it and cannot regard it as anything but unjust. As the pamphlet points out, what may be the right punishment for one child is the wrong one for another, and many forms of anti-social behaviour in children are attempts to compensate for an unsatisfactory emotional or intellectual development. Routine application of a standard punishment is seldom advisable. The pamphlet emphasizes the need to consider the social factors influencing character and conduct.