indication of the position of his injury. After a few hours he was able to understand simple questions, but could not give any information regarding his accident. The convulsions always commenced with flexion of the left thumb, rapidly spreading to the arm, and then becoming general. The head and eyes were always turned to the left side. There was well-marked optic neuritis on the right side.

He gradually passed into a condition of status epilepticus with stertorous breathing, profuse sweating, and complete coma. The temperature was 102° F., and the pulse 130. He was apparently dying, and as there was no definite scar or depression to indicate the position of the injury, it was decided not to operate; but his wife begged that something should be done, and I reluctantly consented.

On September 6th, his head was shaved, and the following operation performed. As he was quite unconscious, no anæsthetic was required. A large semicircular incision was made, so as to reflect downwards the whole of the scalp of the forehead. When this was done it was seen that the right frontal bone and half of the right parietal bone were depressed to the extent of quite one-eighth of an inch. A disc of bone was removed with a 1-inch trephine. The dura bulged to the level of the outer table, so great was the intracranial pressure. Another disc of bone was removed, and then I chipped away with forceps the greater part of the right frontal and the anterior portion of the right parietal bones, leaving an opening measuring $3\frac{1}{2}$ inches by $2\frac{1}{2}$ inches. The dura mater was not opened, and the bone was not replaced, but the wound was stitched up and a drainage tube inserted.

He recovered consciousness in six hours; the temperature rapidly dropped to normal; the pulse, which was 140 when he left the theatre, had slowed down to 100 by the evening, and after a natural sleep, he was able next morning to express himself as feeling better.

He made an uninterrupted recovery; the wound healed by first intention; and in eleven days he was walking about the ward.

On March 12th, 1899, he presented himself for inspection. He had been at regular work ever since his discharge on October 12th, and was feeling perfectly well, and had had no fit or twitching since the operation. He was directed to wear an ordinary cap with a large thick pad to protect his head.

ON THE ATROPHIC MARASMUS OF NURSLINGS (SO-CALLED HOSPITALISM OR ATHREPSY OF PARROT).*

By ADOLF BAGINSKY, M.D.,
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Among the multifarious diseases of children connected with disturbances of the digestive apparatus there is one that has for some time especially attracted the attention of physicians. It is characterised by a persistent decrease in weight, with gradual lessening of bodily powers and progressive exhaustion, with the occurrence of more or less marked complications of the respiratory organs, the skin, and the kidneys. The disease has been noted from time immemorial to occur in institutions where nurslings are taken care of. The affection has always been a source of terror to those in charge of foundling asylums, children's nurseries, and the like, on account of the extremely high mortality which has at times made it appear questionable whether it was worth while to attempt the rearing of children in such asylums at all. Charles West, in his *Lectures on the Diseases of Infancy and Childhood* (fourth edition, London, 1859) expressed the opinion that the affection was due to the exhaustion of the various organs.

Since the important advances which have been made in recent years in children's diseases, especially since the extension of our knowledge of anatomy, physiology, and epidemiology, various attempts have been made to make the clinical and pathological anatomical relations clearer. Investigators seem at the present moment to be of two opinions. One group of writers directs special attention to chronic catarrhal processes in the intestinal tract with their ultimate consequences. They attribute these catarrhal conditions to insufficient or faulty nutrition and to faulty nursing and care. The other group of writers lay more weight on bacterial infection, and above all on the influence of toxic substances. These toxins are the products of intestinal bacteria, and may be absorbed without any real lesion of the intestinal mucous membrane existing, and it is thought that by a process of chronic intoxication they seriously disturb the vegetative functions. The clinical aspects of the whole question were very well set forth by the French writer Parrot some years ago in a series of very comprehensive articles to which very little of importance can be added. To the affection Parrot gave the new name "athrepsia."[1]

I will spare you the exact description of the affection, which is unfortunately only too well known, and will content myself with showing you, for the purpose of recalling some of the more exaggerated features of the disease, a few photographs from my clinical cases. You will recognise the *macies maxima*—the deep sunken eyes, the wrinkled face, the piteous expression of countenance, the withered appearance of the skin as it covers in folds the atrophied muscles, while the fatty subcutaneous layer has practically entirely disappeared. There are, however, no rachitic changes in the bones, which are, on the contrary, well formed and without beading.

