It is a pleasure to have this opportunity of expressing my thanks to my surgical colleagues, Mr. Cotterill, Mr. Cathcart, Mr. Caird, Mr. Hodsdon, Mr. Wallace, and Mr. Thomson, who conducted the operations on the patients belonging to this series of cases.

One or two matters, of more scientific than practical interest, may finally claim our attention. Some of the lantern slides, which, through the kindness of Professor Cunningham, you have had an opportunity of seeing to-night, show that the stomach may assume extremely varying forms under the influence of the muscular coat and its nervous mechanism, as well as in consequence of pathological conditions. Some of these forms we can even discover during life on clinical examination of the patient. The greater curvature of the stomach during life sometimes shows a distinct notch. Undoubtedly this may occur as the result of a spasm on the one hand, or of some cicatricial constriction on the other. An interesting point to which attention has in recent times been often directed, is the action of the pyloric sphincter. Undoubtedly pyloric spasm does take place under various influences, amongst which that of the nervous system cannot be excluded. By means of such a spasm a very considerable dilatation of the stomach may be produced, accompanied by distinct peristaltic movements. The same condition is seen more frequently in the intestines, where a constriction due to spasm may produce stasis with distinct intestinal patterns, and even waves of peristalsis, upon the front wall of the abdomen. The continuance or recurrence of pyloric spasm will undoubtedly in time bring about the development of hypertrophy, and this hypertrophy of the pyloric sphincter will in its turn produce, first a dilatation, and secondly a hypertrophy of the walls of the stomach. Congenital hypertrophy of the pylorus is a subject which has obtained a great amount of attention, and several theories have been advanced in explanation of it. None of these, save that of Thomson,¹⁴ appear to me in the least respect valid. The hypertrophy of the muscle must be, as Thomson holds, secondary to some excessive activity. This view has been strongly urged on another occasion by myself.¹⁵

The causation of some abnormal forms of stomach, and especially of the hour-glass type, has provided material for much speculation, but it is probable that we have now been put in possession of some facts upon which to reason in regard to this subject. The work of Cannon,¹⁶ based on feeding animals with pulpy food containing bismuth, and watching the movements of the stomach with the fluorescent screen, has given considerable impetus to the study of the movements of the stomach. He has shown that the stomach consists of two physiologically distinct parts, pyloric and fundal, of which the fundus is an active reservoir for the food, squeezing its contents gradually towards the pylorus, while the pyloric portion has a succession of waves passing from the middle portion of the stomach to the pylorus. It seems to me that in this graphic description of the normal functions of the stomach we have an explanation of the spasmodic type of hour-glass stomach. Perhaps you will allow me to quote the words of Moynihan¹⁷ upon this subject. He remarks: "On several occasions during the last few years, when operating for chronic ulcer, I have watched the stomach intently for several minutes, and have seen the onset, the acme, and the gradual relaxation of a spasmodic muscular contraction of its walls. Quite gradually the stomach narrows and the wall becomes thicker and almost white in colour; when taken between the fingers the contracted area feels like a solid tumour. The spasm may be so marked as to prevent a finger being invaginated through the segment affected. The appearance presented is very striking. I have seen it in the body of the stomach and at the pylorus, but never at the fundus. As slowly as it comes on, the spasm quietly relaxes and the stomach assumes its natural form." It is satisfactory to know that Cunningham in his recent valuable monograph on the varying form of the stomach¹⁸ fully agrees with Moynihan upon this subject. My own belief is identical with those two authors—hour-glass stomach is never found as a congenital deformity. Of hour-glass stomach as the result of some old-standing ulceration many examples have been before me. Their origin, however, is so clear that no time need be expended upon this matter. The practical bearing of this cicatricial type of biloculated stomach on clinical medicine, whether

as regards diagnosis or treatment, has been already fully discussed in this address.

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- ¹⁷ *BRITISH MEDICAL JOURNAL*, vol. i, 1904, p. 413 and p. 1288. ¹⁸ *Transactions of the Royal Society of Edinburgh*, vol. xlv, 1906, p. 9.

A Clinical Lecture

ON

INFANTILE SCURVY.

