A British Medical Association Lecture ON THE WASTING INFANT.*

BY
ROBERT HUTCHISON, M.D., F.R.C.P.,
PHYSICIAN TO THE LONDON HOSPITAL AND TO THE HOSPITAL FOR
SICK CHILDREN.

THE terms malnutrition, dystrophy, atrophy, athrepsia, "food disorders of infancy," and marasmus, are used more or less indiscriminately in textbooks to designate that condition which is familiar to all of you under the simple name of "wasting." There is no need to describe in detail the clinical characteristics of the wasting infant; they are, unfortunately, only too familiar, although I think one may truthfully say for our comfort that the severe degrees of wasting are not so often met with as they used to be. None the less, the condition in greater or less degree is still common enough and is a source of great trouble and anxiety in practice.

CAUSES OF WASTING.

In order to have any success in the treatment of wasting it is important to realize that it may have many different causes, which may be classified as follows:

1. Organic Disease.

Any organic disease, of course, is apt to be attended by wasting as one of its consequences, but we are now concerned only with those cases in which the wasting is what is sometimes called the "presenting symptom," and the signs of organic disease are inconspicuous or even altogether absent.

Naturally one thinks first of that great wasting disease of childhood—tuberculosis. It must be remembered, however, that tuberculosis is not, as a matter of fact, common during the first six months of infancy, when wasting usually occurs. Nor is it likely to occur without producing some evidence of its presence in the shape of physical signs in the chest, abdomen, or glands, although occasionally, one must admit, it is found after death when its presence had not been suspected during life. In these cases, however, one may be dealing with a secondary infection. Tuberculosis is most to be suspected when there are no signs of digestive derangement and when the rectal temperature is persistently raised.

Congenital syphilis has also to be thought of, but here again I would remind you that, compared with the number of cases of wasting you will meet with, congenital syphilis is a rarity. Apart from the more florid cases of the disease in which wasting will not be the presenting symptom, it must be admitted that a syphilitic "taint" may interfere with nutrition, and it is possible that some of the wasting infants who improve so remarkably under the administration of grey powder, as some of them undoubtedly do, may be affected in this way. If in doubt a blood test should settle the matter.

Diseases of the lungs, such as chronic bronchopneumonia and "latent" empyema, may be unsuspected causes. The former is often curiously unobtrusive in young infants and may be entirely afebrile, so that its presence is only revealed by a few moist sounds in the lungs of doubtful significance. On the other hand, careful percussion ought to detect an empyema, provided it has been looked for.

The only remaining organic cause to be borne in mind is congenital disease of the heart. Here again there will usually be signs on auscultation, but it must be remembered that every now and then congenital disease of the heart occurs without a murmur, and such cases are apt to be overlooked, although they may, in some obscure fashion, interfere seriously with nutrition.

2. Starvation.

It is apt to be forgotten that wasting may be due to nothing more recondite than mere insufficient intake of food. This may be the result of pure *underfeeding*, either

from ignorance or carelessness, or, sometimes, with the best intentions on the part of the mother. Thus I once saw twins who had been fed for some weeks on barley water *only*, and yet, curiously enough, they were not as wasted as might have been expected. A common cause is the use of condensed milk in too high a degree of dilution or the giving of too small feeds.

Inability to suck from some mechanical difficulty, such as hare-lip, facial paralysis, nose-blocking, or mere weakness, may obviously and naturally lead to insufficient nutrition. The same result ensues if sucking is painful, as in some cases of stomatitis, or if the hole in the teat of the bottle is too small.

A child, for instance, was once brought to me a month old and weighing only 6 lb. 5 oz. At first I could find no cause for the wasting. A slight modification of the feeding was made, but at the end of a week the weight was an ounce down. Still I could find no cause. Next week another two ounces were lost. I then looked at the teat and found that the hole in it was very small. A new teat was ordered, and in a fortnight the weight had gone up 1 lb. 8 oz.