The first question that arises for our consideration is whether, since the disease is so notoriously common in children's asylums, we have to think of it as an infection or not. In answer to this question I may say that I have never seen this progressive atrophic condition spread from bed to bed in a hospital, but have always found that cases occur sporadically. When cases are observed side by side the relation is merely an incidental one; the children fall away gradually but very slowly, and at different rates of progress, not as though affected by the same pathological conditions, but finally reaching the same pitiable goal. Especially we find chronic dyspeptic disturbances occurring with varying degrees of severity, with occasional mild exacerbations, diarrhœa that is not severe running its course with or without vomiting, sometimes even constipation or normal-seeming motions, while furuncles may occur on the skin, and otitis media duplex may supervene.

As may well be imagined all this brings the children down in condition more and more, at times one complication, at times another seeming responsible for the deterioration in health. Remissions, however, may take place, and there may even be a gain in weight, which, however, is usually lost again in a short time.

No one can doubt that in all varieties of the affection bacterial influences have a place, and that the bacillus coli, the streptococcus, staphylococcus, pyocyaneus, and pneumococcus play a certain *rôle* in the etiology; yet these influences are not such that we can attribute to them any specificity in the production of the anomalies of nutrition that result. The really efficient cause of the condition in children's asylums is the same as that which is at work in the homes of the poor: It is the want of proper and sufficient care. The errors are, perhaps, only slight, but they suffice, when added together, to produce the condition. It may be that the child is let lie too long in damp or dirty wrappings, that it does not receive thorough cleaning immediately after vomiting, so that minute particles of vomited material are carried by aspiration into the lungs. When the child is fed by the bottle, it may be that the milk is given, now warm, now cold, or with the child lying down, so that again opportunity for the aspiration of food particles into the lungs is afforded. Even when apparently the greatest care is taken as to cleanliness of both bottle and nipple, other causes remain at work. In short, it is to a series of defects in care that the serious disturbances that are ultimately produced are really due. If each child could receive special attention for itself these defects might be avoided. Of course, I cannot deny that in a nursling department the communication of disease by child to child may easily take place, but for this the hospital as such is not responsible, but it is the fault of the nurses, who are either too few in number, or lacking in proper training, or who do not conscientiously fulfil their duties.

What are, then, the physiological and the anatomical conditions found *post mortem*? With the help of my chemical assistant, Dr. Summerfield, I have recently made a series of

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researches on the metabolism of such cases. In one case I found the following conditions:

G., aged 2½ months, was admitted into the hospital on February 6th, 1898, suffering from extreme marasmus. The child was very much wasted, and the following table shows the results of two series of researches, directed to ascertaining the quantity and kind of nourishment actually absorbed.

I.—*As Nourishment taken, Milk, 1; Rademann's Meal, 2. 10 c.cm. contained 0.0242 g.N.*

Date.	Nourishment Taken.			Urine.	Sp. G.	N.	Fæces.
	c.cm.		g.N.	c.cm.		g.N.	
Feb. 16	620	=	1.500	205	1004	0.85	Feb. 16 to Feb. 19: g. g.N. 97.6 = 3.133
,, 17	510	=	1.234	160	1010	0.85	
,, 18	660	=	1.597	230	1000	0.74	
,, 19	650	...	1.573	205	1004	0.67	

	Total.
Excreted N. from urine, 3.11 + Fæces, 3.133 =	6.243
N. taken in Nourishment...	5.904
Lost	0.339 g.N.

From N. 5.904 g. = 52.7 per cent. have passed the intestines without digestion, and given out by fæces.

