was normal, and there were no enlarged glands. I opened it; the mucous membrane was normal, and bacteriological examination of the bile showed it to be sterile. The stomach and duodenum were normal. The appendix was enlarged to the size of my index finger, and was adherent at its base. On cutting into it its walls were thickened; it was strictured at its base and contained muco-pus.

This is typical of the first group, and the diagnosis can only be made certainly if there is definite tenderness in the right iliac fossa during an attack. To show how necessary general exploration may be I will give you another example.

In 1919 I was asked to see a lady with a view to removing her appendix for dyspepsia. The following was the history given me: a year previously she had waked in the middle of the night with abdominal pain, diagnosed as acute appendicitis. She was kept in bed fourteen days. Since then she had had much nausea and occasional abdominal discomfort, both waking her and keeping her awake, and three months previously a milder attack of nocturnal pain. The report after a barium meal was that she had viscerotaxis. Acting on the rule I have laid down, that although you may diagnose appendix dyspepsia you should never act on it—that is, never be content with removing the appendix alone—I found an atrophied adherent appendix, which I removed. On exploring the upper abdomen the cause for her abdominal pain became evident—there was cholecystitis with gall stones.

In chronic appendicitis causing dyspepsia the pain is not right-sided, although during an attack there may be tenderness in the right iliac fossa, and pressure here causes epigastric discomfort. But I wish to press home the fact that in these cases thorough examination of the whole abdomen is necessary at operation; only then can a certain diagnosis of appendix dyspepsia be made.

The object of choosing this subject has been to draw attention to the necessity of early operation in cases of acute appendicitis and to the care necessary in their diagnosis; and secondly, to warn you against too readily diagnosing chronic appendicitis—it is a disease that does exist and cause symptoms, but is not nearly as common as is often supposed.

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AN ANOXAEMIC TYPE OF INFANTILE ATROPHY.

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THE term "atrophy" is used to describe a condition in infants which is characterized by an obstinate refusal to gain weight and thrive, in spite of dietetic or medicinal treatment, and in whom no cause can be found to account for this failure to grow. Atrophy is seldom associated with dyspepsia. If this is present at all it is very slight, quite insufficient to account for the infant's lack of progress, and quite possibly a result, rather than a cause, of the condition. In a few cases, however, there may be a history of previous dyspepsia, following which the atrophic state becomes evident; such cases may be classified as dyspeptic atrophies and may or may not respond to dietetic treatment. Again, atrophy may be due to endocrine or vitamin deficiency, and this type responds to the appropriate treatment. There is still, however, a form of atrophy which fails to respond to dietetic, endocrine, or vitamin treatment, and it is with cases of this kind that this preliminary note is concerned.

It is a common experience that at necropsies on cases of infantile atrophy little or no organic change is revealed which throws any light on the causation of the disease. This in itself might suggest that some physiological factor, other

than those mentioned above, is at fault. The idea that these particular cases are due to the disorder of some fundamental physiological function led to the consideration of the possibility of anoxaemia, either general, due to some deficiency in the functioning of the lungs, or local, due possibly to capillary stasis in the intestine as a result of initial digestive disturbances of either a mild or severe type. Regarding the former, it is well known that failure to saturate the blood in the lungs with oxygen, or deficient saturation brought about by other means, can profoundly affect the physiological condition of an animal. The subject has been fully dealt with by Haldane¹ and need not be elaborated here. That cyanosis and anoxaemia may occur in premature infants has been recorded by Bakwin,² who has described the results of treatment of two such cases with oxygen. About the occurrence of local want of oxygen in a tissue or organ, however, we have few or no data. It is true that this must play a part in certain well recognized pathological states—for example, in the liver and kidney in cases of mitral stenosis and in other conditions of circulatory failure, either local or general. Whether simple capillary stasis can occur in a single organ due to localized abnormal conditions and thus be a cause of local anoxaemia is, however, not so certain. It may be remembered that surgical shock is generally believed to be associated with widespread capillary stasis. The phenomena of shock can also be closely simulated in animals under anaesthesia by injection of histamine, which is a product capable of production by tissue breakdown. It does not appear inconceivable therefore that, under certain conditions, dysfunction of a tissue may be brought about locally through the action of toxic substances produced by bacterial action and that this may have, as a concomitant, local anoxaemia due to failure of the capillary circulation.