3. Prematurity.

Premature infants are very apt to become the subjects of wasting. There are probably several reasons for this. In the first place, the child is often too weak to suck properly, so that it gets an insufficient quantity of food. In the second place, I think it is probable that the premature infant is, metabolically speaking, an extravagant machine and requires a large amount of fuel to maintain its body-temperature, which leaves no surplus for the purposes of growth. In the third place, such infants have often but a feeble power of digestion and possibly also of assimilation. It is not to be wondered at, therefore, that with all these difficulties to contend with they often waste.

4. Improper Feeding.

This is such a common cause of wasting that some people are apt to speak of wasting as if it were synonymous with the "food disorders of infancy." This, however, as we have already seen, is by no means always the case. None the less, unsuitable feeding is undoubtedly the starting point of the majority of cases of wasting.

The error may be a *quantitative* one—as in the case of giving too small or too weak feeds, which we have already considered. Paradoxically enough, however, too large or too strong feeds may have a similar result, and a child will therefore sometimes start to gain weight when the amount or strength of the feeds is reduced. Far oftener, however, the error is a *qualitative* one and consists in giving an ill-balanced mixture which is too rich in one of the food constituents, a relative excess of fat being the commonest fault. It should be noted that the want of "balance" in the diet need not be an absolute one, but only relative to the digestive capacity of the particular infant. Some babies, for example, appear to have a congenital inability to deal with a normal proportion of fat in the diet, especially the fat of cow's milk, and in them a fat dyspepsia is easily set up. In other and less common cases there is a similar disability as regards casein or sugar.

Although a lack of balance in the diet may result merely in a failure to gain weight (the so-called "disturbance of equilibrium"), it is very apt, if persisted in, to lead to the next great cause of wasting—

5. Dyspepsia.

This is so common, either as a cause or as a concomitant of wasting, that there are few marasmic infants who are not also dyspeptic.

The symptoms of dyspepsia may arise suddenly, as in cases of summer diarrhoea, and become persistent, leading eventually to chronic wasting; or, more frequently, the dyspepsia comes on gradually as the result of the continued use of an unsuitable or ill-balanced diet. Finkelstein, Czerny and Keller, and others have attempted to make a classification of the different varieties of dyspepsia according to the food constituent responsible for the digestive derangement, and Finkelstein has described their culmination in a condition to which he applies the ill-omened term

* Delivered before the Sheffield Division on February 8th, 1924.

"decomposition"—which appears to be very much marasmus as we usually know it—and finally in a state of "intoxication," the symptoms of which appear to be indistinguishable from those of a severe case of infective diarrhoea. His views have obtained a considerable amount of support both here and in America, but, although they are undoubtedly suggestive, I must confess for my own part that I am unable to reconcile them satisfactorily with the actual conditions seen at the bedside, and they appear to me to be an attempt to effect a rather arbitrary simplification of a very complicated subject. Be that as it may, there is no doubt that some guide to the nature of the feeding error can be derived from a study of the stools. Excess of fat in the diet is the commonest starting point of a dyspepsia, and is indicated by constipated, pale, crumbly or soapy stools which do not adhere to the napkin. Excess of casein—contrary to what was long supposed—is much less likely to be a source of difficulty, and when it occurs leads to vomiting of curd and the passage of tough white particles in the stools, which are insoluble in a mixture of alcohol and ether. Sugar is the most easily digested of all the food constituents and least likely to lead to trouble, but if present in the food in an amount which is beyond the infant's digestive capacity the stools are apt to be loose, watery, and acid, and their passage attended by much flatulence. It is rare, however, to meet with pure examples of any of these different forms of dyspepsia. Usually the digestion of all the food constituents is impaired, and in severe cases it always is so.

The matter is further complicated by the fact that a secondary bowel infection may be grafted upon a simple dyspepsia and the green slimy stools of a true enteritis make their appearance. It must be remembered, too, that wasting of itself impairs digestive capacity, so that a case of marasmus which originated in, say, underfeeding may easily pass into a state of dyspepsia which accelerates the downward progress.