II.—*Nourishment the Same. 10 c.cm. = 0.0242 g.N.*

Date.	Body Weight.	Nourishment N.	Urine. Quantity.	Urine. Sp. G.	Urine. N.	Fæces.
Mar. 1	3800					
,, 2	3750					Total = 64.9 g.
,, 3	3750	c.cm. g.N.				
,, 4	3680	520 = 1.258	120	1009	2.627	1 g. = 0.039 N.
,, 5	3620	600 = 1.452	105	1009	2.286	
,, 6	3570	610 = 1.476	257	1007	2.018	Total: 2.531 g.N.
,, 7	3550	540 = 1.306	150	1010	1.996	
,, 8	3500	550 = 1.331	125	1008	2.479	
,, 9	3550					
,, 10	2520	2820 = 6.823				

Total taken: 6.823 N.

Excreted with urine,	11.403	Excreted with fæces, 35.5 per cent. N.
,, ,, fæces,	2.531	Lost N. total 7.111 g.N.
	13.934	

Most of the child's loss in weight was found to be due to its loss of nitrogenous substances. Is this result to be explained on the theory of a special condition of the intestinal tract, or are there other circumstances that account for it? The intestinal canal was acting under especially favourable conditions, for 37.1 per cent. of the nutritive substances introduced into it were unassimilated, and excreted again in the fæces.

I also made a series of detailed anatomical studies of the condition of the intestinal canal *post mortem*, and found sundry departures from the normal.

When I first began my researches on this question[2] I came to the conclusion that in these cases serious lesions existed, which were at times utterly destructive. I found that these lesions were not of equal severity over the whole intestinal canal, but I could demonstrate perfectly healthy parts lying in the neighbourhood of parts that had suffered severely. I have lately continued my researches on the intestinal canals of children who had died of marasmus. Specimens were in every case removed in less than two hours after the death of the subject, so that I consider myself thoroughly guarded against the possibility of error through *post-mortem* changes. My recent researches have confirmed those previously made. Especially striking was the fact that beside absolutely healthy parts of the intestinal tract I found others involved in the severest destructive processes in the mucosa, with atrophy and sclerosis of the remain-tissues of the intestinal wall. The gastric mucous membrane as a whole seemed well preserved, at least not markedly atrophied; not so the intestinal walls. Changes were, however, already in progress. In the upper part of the small intestine these were of the nature of an inflammatory proliferation; further on in the small intestine they took on an atrophic character. Where the former process was in progress one could see peculiar prolongations and outgrowths of Lieberkühn's glands and of the villi, presenting an unusual bent and twisted conformation; while there was an overgrowth of the epithelial layer and an exudation of round cells into the interstitial tissue. The process was evidently one of chronic catarrh, perhaps even associated with inflammation.

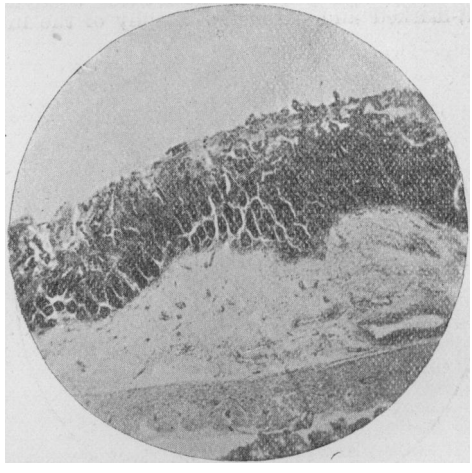

Fig. 1.—Commencing proliferation of the mucosa (chronic irritation).

In such a patch the submucosa and the muscularis are, on the whole, not much altered; at most there is noticeable a great overfilling of the blood vessels, with exudation of round cells. Close by such a patch, however, other patches may be found with perfectly unaltered mucous membrane and villi, the whole epithelial layer, especially the layer in which the glands are situated, being absolutely normal.