DELIVERED AT THE HOSPITAL FOR SICK CHILDREN,
GREAT ORMOND STREET,

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GENTLEMEN,—Infantile scurvy is often spoken of as if it were a disease of recent discovery and of modern development. Its recognition all over the scientific world is justly associated with the names of two distinguished members of the consulting staff of this hospital, Dr. Cheadle and Sir Thomas Barlow, to whose investigations is due almost all that is known of the pathology of the disease. But to suppose that infantile scurvy was unknown, or even that its true nature as a scorbutic condition complicating rickets was unrecognized until the nineteenth century is an error, and one that has crept into many textbooks.

As long ago as the middle of the seventeenth century, Glisson, in his *Treatise on the Rickets*, had not only described the characteristic symptoms of infantile scurvy, but, with more accuracy than some of his successors, had recognized that it was scurvy complicated with the rickets and not an acute form of rickets. In the English translation, published in 1651 (p. 249), he says: "The Scurvy complicated with this affect (rickets) hath these signs: (1) They that labour under this affect do impatiently endure Purgations; but they who are only affected with the Rachites do easily tolerate the same. (2) They are much offended with violent exercises, neither can they at all endure them. But although in this affect (rickets) alone there be a kind of slothfulness and aversion from exercise, yet exercise doth not so manifestly, at least not altogether so manifestly, hurt them as when the Scurvy is conjoined with the rachites. (3) Upon any concited and vehement motion they draw not breath without much difficulty, they are vexed with divers pains running through their Joynts, and these they give warning of by their crying. . . . (4) Tumors do very commonly appear in the Gums. (5) The urin upon the absence of the accustomed Feaver is much more intens and encreased."

Glisson (*ibid.*, p. 227) actually emphasizes what is not yet sufficiently recognized, that there is no essential connexion between infantile scurvy and rickets; he says "the Scurvy is sometimes conjoined with this affect," and after mentioning as causes heredity, infection, and the possibility that it may be "produced from the indiscreet and erroneous regiment of the infant," though he attributed it chiefly to climatic influences, he adds, "for it (scurvy) scarce holdeth any greater commerce with this Disease (rickets) than with other diseases of longer continuance."

Such terms as "scurvy rickets," "acute rickets," and "haemorrhagic rickets"—names which would imply that scurvy is a variety of rickets—have been responsible for much confusion. Obscure though the exact etiology of infantile scurvy may be, it is clear from clinical facts that this affection arises from causes entirely distinct from those which produce rickets; severity of rickets raises no special probability of scurvy; indeed the worst cases of scurvy

are often associated with very slight rickets, and in some cases with none. Both diseases are due to faults of diet, and it is likely enough that a diet which is faulty in one respect may be faulty in another; the particular fault which produces rickets may be, and no doubt often is, present when the diet shows also the particular fault which produces scurvy. But the fault is not one and the same; witness the many infants who suffer with severe rickets after being fed from an early age with potato, which would seem when given at that age to favour if not actually to cause the occurrence of rickets, whereas there is probably no more powerful agent than potato in the prevention or cure of infantile scurvy.

The clinical picture of the fully-developed disease is striking enough; an infant who has been fed upon one of the patent foods, with or without milk, or on milk which has been condensed, sterilized, or otherwise altered, has been ailing for some weeks, has taken food badly, and probably lost weight. Moreover the mother says it cries whenever it is touched, and, as she puts it, "has lost the use of its limbs." The infant is pale, it lies quiet perhaps until it is approached, when it cries out in obvious dread of being touched; the legs lie motionless, usually with the thighs slightly abducted and everted and the knees slightly flexed; the arms are less often affected. There may be some swelling of part of one or other of the limbs, obliterating the natural curves. Any handling of the affected limbs causes a piteous cry, evidently of acute pain. If teeth are present the gums around them are swollen and purple, occasionally projecting like a mass of granulations, almost completely hiding the teeth, and bleeding readily when touched. The urine is perhaps smoky, if not red with blood.

Such in outline is the characteristic picture of infantile scurvy, which I shall now consider in more detail, chiefly with reference to diagnosis and treatment.

Age-Incidence.

The age at which infantile scurvy begins is a point of considerable importance in diagnosis. As the accompanying chart of fifty-four cases under my own observation shows, its onset is almost limited to the later half of the first year of life, and in three-fourths of the cases the disease begins between the age of 6 months and 10 months.

Chart showing age onset in fifty-four cases of infantile scurvy.