6. Primary or Idiopathic Wasting.

It is to primary or idiopathic wasting—that is to say, wasting of unknown cause—that the term "atrophy" should be specially reserved. In cases of this sort there does not seem to be anything wrong with the food and there are no signs of dyspepsia, but the child simply wastes, and after death no cause can be found for the failure of nutrition. Such cases are commonest in very young babies, and often the patient is the youngest child of a large family or is a twin or illegitimate. We appear to be dealing here with a true failure of assimilation, but why it fails is quite unknown. I need hardly say that some "endocrine" disturbance has not failed to be invoked as an explanation, but such a hypothesis is purely speculative. It is best to admit frankly that the true nature of such cases has so far baffled us.

COMPLICATIONS.

However wasting may have come about it is apt to become attended by complications of one sort or another. Thus such babies are—understandably enough—very prone to infections by pyogenic organisms which cause boils and abscesses, by the tubercle bacillus, and by the *B. coli*. They are also very apt to contract an infective enteritis or a bronchopneumonia. The clinical picture may further become blurred by the supervention of symptoms of acidosis or nephritis, or by the appearance of an "idiopathic" oedema on the one hand, or of the signs of "dehydration" on the other. It will thus be readily understood how protean the signs and symptoms exhibited by a wasting infant may be and how they may vary greatly almost from day to day.

PROGNOSIS.

The prognosis in a case of wasting is always doubtful. Even when things seem to be going well at the moment there is always the possibility of an abrupt and apparently inexplicable collapse which proves fatal. On the other hand, a sudden and seemingly spontaneous improvement may occur at any moment even in the worst cases. More frequently, in the cases which ultimately do well, one finds a very gradual improvement chequered by many relapses. Ground is often gained more rapidly after teeth are cut. In fatal

cases death is usually the result either of gradual inanition or of the supervention of an infection, a sudden food "intoxication," or of an attack of diarrhoea. In all cases the outlook is decidedly more rosy in private than in hospital practice. It is also better, curiously enough, in the cases in which there seems to be most wrong. If there is dyspepsia it may be possible to correct it, but the worst cases are those in which everything seems to be right except the power of gaining weight.

TREATMENT.

The treatment of a case of wasting demands the highest degree of patience, optimism, and resource on the part of both doctor and nurse. At the outset it must be realized that the treatment is not merely dietetic—the general hygiene of the infant is of the first importance. Fresh air, sunshine (if obtainable), cleanliness, and warmth (especially keeping the feet and legs warm) are all great aids to success.

Nothing, probably, makes more difference than good "mothering." To look after a wasting infant is one woman's job, and he should not be left to lie alone in his cot, but should spend a good deal of his time in the mother's or nurse's arms; he wants plenty of "cuddling" and amusing. Great care should be taken that he does not get chilled when being washed. The impossibility of providing all these desiderata in institutions is one of the main reasons, I believe, why these cases often do better even in a poor home. As to the all-important question of feeding, it is only possible to indicate some general principles.

You will do wisely, in the first place, to go slowly and play for safety, remembering that overfeeding is more perilous than underfeeding, and that one must not be too ambitious in the matter of increasing the weight. I believe that many cases of wasting are really brought about by the exaggerated importance attached to mere weight as an index of a baby's progress which is now so prevalent. Monthly nurses are frequent offenders in this matter. The nurse is so anxious to get the baby "on" and to have a good result to show at the weekly (or daily!) weighing that she tends to increase the strength of the feeds to a point which is beyond the digestive capacity of the infant, with the consequence that though all goes well at first a dyspepsia is soon set up which rapidly leads to wasting. My next advice is always to change the feeding reluctantly, cautiously, and never without good reason, bearing in mind that fat is the ingredient most likely to cause trouble. Before starting a new food it is well to clear out the bowels with a small dose of castor oil, and if acute symptoms of "intoxication" have supervened to suspend all food (except glucose and water) for a few hours, but not for long, remembering that wasted infants stand starvation badly. If the food has been too rich in sugar do not reduce the proportion of the latter too rapidly or collapse may ensue.