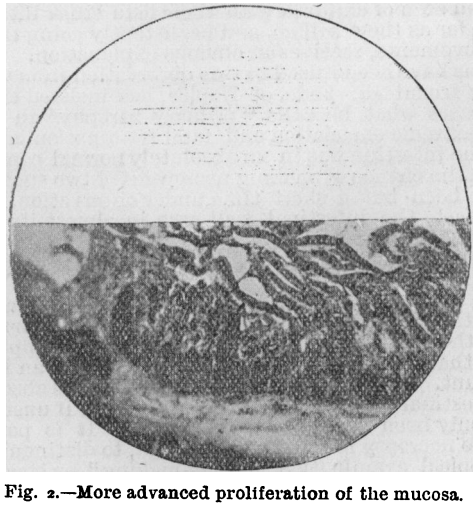

Fig. 2.—More advanced proliferation of the mucosa.

In striking contrast to these one comes suddenly upon patches that give the impression that most serious destructive processes are at work, the epithelial layer destroyed, and the villi to glandular layer almost completely gone, so that the observer is scarcely able to recognise what the original condition of the mucosa was from its ruined remains. In place of the former villi and gland layer nothing is left but collections of utterly irregular-deformed cells, some with enlarged or irregularly-shaped nuclei.

The whole mucosa is reduced in thickness to a minimum,

the submucosa and muscularis sharing the same fate. The whole process makes a complete picture of progressive atrophy. The disease involves also the lower end of the small intestine, and extends into the large intestine. At times we find the large intestine also studded with round cells and with thickened interstitial layers and proliferation of glands and epithelial cells. The proliferation spreads superficially, and also deep, so that the appearances are those which I indicate in the works I have mentioned as characteristics of chronic catarrh.

The chief characteristic in the pathological anatomy of the marasmus of children, then, is that, in addition to the catarrhal lesions and evidently as a result of these, there is a more or less well-marked and extensive atrophy of the intestinal

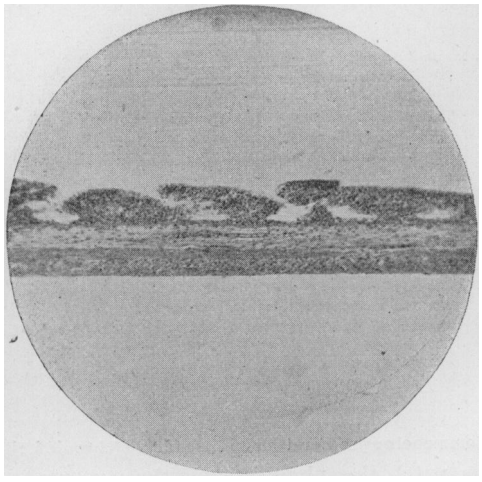

Fig. 3.—Atrophy of the mucosa.

walls. All the layers of the intestine are affected, and the consequence and quintessence of the whole process is the evident incapacity of the subjects to assimilate and absorb the nutritive material ingested. As a consequence of this loss of the nitrogenous substances of the body muscles, etc., there is persistent thirst and hunger, so that the clinical picture so often seen of children with their fists stuck into their mouths as far as they will go, and persistently going through sucking movements, receives an obvious explanation.

Objections have been raised to this observation from various sides. My friend Fr. Fede, of Naples, has insisted that he has seen cases what he calls "primary atrophy" in which there was extreme emaciation and fatal termination, and yet in which the intestine was in an absolutely normal condition. I have had the chance of making necropsies of two such cases which had been but a short time under observation. As a matter of fact, the intestinal wall was in almost its whole extent perfectly normal, certainly not atrophic, though here and there the signs of a catarrhal and inflammatory process were evident.