So marked is this special age-incidence, that given an infant of 6 to 12 months and a history of tenderness in the limbs, the diagnosis of scurvy should at once suggest itself as a possibility. In several of the cases in this series the disease had been diagnosed as "rheumatism"—a mistake which ought never to occur, for rheumatism is practically an unknown disease under the age of 18 months, and indeed is exceedingly rare under the age of 3 years. This chart shows also that in no case did scurvy begin before the age of 5 months—a point which distinguishes this disease from many cases of syphilitic epiphysitis which begins most often under the age of 3 months.

Mode of Onset.

So much stress has been laid upon the affection of the gums and upon the tender swellings in the limbs in infantile scurvy that there is danger of forgetting that in many cases there are other more insidious symptoms which may appear some weeks before these obvious manifestations. An infant who has thriven hitherto upon

some patent food begins to ail, the weight ceases to rise or actually falls, the feeds are taken badly, and the child is fretful and miserable; there is no swelling of limbs, perhaps no definite tenderness anywhere, and the gums are perfectly normal; but that these early symptoms indicate scurvy may be shown not only by the rapid improvement within two or three days on antiscorbutic treatment, but also in some cases by the presence of blood in the urine even in this early stage.

I doubt whether the onset of scurvy is ever really sudden, but undoubtedly the more pronounced symptoms may appear quite suddenly, and are then apt to be mistaken for the results of traumatism; for example, an infant, aged 8 months, was brought to me because he was "not getting on"; there had been some tenderness in the legs for at least four weeks, but the nature of this had not been recognized; one morning the child had his usual bath and then went to sleep for a short time: he awoke with the left eye partly closed by great swelling of the lids, which were discoloured and soon looked as if bruised; it was concluded that during the bathing the child must have struck his eye against the side of the bath. The history, however, of feeding with Allenbury and Benger's foods, the tenderness of the legs, and the presence, as examination showed, of blood in the urine, all pointed to scorbutic haemorrhage, and the rapid disappearance of the swelling and haemorrhage from the lids under antiscorbutic diet confirmed the diagnosis.

It would, I think, be rash to assert that the supposition of traumatism is wrong in all such cases. The haemorrhagic tendency of scurvy may well favour the occurrence of haemorrhage from slight traumatism, which would have no such effect in a healthy infant, and this view is supported by the situation of superficial haemorrhages; for example, in one case the only subcutaneous haemorrhage was over the bony prominence of the sacrum, a part obviously exposed to pressure and jarring; in another which was sent to me as a case of infantile paralysis the efforts of the doctor to obtain a knee-jerk had resulted in a subcutaneous haemorrhage over the patella.

Limb Affection.

Perhaps the most striking feature of infantile scurvy is the tenderness of the limbs and loss of movement. These symptoms occur much more often in the lower limbs than in the upper. In the 54 cases tenderness or swelling was present as follows: In 39 in the legs only, in 10 in legs and arms, in 1 in the arm only. In 4 out of the 54 cases there was no tenderness in any of the limbs. The diagnosis in these cases rested on the association of a scorbutic diet with haematuria, confirmed in two where teeth were present by affection of the gums, and in all by the rapid cessation of haematuria on antiscorbutic diet.

With the tenderness there is sometimes associated some visible or palpable swelling of the affected limb, but in many cases this is so slight that it is easily overlooked; and as it is the result chiefly of subperiosteal haemorrhage it may be detected only by careful—and, I would add, most gentle—palpation of the bone as a vague, deep thickening. The situation of this thickening is of some importance; it is not limited to the epiphysal region like an epiphysitis, but extends some distance along the shaft of the bone—a point which should distinguish it also from any joint affection. In my own cases the swelling has been most often about the lower third of the tibia or the femur, but any part, or the whole, of the shaft may be surrounded by subperiosteal haemorrhage, and occasionally, though much more rarely, similar deep haemorrhage may occur over the flat bones of the skull, producing large bluish swellings on the head which may simulate sarcoma, or over the scapula, as in a specimen in the museum of this hospital.