Breast milk is the best food in most cases, but is unfortunately often unobtainable. In selecting an artificial food begin with one which is rather poor in fat and relatively rich in carbohydrate and whose protein is in a digestible form. Sweetened condensed milk (2 drachms to 3 ounces) or a half-cream dried milk (1 drachm to 1 ounce) with the addition of dextri-maltose fulfils these conditions. Some indications for feeding will also be furnished by the stools. If they point to severe fat indigestion it may be necessary to feed on a mixture of whey and Mellin's food (dextri-maltose), or whey with the addition of a modified starchy food; if casein is the difficulty the milk should be fully peptonized or citrated; or a dried milk may meet the case.

A useful formula is as follows: Take 2 flat dessertspoonfuls of Benger's or Savory and Moore's food and mix in a basin with 5 oz. of milk. Bring 15 oz. of whey (prepared with rennet) to the boil in a double saucepan and pour slowly on to the cold mixture, stirring meanwhile. Allow to stand covered up for thirty minutes, then stir and bring slowly to the boil; strain through a linen handkerchief, and the food is ready for use. Such a mixture is poor in fat and casein, but relatively rich in lactalbumin and carbohydrate.

In those—fortunately less common—cases in which there is sugar indigestion and "intoxication" so-called "protein milk" is often very useful. It is most easily prepared by the use of casein.*

* Supplied (with directions) by Mead, Johnson and Co.

As to the size or intervals of the feeds you must feel your way in each case and not be bound by too rigid rules, but the more wasted and exhausted the child is the smaller and more frequent must the feeds be.

Drugs are of little use in cases of wasting except to meet special indications such as flatulence, colic, etc. I have already pointed out, however, that grey powder sometimes does good even in cases in which there is no reason to suspect a syphilitic taint—possibly by correcting constipation or by stimulating the digestive secretions—and alcohol is also helpful, especially if there is much exhaustion with a subnormal temperature. It should not be added to the feeds, but given as a medicine and not too dilute. The administration of thyroid, which is sometimes advocated, appears to me to be irrational.

It is a common custom to anoint wasting babies with cod-liver oil. This is a dirty practice, the utility of which is in the highest degree doubtful. It is inconceivable that the child can obtain any appreciable amount of nutriment by such a method, and at the most it can only help to a slight degree in the conservation of body heat. Almond oil would do this as effectually and much less offensively than cod-liver oil, while adequate clothing is far more effective than either.

When all is said and done, you will lose a good many of your marasmic babies in spite of all your care and skill. Do not be cast down over it; the fault is not yours. It is a delusion to suppose that there is for every one of these cases some magic plan of feeding which, if one could only hit upon it, would immediately change the whole situation. On the contrary, the fault in many, perhaps most, cases is in the child and not in the food, and once at least that the disorder has progressed to a certain length no treatment makes any dramatic difference; all one can do is to keep patiently "pegging away," but remembering always the first rule of therapeutics—not to do harm.

THE MECHANICAL EFFECTS OF ENLARGEMENT OF THE PULMONARY GLANDS.*

BY

NEWMAN NEILD, M.B., M.R.C.P.,

PHYSICIAN, BRISTOL GENERAL HOSPITAL; LECTURER IN PHARMACOLOGY AND THERAPEUTICS, UNIVERSITY OF BRISTOL.

THIS subject is one in which I have been particularly interested during the last ten years, and whilst I make no claim for discovery or priority I do claim that my statements are largely based upon my own observations and deductions.

The *anatomical position* of the lymphatic glands is all-important in the consideration of the mechanical effects of their enlargement. The lymphatic glands are said not to extend into the lung beyond the fourth branching of the air tubes, or where the tube is less than 1 mm. in diameter. A section through a bronchiole at a level where there is lymphoid tissue shows the bronchiole accompanied by arteries, veins, lymphatics, and nerves.