In these two cases, however, all the external appearances pointed to the fact that they had actually died of starvation. Not that they had failed to absorb nutriment supplied to them, but that they had in all probability ingested an insufficient amount. Such children may of course have absolutely normal intestinal mucous membranes, especially if unsuitable food has only been given for a short time. It is possible, then, if the necropsy is done soon enough, to distinguish by a microscopical examination "angel-making" by starvation from marasmus due to atrophy of the intestines. This may be of importance from a medico-legal point of view. There should be no question, to my mind, of talking, in the cases of really starved children, of death from "primary atrophy," as if this were a disease; on the contrary, we should call it very simply, in contradistinction to the sharply defined clinical and pathological picture that marasmic atrophy of the intestines gives us, death by starvation.

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TRACHOMA AND RACE.

BY MAJOR M. T. YARR, R.A.M.C.,
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OF the many problems connected with trachoma—a disease which as a scourge of the human race may fitly rank with leprosy, tubercle, and syphilis—perhaps the most interesting is the influence of race on its genesis and spread. The literature of the subject is scanty, and many points require further elucidation, but already one great fact stands out sufficiently clearly—namely, that the dominating influence in the etiology of trachoma is race. The labours of Swan Burnett, Chibret, and others show that the races composing the population of the world vary to a most extraordinary extent as regards receptivity of the trachoma virus, and that this variation cannot be explained by differences of civilisation and sanitary surroundings, or even, except in a limited sense, by differences of climate. Hence any attempt to study the distribution of the disease from a purely geographical standpoint—to construct a "trachoma map" of the world, so to speak—is foredoomed to failure; the trachoma problem is one of race, not place. No one can deny the influence of want, overcrowding, filth, and insanitary surroundings on the spread of what is, to a certain extent at all events, a contagious disease, but these influences are entirely subordinate to two great factors—first, race, and, secondly, but *longo intervallo*, climate.

RACE.

It can be shown that one race at least seems to be incapable of contracting trachoma; that other races enjoy a relative immunity; that others, again, are receptive to an extraordinary degree; and that between the two latter classes come races which are neither immune nor exceptionally receptive.

1. *Absolute Immunity.*

It appears to be certain that the Canadian indigenous tribes, including the Esquimaux, are entirely immune; both the specialists and the general practitioners of the Dominion are unanimous in their belief in this *absolute* immunity. Dr. Foucher, the well-known ophthalmic surgeon of Montreal, has never seen a case of trachoma amongst the savage tribes. An inquiry addressed by him to Desjardins of Montreal, Simard (Quebec), Orsey (Ottawa), Dudge (Halifax), Agnew (Winnipeg), and Benoit (Edmonton), all oculists of note, elicited a similar experience from his *confrères*; and confirmatory evidence from the general body of the profession was obtained in response to a circular note sent through the Medical Union of Canada.[1]

This immunity is the more extraordinary, inasmuch as these savages live under all the conditions usually considered most favourable to the development and spread of trachoma. They herd together in tiny hovels, their habits are filthy, and their food insufficient; tubercle and syphilis are rife, and epidemic diseases, such as scarlet fever and small-pox, ravage them to an extent unknown amongst civilised peoples. Dr. Foucher gives two striking examples in tribes with which he is personally familiar. The Cauglinawaga tribe, the nearest to Montreal, live on a reservation situated in a low-lying, damp, marshy country; their huts are overcrowded to an unheard of extent; cleanliness is unknown; a large proportion are syphilitic, tuberculous, or both; epidemic diseases are common; eye affections, too, are common, with one exception—trachoma—which is never seen. A still more remarkable instance, showing the powerlessness of contagion to affect racial immunity, is that of the Cris and Santeux of Manitoba, who live side by with the Russian Mennonites under precisely the same conditions of defective hygiene. The Mennonites suffer greatly from trachoma, blindness from this cause being very common their neighbours, the savages, are never attacked.

The Indians of the United States, who do not belong to the same race as the Canadian Indians, are by no means free from trachoma. Indians from the Canadian tribes are found in Pennsylvania, and are presumable immune, but unfortunately we have no information.

2. *Relative Immunity.*

Pure-blooded negroes, such as those from Senegambia, Guinea, and adjacent parts of the West Coast of Africa, enjoy