The loss of movement in the affected limbs is so marked that the disease had been mistaken more than once in my series for infantile paralysis—a mistake which hardly ought to occur, for the acute tenderness, the severe pain on passive movement, the swelling and local thickening, and the associated conditions of the gums and urine are all features quite foreign to infantile paralysis. The loss of movement is no doubt due partly to fear of the acute pain which movement causes; but it may also be due partly to mechanical disability, for not only is the periosteum stripped off the bone by the underlying haemorrhage, but, as I have seen in some autopsies on

infantile scurvy, the muscles also in severe cases are infiltrated with serum and extravasated blood. Occasionally there is further disabement from separation of epiphyses by extensive haemorrhage at the epiphysal line; in one case which I examined epiphyses had been separated in all four limbs. This danger of separation of epiphyses, which is due to effusion of blood at the epiphysal junction, is a point to be remembered in the handling of these infants with scurvy. Cases have been recorded also in which fracture occurred in the shaft of the bone.

Oedema.

The occasional occurrence of oedema over the thickened part of the limbs in scurvy is worthy of notice. In the diagnosis between scurvy and suppurative periostitis it might have been thought that oedema pointed to the presence of pus. This is certainly not so; I have seen well-marked pitting of the skin on gentle pressure in cases of infantile scurvy.

Gums.

The characteristic appearance of the gums has already been mentioned, but it is to be remembered that any great swelling or discoloration of the gums is by no means a constant feature; often it requires careful observation to detect the little affection that exists. There may be only a thin purple line or minute patch of red discoloration at the free edge of the gum; and I have specially noted that in some cases it was only on the posterior edge of the gum behind the tooth, and therefore very easily overlooked.

It is generally stated that where there are no teeth there is no gum affection; this is not strictly accurate, for where the teeth are close up to the surface of the gum but not actually through I have seen haemorrhage in the gum over them. When teeth are present the gums are not necessarily affected; in 7 out of 39 cases in which teeth were present in my series the gums showed nothing abnormal.

Very rarely the gums are markedly affected when there is no other obvious symptom of scurvy. In one of my cases there was no tenderness whatever in the limbs, although the gums showed very marked swelling and purple discoloration. In another some tenderness had been noticed in the legs, but had passed off when I saw the child, whose gums were swollen and purple. In both these cases the existence of scurvy, suggested by the diet and the gums, was proved by the finding of blood in the urine.

Palatal Haemorrhage.

I wish to call attention here particularly to a manifestation of scurvy which has not, I think, attracted much notice, namely, haemorrhage in the mucous membrane of the hard palate. The discoloration occupies the middle part of the vault of the hard palate; it has not in my cases extended forward to the alveolar portion, nor backward to the soft palate. The colour varies from a bright blood red to a deep almost black purple. As in the gums, the affected area may become secondarily inflamed and the mucous membrane over it roughened. Such were the appearances in 4 of my cases, but as I had not looked for this manifestation in the earlier cases I cannot tell in what proportion it is to be found. This palatal haemorrhage is certainly noteworthy, as it may assist diagnosis when it occurs—as in 3 out of my 4 cases—in infants who, owing to absence of teeth, show no gum affection.

Orbital Haemorrhage.

"Black-eye" in an infant 6 to 12 months old, without obvious traumatism, should suggest the possibility of scurvy; but it is not a very frequent symptom. It was present in 6 out of the 54 cases, and in 5 of these was sufficient to cause proptosis of the eye. Curiously enough, in all 6 it was the left eye that was affected. The discoloration appears usually chiefly or only in the upper lid.

Urinary Symptoms.

The diagnostic importance of the urine in infantile scurvy is not sufficiently appreciated. Urinary changes are actually more constant than the gum affection, and an examination of the urine will usually clinch the diagnosis if there be any doubt. During the last few years I have examined the urine of all cases under my notice as far as possible, and out of 34 cases only 4 showed nothing abnormal. In 11 there was blood only, in 8 blood and

casts, in 2 blood and pus, in 1 pus only, and in 7 albumen only; so that the proportion of cases showing some urinary change was 88 per cent., and the proportion showing haematuria was 60 per cent.

But it must not be supposed that blood is to be detected by the naked-eye appearance of the urine in this proportion of cases. In many the amount of blood was so small that it was seen only by careful microscopic examination; and when pus was present, although sufficient to attract attention at once under the microscope, it was not sufficient in any of my cases to be detected by the naked eye, although in one there was enough to make the urine slightly turbid.