These were the underlying ideas that prompted us to try the effect of oxygen administration in certain cases of infantile atrophy. The cases are not as a rule cyanosed, but a slight degree of this condition may sometimes be observed. It seemed that a direct answer to the question whether a generalized anoxaemia was present would be best obtained by determining the results of oxygen therapy. On the other hand, oxygen therapy in localized anoxaemia due to capillary stasis would only act by increasing the dissolved oxygen of the blood, and this might be expected to yield good results only if continued for a considerable length of time and if the local anoxaemia were not severe.

The results we have obtained have so far been promising, and although the work is by no means completed it seems worth while placing them on record at this stage of the inquiry, which promises to be a long one. The preliminary experiments were carried out by two of us at the Leeds General Infirmary, through the kindness of Dr. C. W. Vining, but the greater part of the work has been done at the Manchester Babies' Hospital. For the facilities accorded to us there we are very greatly indebted to Dr. Catherine Chisholm. The cases chosen for oxygen treatment have been those which, after a period of dietetic treatment, have shown themselves to be refractory, as judged by failure to gain weight. In these cases digestive disturbances are never a marked feature, although they may occur, and are then of a mild type. When failure to gain in weight was established and the child was still on a diet to which it had not responded, the oxygen treatment was commenced. Thereafter the composition of the diet was not changed, but increased amounts of food were given as necessitated by the increase in weight and appetite of the child. The oxygen treatment was carried out in the small closed cot which was exhibited by us to the Physiological Society two years ago.

It consists essentially of an air-tight box (29 in. by 18 in. by 20 in.) with a glass lid and apertures for admitting oxygen at a known rate and for sampling the air within it. The infant is placed inside on a hammock. A small fan inside serves for mixing the contained air and providing for its movement. No arrangements are made for removing the carbon dioxide which accumulates while the infant is in the cot. In a two-hour period the carbon dioxide content of the air rises to between 2 and 3 per cent., depending on the size of the child and the amount of movement it makes. The oxygen is admitted from a cylinder provided with reducing valve and pressure gauge, and passes through a modified Pitot's tube, whereby, after a preliminary standardization, the rate of flow can be determined. After placing the child in the cot, the

glass lid is clamped on, the fan started, and oxygen rapidly admitted in sufficient amount to produce an atmosphere in the cot containing about 45 per cent. of oxygen. When this is attained (in four to six minutes) the flow is diminished to that amount which is necessary to maintain the oxygen concentration at about the same level. The infant is kept in the cot for two to two and a half hours and then removed for feeding, etc. It is then replaced in the cot, the air again enriched with oxygen, and so on for a further period of two to two and a half hours.

This treatment has been carried out during the day only. At the commencement the period of treatment with oxygen is limited to two hours on the first day, four on the second, and is increased to a maximum of about eight hours by the fourth day. The length of time during which it has been carried out has varied between two and three weeks. Except for one case which died within two days, and in which treatment was only instituted when the child was almost moribund and had lost weight rapidly, most of the infants treated have responded with a notable gain in weight, which has been continued after the treatment was stopped. Since the treatment has been continued over a period of two or three weeks it was necessary to rule out the factor of coincidence, because atrophic infants may occasionally begin to gain weight for no apparent reason. We have now treated 16 cases, of which 13 have shown a satisfactory gain in weight within a week from the beginning of the oxygen administration, and in our opinion this cannot be accounted for as a mere coincidence, but is due to the treatment.