The various systems of tubes lie with glands in close relationship, and are surrounded by alveolar lung tissue. These glands are anchored by the lymphatics which anastomose round about the veins and arteries, and any enlargement of the glands must of necessity press upon these systems of tubes. In part, at any rate, the result of such pressure will depend upon the power of resistance of the tubes. The resistance will depend in each case upon the resistance of the wall of the tube and the distending pressure within the tube. The tube with the greatest resisting power of the wall will be the bronchiole, strengthened as it is by the scales of cartilage. Where there is a gland there will also be found at least one scale of cartilage ready to protect the wall of the bronchiole against the pressure which would arise should the gland enlarge.

The resistance of the wall and the pressure of the blood within the vessels is greater in the arteries than in the veins. The veins will therefore be compressed earlier and to a greater extent, at first, than the arteries—given that the advantage of position of the gland in the case of artery and vein is equal. Last in order of effective resistance come the lymphatics, for the exceedingly thin walls and the low pressure of the contained lymph offer the least resistance of any of these systems of vessels. Moreover, upon the lymphatics the obstructive effects of the enlargement of the lymph glands is caused in two other ways beside that of the direct pressure upon them by the enlargement of the gland as a whole. When there is active congestion of the gland there is obstruction to the passage of lymph, and also the lymphatics may be nipped in the hilus of the glands and where the lymphatics pass obliquely through the capsule. Enlargement of the glands will therefore affect these tubular systems in the following order: lymphatics, veins, arteries, and, lastly and least, the bronchioles.

What part does the enlargement of the glands play in various pulmonary diseases? Let me take first *pneumonia*. In this condition the enlargement of the glands is often enormous. The glands in the hilus of the lung are in a position of great mechanical advantage from the point of view of pressing upon the large blood vessels as they leave or enter the lung, and there can be no doubt that at first there is a considerable amount of back pressure in the veins which drain the parts first affected, resulting in the addition of passive congestion to the active congestion already begun.

The back pressure in the lymphatics and veins assists the outpouring of the fibrinous exudation from the actively inflamed part. This passive congestion assists in causing the haemorrhage that occurs at the same time. The passive congestion where only a portion of a lobe is pneumonic would be greater than it is were it not for the freedom of anastomoses in the veins—a freedom that is in sharp contrast with the absence of anastomoses in the arteries of the lung. Not only is there enlargement of the glands round the medium-sized bronchioles leading from the pneumonic area, but also of those in the hilus, and this explains the alteration in percussion note of the non-pneumonic areas of the affected lung.

What is the advantage of this pressure upon the veins? Many chains of lymphatic glands in the body lie against veins and cause a similar pressure. In the leg a passive hyperaemia produced by the glands responding to a distal septic focus that has no external outlet would go some way towards producing the line of treatment associated with the name of Bier. If there is any advantage in Bier's treatment, then that advantage is obtained for the affected lung, with the difference that the hyperaemia is continuous. Should a septic focus in a leg be open, then this obstruction will help to maintain a toxin-containing discharge, toxin that otherwise would have circulated round the body. Should the wound be sealed by drying, the general toxæmia becomes obvious. The lungs, however, are kept moist but the alveoli are soon plugged by fibrin, which prevents a further outpouring. In bronchitis the outpourings are expectorated more or less freely; in pneumonia the expectoration is not large in amount as a rule.

May the enlargement of the glands play a part in the causation of any complications or sequelae of pneumonia? Dr. Percy Kidd says that out of 128 fatal cases of pneumonia oedema was found *post mortem* either in the lobe nearest to the one consolidated or in the opposite lung. How much of this oedema is due to heart failure—the usual explanation given—is difficult to estimate, but in the case of oedema of a lobe adjacent to the affected lobe back pressure from gland enlargement is certainly a factor to be reckoned with. Had heart failure alone been the cause, then the other lung would also be oedematous. Again to quote Dr. Percy Kidd, "collapse of a whole lobe, although by no means rare, is far less often observed than the oedema; it has a similar distribution."

Whatever other factors there may be in the causation of collapse of a lobe, the enlargement of the glands plays a large part. Should there be, early in a case of pneumonia, a temporary occlusion of the bronchiole aerating the

* Abridged from a Presidential Address to the Bath and Bristol Branch of the British Medical Association, 1923.