It is generally stated that haematuria may be the only symptom of infantile scurvy; and if the haemorrhagic manifestations are to be taken as the only evidence of scurvy, this is certainly so. In 2 of the cases mentioned above there was no affection of the limbs or gums—in fact, no characteristic symptom except haematuria; but in both there had been wasting recently, and one was fretful and miserable, and the other would not take his food; there were in fact—as I suspect there usually are in these cases where haematuria is stated to be the only symptom of scurvy—these vague symptoms of disturbed nutrition which I have described as marking the onset of scurvy. The presence of casts in the urine in association with the blood may indicate a nephritis in some cases. As I have pointed out,¹ the casts are sometimes numerous, and easily found without centrifuging, and the albuminuria occasionally persists for many weeks or months. The occurrence of pyuria with acid or neutral urine, and without any special evidence of irritability of the bladder, would seem to point to a pyelitis, and this was the clinical condition in three of my cases. The recognition of this pyuria is of some practical importance, for, like the nephritis, it is apt to persist after all the acute symptoms of scurvy have passed away. In one case where the tenderness and loss of movement in the legs had begun to improve within forty-eight hours, and were as usual cured in a few days by antiscorbutic diet, the pyuria persisted nearly five weeks, and during this time the child remained pale, and failed to gain weight until the pyelitis began to subside. Not only may the pyelitis retard the infant's recovery, but it seems likely that this condition, which when it occurs as a primary affection in infants causes severe pyrexia, may account in some cases for the high temperature which sometimes accompanies infantile scurvy. In the case in which the urine showed pus without blood the temperature was 104.4°; and in one of the cases in which the pus was associated with blood, a slighter degree of pyrexia persisted for nine days after antiscorbutic treatment began although the gum symptoms and tenderness of limbs had rapidly subsided; the pyuria in this case lasted twelve days.

Temperature.

Pyrexia in infantile scurvy, although rather the exception than the rule, is by no means a rarity; the temperature may be raised to 101° to 102° F. when the infant comes under treatment, but it subsides generally within a few days as the acute symptoms of scurvy disappear. In some cases, however, the temperature rises to 103° to 105° F., and, as I have pointed out, this may depend upon the presence of pyelitis. The occurrence of fever in infantile scurvy is worth remembering, for this symptom, like the oedema to which I have referred, has been regarded as pointing to suppurative periostitis—a very serious mistake if it leads to incision, a measure which would only increase the risks of infantile scurvy. If the possibility of scurvy is borne in mind, and the history of feeding, the gums and the urine are investigated, this error is not likely to arise.

Other Haemorrhages.

Skin.—Haemorrhage into the skin is quite the exception in infantile scurvy; it was present in 6 out of the 54 cases, excluding those in which orbital haemorrhage showed as "black eye"; in two cases it showed as a linear bruise running downwards and outwards for nearly an inch from just below the inner canthus; in one the situation was the scar of vaccination.

Mucous Membranes.—I have already mentioned haemorrhage in the mucosa of the hard palate. The visceral mucous membranes may be similarly affected; in a specimen figured in the museum of the Children's Hospital,

Great Ormond Street, haemorrhage has occurred into the mucous membrane of the bladder. Bleeding from mucous membranes is not very common; in three of my cases slight epistaxis had occurred, and in one of these there was bleeding, also from one ear. Less rare is the passage of streaks of blood with the stools (noted in 8 cases).

Visceral Haemorrhage.