It is possible that the mere increase of carbon dioxide in the air in the cot might be responsible for the good results obtained. A number of cases have therefore been treated in a similar cot in which the oxygen content was kept at the usual atmospheric level, but the carbon dioxide was allowed to accumulate. Of 5 cases thus treated, 4 began to gain weight slightly, whereas 1 remained stationary. The gain in weight was not so marked as that obtained in other cases treated in the oxygen-enriched air. All these 5 cases subsequently gained weight, but at a much more rapid rate when treatment with oxygen-enriched air was instituted. The following brief data with weight charts illustrate the kind of result obtained in successful cases.

CASE I.

A male infant was admitted to hospital at the age of 2 months with a history of loss of weight and some dyspepsia: he was pale and thin but not wasted, and nothing abnormal was detected on examination. During nine weeks in hospital the infant's weight remained obstinately stationary in spite of various changes in diet; throughout this period his appetite was poor, but dyspeptic symptoms were slight; vitamin and glandular extracts produced no beneficial results.

CASE I.—Weight Chart. Feeds: Week 1—simple milk and water; week 2—Bengerized milk and water; weeks 3 to 6—lactified milk and water; weeks 6 to 15—Bengerized milk and water; week 16—simple milk and water. A, admission; D, discharge.

Ultimately the infant was placed in the oxygen cot for three weeks, the diet remaining the same in type as during the three previous weeks. While in the cot he gained 1 lb. in weight and another pound was added during the ensuing three weeks before discharge. The child's appetite commenced to improve after the initiation of the cot treatment; the colour also improved and the muscular tone became much more firm.

CASE II.

A male infant, aged 2 months, was admitted with a history of failure to gain weight and of some previous mild dyspepsia; the type of food had been altered two or three times without pro-

ducing any good results. On admission the infant was pale, with flabby muscles, but on the whole development was quite good. No abnormalities in the organs were present. The weight after six weeks was the same as on admission, though the appetite had been fair and no dyspeptic symptoms had been present. During

CASE II.—Weight Chart. Feeds: Week 1 to discharge—simple milk and water. A, admission; D, discharge.

twenty-three days in the oxygen cot, on the same form of diet, 12 oz. of weight were gained, and a further 17 oz. in the interval between the termination of the cot treatment and discharge. Muscular tone and general condition had also improved markedly since the commencement of the oxygen treatment.

CASE III.

A male infant, aged 1 month, was admitted with an indefinite history of failure to thrive in spite of partial breast-feeding. He was, on admission, a small, pale, and slightly wasted infant, with a spotty skin; the Wassermann reaction was negative. The child was tried on various kinds of food, and for a time even received some breast-milk feeds; he took feeds well, with only one slight initial attack of dyspepsia, but he completely failed to put on weight, and after six weeks in hospital only weighed 1 oz. more than on admission. With no alteration of diet, the infant was then placed for three weeks in the carbon dioxide cot (in which the carbon

CASE III.—Weight Chart. Feeds: Week 1—simple milk and water; weeks 2 to 4—Bengerized milk and water; weeks 4 to 16—lactified milk and water; week 17—simple milk and water. A, admission; D, discharge.

dioxide was allowed to accumulate but the oxygen content was kept approximately equal to that of the atmosphere). During this period 7 oz. of weight were gained. After three days' interval he was transferred to the oxygen cot for three weeks, and in this cot 17 oz. were gained, and another 27 oz. before discharge six weeks later. Later on the child developed some septic areas on his scalp with subsequent glandular enlargement, and also some aural discharge. All these conditions, however, cleared up satisfactorily, and on discharge the infant's general condition had greatly improved.

Since the small increase in carbon dioxide in the air of the cot without any oxygen enrichment was beneficial, we are inclined to accept this as supporting the view that some degree of general anoxaemia was present in these cases and that this was the cause of their failure to thrive. The increased ventilation of the lungs which the greater amount of carbon dioxide produces would in any case be of benefit if anoxaemia were present, but one would expect a better result by enriching the air with oxygen as well. This has been our experience.

It is obvious that further data from a much larger series of cases will have to be collected before more can be said about the treatment than that placed on record here. The work is therefore being continued and extended.

We wish to express our thanks to the Medical Research Council for defraying a part of the expense of this work.

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