Post-mortem examination showed in one of my cases that extensive haemorrhage may occur in connexion with viscera, though such an occurrence is probably rare. A boy aged 13 months, who had been fed on Savory and Moore's food mixed with condensed milk since the age of 4 months, had much swelling and tenderness in the left thigh and leg, and purple swelling of the gums. He was admitted under my care at King's College Hospital, and at once put upon antiscorbutic diet, but developed diarrhoea and died within a few days. Autopsy showed, in addition to the subperiosteal haemorrhage in the leg, that there were strands of old adhesions over the right lung, and in the meshes thus formed there was a layer of fresh blood clot about one-eighth of an inch thick over a large portion of the lung. Another case in which visceral haemorrhage raised an important practical point was one seen in consultation with Dr. H. J. Spon. The child, aged 8 months, had been fed on the Allenbury foods, and had tender swelling about the lower third of the right femur, and some scorbutic affection of the gums; the infant was extremely ill, and was passing blood and mucus frequently from the bowel; it had vomited twice on the day when I saw it. The abdomen was flaccid and hollow, but in the direction of the transverse colon it showed an elongated sausage-like tumour which on palpation was firm, and seemed to vary in hardness at intervals during palpation. Had there been no scurvy I think no one would have doubted that there was an intussusception. The child continued to vomit, but the bowels were open, and after consultation with my surgical colleague, Mr. F. J. Steward, and an unsuccessful attempt to reduce the tumour by injecting fluid into the bowel, it was decided to postpone operation, as the possibility of the tumour being haemorrhagic was in our minds; moreover, the child's general condition seemed almost desperate. Under antiscorbutic treatment the child improved, and I heard subsequently from Dr. Spon that the abdominal tumour became less, and gradually disappeared after several weeks. I suppose there can be no doubt that in this case there was a mass of blood clot surrounding the bowel, perhaps in the wall of the bowel.

Joints.

As already mentioned, infantile scurvy is sometimes mistaken for some joint affection; careful examination, however, shows that the tenderness and swelling is not connected with the joints, but with the shaft of the bone. Whilst, however, I know of no clinical evidence that arthritis ever forms part of infantile scurvy, haemorrhage into joints certainly may occur; in one of my cases there was found at autopsy a fresh blood clot about the size of a shilling, lying free in the intercondylar part of the knee-joint. In a drawing preserved at the Children's Hospital, Great Ormond Street, a haemorrhage is shown under the synovial membrane of the acetabulum in a case of infantile scurvy.

Prognosis.

There are few diseases in which the effect of treatment is so striking as in infantile scurvy; under efficient antiscorbutic diet the tenderness and pain on movement is usually appreciably less in forty-eight hours, and I would lay it down as a rule that if proper antiscorbutic dieting has produced no definite improvement within four days the diagnosis of scurvy should be questioned.

But although such rapid diminution of the tenderness and pain may be expected, other symptoms persist longer; the subperiosteal haemorrhage if large may cause some deep thickening to remain for two or three weeks. Occasionally the thickening remains for many weeks or months, in spite of the complete disappearance of all other symptoms of scurvy. This is explained by a specimen in the museum of the Children's Hospital, Great Ormond Street, which shows a layer of bone deposited by the separated periosteum and forming a bony sheath enclosing the subperiosteal haemorrhage. I am indebted to Dr. Lees for kind permission to mention this case, which was under his care.

But apart from the mere persistence of symptoms, there are other more serious risks in infantile scurvy. I have several times heard medical men make light of the danger of scurvy from patent foods on the ground that not only is scurvy an uncommon result but that when it occurs it is speedily cured by simple changes in the diet. No doubt the risks of scurvy would be minimized if the disease were detected at its earliest onset; as a matter of fact, it is hardly ever recognized until the infant has already passed through weeks of needless and intense suffering. This, surely, is serious enough; but there is also a very real risk to life; in 3 out of the 54 cases the disease proved fatal by diarrhoea and exhaustion, and even when death does not result there is sometimes prolonged disturbance of nutrition so that the infant fails to gain weight and remains ill-nourished and frail for weeks or months after all other symptoms of scurvy have disappeared. The renal symptoms may influence the prognosis—in one of my cases of nephritis with scurvy the child showed a trace of albumen in the urine, and was frail in appearance two years after the occurrence of scurvy—and pyelitis may persist for several weeks and nutrition may suffer therewith. The mere presence, however, of blood in the urine does not seem to be of serious import; it usually ceases in a week or ten days under antiscorbutic treatment.

Photograph of femur, showing at upper part periosteal ossification, enclosing subperiosteal haemorrhage.

Prevention and Treatment.

Scurvy is an entirely preventable disease, and it is important, therefore, to know the foods upon which it is liable to arise. In the subjoined table are stated the kinds of food in use for several weeks or months before the appearance of scurvy in this series of cases:

Patent foods containing dried milk, prepared according to directions, with water only	14 cases.
Patent foods prepared with fresh milk	18 "
" " condensed milk	11 "
" " sterilized milk...	1 case.
" " fresh (unboiled) milk	1 "
Condensed milk diluted with plain water, barley water, or lime water	5 cases.
Sterilized milk	3 "
Peptonized milk	1 case.
Boiled milk	1 "

In the above table where "fresh milk" was used in mixing the food, it was usually, if not always, heated to boiling during the process.

It is clear that the mere fact that cow's milk is mixed with a patent food is not sufficient to prevent scurvy. Nor can it be shown that the occurrence of scurvy when fresh milk is thus used is due to the degree of dilution rather than to the patent food, for in some of my cases the milk had been used for many weeks in the proportion of not less than two parts of milk to one part of water, and in others with equal parts of milk and water. The extreme rarity of scurvy from plain boiled milk when used even more diluted than this seems to prove also that the scurvy in these cases is not due to the boiling of the milk but to the addition of the patent food. In one of my cases in which scurvy resulted from the use of diluted milk with Mellin's food the milk had been heated only to about 167° F. for some months, and had been used fresh without even pasteurization for three weeks before the onset of scurvy (the milk was examined chemically and bacteriologically, and certainly had not been sterilized, and there seemed no ground for disbelieving the statement that it had not been heated, except to the usual temperature of the feeds). The antiscorbutic power of fresh unboiled milk is evidently slight, and is diminished or destroyed by heating, but from the rarity of scurvy on boiled milk compared with the fact that boiled milk is probably the commonest of all infant foods, it would seem that milk has but little tendency to produce scurvy if merely heated to the boiling point for a few seconds, as is commonly done.

In order to prevent the occurrence of scurvy in an infant fed upon some scurvy-producing food, it is advised to add some antiscorbutic food to the diet, and for this purpose raw meat juice, orange or grape juice are commonly used. It might seem hardly necessary to point out that the meat juice must be from fresh meat, and probably from uncooked meat; but I have found in at least three instances that some patent meat juice, or one of the concentrated meat preparations which are so widely advertised, was being given with the idea that it would prevent or cure scurvy. As far as I have seen, such preparations are entirely worthless as antiscorbutics. The red gravy from cooked meat was shown to be ineffectual in one of my cases where the child was having one table-spoonful of this red gravy daily as an addition to its diet of Allenbury food No. 2, with one feed of baked flour with water and a little cream. In another case occasional feeds of beef tea had not prevented the onset of scurvy. Even raw-meat juice, unless given sufficiently freely and regularly, may not prevent scurvy. In one of the most severe cases raw-meat juice had been given for five months before the disease began; and in the only case in my series which arose on peptonized milk the infant was having a teaspoonful of raw-meat juice three times a day on every alternate day, then, owing to some looseness of bowels, it was omitted fourteen days, by the end of which time the infant showed well-marked scurvy.

Fruit juice, though probably a more powerful antiscorbutic than raw-meat juice, is not an unconditional safeguard. In the case already mentioned where red gravy of cooked meat was being given with the Allenbury food, the juice of two black grapes had also been given daily for four and a half months before the scurvy began; in another case where scurvy had been recognized the juice of four grapes had been given daily for fourteen days with no apparent improvement (on potato there was definite improvement in two days).

No doubt this failure of fruit juice depends upon insufficiency of dose; but it may be that this insufficiency is relative, not absolute—that it requires a larger amount of fruit juice to prevent the scorbutic tendency of some foods than of others. The superior potency of potato as an antiscorbutic seems to me quite certain, but as I have almost always given also raw-meat juice or orange juice my own results do not illustrate this point, and some of them have shown very rapid improvement on orange juice alone. While, however, potato seems specially valuable in the treatment of scurvy, it is less suitable for prophylaxis, for although it is usually tolerated well for a few weeks during treatment it is more apt to cause digestive disturbance than is fruit juice, and is therefore less suitable for prolonged use as a regular addition to the diet.

The potato is prepared by boiling or steaming in the ordinary way with care to obtain a floury potato; the outer floury portion is then scraped off and beaten up thoroughly with enough milk to make a smooth cream, sufficiently thick to pour out of a jug rather heavily—potato 2 heaped teaspoonfuls (with average 2-drachm teaspoon) to 1 oz. of milk; $1\frac{1}{2}$ to 2 teaspoonfuls of this potato-cream are given three or four times daily; after two or three weeks the dose of this should be gradually reduced, and omitted altogether within four weeks from the commencement of treatment. The mode of giving the potato-cream is of some importance; most infants take it best mixed up with the ordinary feed, but if this is done it should be mixed with a portion only of the feed to ensure the whole of the potato-cream being taken, otherwise if part of the food is left the child does not get the full dose of potato. Occasionally it is taken more readily given separately; in either case it is often disliked at first, but I have rarely had any serious difficulty in getting it taken. In addition, 2 teaspoonfuls of raw-meat juice may be given three or four times in the twenty-four hours, and sometimes $\frac{1}{2}$ teaspoonful of orange juice two or three times a day, but any looseness of the bowels should make us cautious in adding this to the potato-cream, for diarrhoea in infantile scurvy is a serious complication, as Glisson seems to have observed. At the same time, the child is placed on a diet of milk which has been heated just short of boiling point and diluted with water; the scurvy-producing "food" is of course stopped.

The necessity for moving and handling the infant as

little as possible is obvious; the pain caused by pulling on socks, shoes, and tight sleeves, and by bathing is so evident and so piteous to behold that it is only humane to have the infant wrapped in loose clothing and let him lie undisturbed as much as possible; the duration of the tenderness and pain is only a few days after proper treatment is begun, so that all ordinary dressing and bathing and outing may well be in abeyance until all tenderness and pain have gone.

REFERENCE.
¹ *Lancet*, August 13th, 1904.

ERRORS OF REFRACTION AMONG CHILDREN ATTENDING ELEMENTARY SCHOOLS IN LONDON.

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WHILE holding the position of oculist to the elementary schools under the London County Council I took the opportunity during nine months of last year (1905) to gather statistics as to the relative frequency of the different forms of refractive error among the children. In the "provided" schools I saw all those children—except those who happened to be absent at the time of my visit—who had been previously picked out by the teachers as having failed to pass the somewhat rough test which has been in force in order to detect those whose vision is seriously impaired. This test consists—for the upper standards (V, VI, and VII) in reading $\frac{1}{2}$ accurately with both eyes open; for the lower standards (I to IV) in reading $\frac{1}{8}$. It is found that by means of this test, on an average about 10 per cent. of the total number of children in either the boys' or girls' department of any elementary school are picked out as having defective vision.¹ When children already wear glasses the test is made with the glasses on, so that a certain number of children with a considerable refractive error are not included in my statistics. Although, however, in a few schools in comparatively well-to-do neighbourhoods the number of children wearing glasses—more especially myopes in the upper standards—was already considerable, yet the actual proportion with glasses in the whole number of schools that I visited in the two School Board divisions of East Lambeth and Chelsea was very small. In this way 31 boys', 33 girls', and 2 mixed departments, with a total register of 24,095, were dealt with, of whom 1,732—778 boys and 954 girls—of those who were brought under my special notice by the teachers, all between the ages of 7 and 14, were classified by me into the four categories that appear in my tables.

My method was to apply the shadow test to each of the children seen. Usually I was able to take them into the teacher's room, and with the gas lit and blinds drawn, while the child was directed to look at some distant object, to reflect sufficient light into each eye from a plane mirror to at once determine in which direction the shadow moved. If "against" in both meridians I put down the eye as myopic; if the shadow moved "with" in one or both meridians, by employing a trial frame with four convex glasses (+1, +2, +3, +4) I was able quickly to determine to which of the remaining three categories the eye belonged, and to classify the children that I saw according as I found them to have:

1. Hypermetropia of 2 D or more with or without astigmatism not more than 1 D.
2. Hypermetropic astigmatism of more than 1 D.
3. Mixed astigmatism of more than 1 D.
4. Myopia (including myopic astigmatism) if 1 D or more.

A certain number, namely those with H less than +2, with astigmatism not more than 1 D, with apparent myopia below -1, or with emmetropia, I did not classify at all. I also left out those with serious corneal lesions due to interstitial keratitis or phlyctenular disease, in fact all in whom I judged disease and not refractive error to be the main cause of bad vision. On the other hand, cases of slight corneal nebulæ, if these appeared to me to be less important than the refractive error, I included. In all those cases where the refraction of the two eyes was different, I classified the child according to his *better* eye; and in this connexion I considered simple hypermetropia as better than hypermetropic astigmatism, this, again, better than mixed, and mixed astigmatism better