ELLIOT (G. T.) & MERRILL (Wm H.)

A Further Study of Alopecia Prænatura or Præsenilis, and its most Frequent Cause—Eczema Seborrhoicum.

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Professor of Dermatology at the New York Post-graduate Medical School, etc.

And a Preliminary Bacteriological Report on

Eczema Seborrhoicum:

For the First Time Successful Production of the Disease by Inoculation of Pure Cultures of Certain Diplococci.

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A FURTHER STUDY OF ALOPECIA PRÆMATURA OR PRÆSENILIS, AND ITS MOST FREQUENT CAUSE— ECZEMA SEBORRHOICUM.*

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PROFESSOR OF DERMATOLOGY
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AND A PRELIMINARY BACTERIOLOGICAL REPORT ON ECZEMA SEBORRHOICUM:

FOR THE FIRST TIME SUCCESSFUL PRODUCTION OF THE DISEASE BY INOCULATION OF PURE CULTURES OF CERTAIN DIPLOCOCCI.

By WILLIAM H. MERRILL, M. D.,
PEPPERELL, MASS.

At the meeting of the American Dermatological Association held at New London in 1892, I read a paper on Alopecia Præmatura,† in which it was claimed that in the overwhelming majority of cases the cause of the condition was a local one, represented by various grades of that form of disease known in more recent days under the name of eczema seborrhoicum, but formerly as seborrhœa sicca, pityriasis capitis, etc. I had based my conclusions upon an analysis of two hundred and thirty-four cases treated in

* Read before the American Dermatological Association, at Montreal, September, 1895.

† Elliot, *New York Medical Journal*, 1893.

my private practice, of which two hundred and seven, or 88·46 per cent., had been due to one or another grade of this same local process, while the remainder—twenty-seven in number—had been the result of various general and systemic diseases and conditions. The discussion of my paper by the members present at the meeting elicited, however, views materially different from my own. Alopecia, in their opinion, was due to hereditary causes rather than to local conditions, or it proceeded from mechanical irritation, bad hygiene, or other factors of similar import. Dr. Jackson alone acknowledged a distinct causal relation between alopecia præmatura and the local affection, eczema seborrhoicum—but he qualified his acknowledgment by ascribing a greater influence to heredity than to the local disease. In view of this great divergence in opinion between myself and those members of the association who took part in the discussion, I have been led to investigate minutely each case of alopecia which has come under my care, as to the existence or non-existence in any one of them of a basis for claiming heredity as a cause. The condition of the parents' hair, whether either one of these had been or were subjects of alopecia, was rigidly inquired into, and as far as possible the cause of such loss, if existent, was ascertained.

At the same time the state of the patient's scalp at and prior to the date of consultation was investigated, and the duration of the alopecia in each was noted.

For the sake of accuracy, the material included in this paper was obtained from my private practice alone, and the cases included were those treated during the two and a half years—January 1, 1893, to July 1, 1895. Their total number was three hundred and forty-four.

The ætiological causes found active in the production of the alopecia were:

Alopecia areata	4
Constant headaches and neuralgia	4
Neurasthenia	4
Senile alopecia	2
Malarial and scarlet fever	2
Erysipelas	1
Diabetes	1
Pregnancy	1
Without apparent or traceable cause	5
Heredity... { From mother's side, 1 / From father's side, 3 }	4
Eczema seborrhoicum in various stages and degrees	316
	344

It was thus found that five, or 1·45 per cent., originated from unknown causes; nineteen, or 5·52 per cent., were due to general systemic disturbances; three hundred and sixteen, or 91·86 per cent., were the result of local disease; and only four, or 1·16 per cent., could be attributed directly to heredity—that is, in these four no other factor or cause existed or was present except the loss of hair at an early age in one or other of the parents, and no other cause was present in the patients themselves. If the fact alone that the parents lost their hair at an early age was entertained in investigating the ætiology and the pathological condition of a patient, then there were sixteen among the three hundred and sixteen cases which could be also included in the category of heredity. Nevertheless I have excluded these, and I believe with entire justice, for, though three of these patients had mothers whose hair was always thin and poor in quality, and thirteen had fathers bald in various degrees at early ages, yet not one of the sixteen had shown the slightest symptom of alopecia

until some months or years after the development on the scalp of some one or other grade of seborrhoic eczema. It is for this reason that I have included these sixteen cases among those others due to the local process eczema seborrhoicum, and as examples of some of them I would mention:

Case I.—A man, aged twenty-eight years, in excellent general health. Father became progressively bald from no traceable cause at the age of twenty-five years. Patient always had abundant thick hair, but alopecia had begun at the age of twenty-three years. Three years before, eczema seborrhoicum squamosum of a severe grade had developed, and when I saw him the symptoms consisted of red patches and areas covered with greasy crusts and a fatty incrustation distributed generally over the vertex; itching and burning were severe at times. Hair much thinned over affected area.

Case II.—A woman, aged twenty-two years, delicate in health, but otherwise well. Mother's hair always scanty and thin and also grandmother's. Her own hair always scanty, but not changing at all until one year before I saw her, when the alopecia became instituted. Six months previously, pityriasis capitis had developed. When seen by me, there were red, circumscribed patches covered with greasy scales and crusts distributed over the vertex, and a pityriasic condition over the temporal and occipital regions. I would also particularly mention, in view of the patient's statements in regard to heredity as the cause of her infliction, that the mother was also seen and examined by me. She was found to have the *same* local *disease*, and to have lost the greater part of her hair after the daughter was ten years of age.

Case III.—A man, aged thirty, in perfect functional health. Family history gouty. Father became entirely bald over vertex at a very early age, and from some undescribed cause. Patient's hair exceedingly thick and curly over the entire scalp. Pityriasis capitis for several years. Alopecia one year. Marked eczema seborrhoicum squamosum at time of consultation. Patient's wife treated also for severe alope-

cia. No alopecia in her family on either side, but eight months before I saw her she had had severe eczema seborrhoicum over the scalp.

Two sisters of the patient in Case III, likewise treated by me, gave the same history of father's alopecia. Both abundant and magnificent hair. Both severe grades of eczema seborrhoicum, with itching and burning for several years. Alopecia respectively eight and six months.

It certainly does not seem to me that, in view of the facts presented by these cases, any warranty exists for accusing heredity as a direct causal factor of the alopecia, and the same may be said in regard to the remaining thirteen of the sixteen referred to here, whose histories were practically identical with those already given. I assuredly do not deny that alopecia may be hereditary, but I do not believe that in cases similar to these it can be looked upon in any other way than as a predisposing factor, in so far as an individual may acquire by heredity hair poor in quantity and quality. When this is the case the effects of any disease of the scalp may be more severe, but the alopecia which takes place is certainly produced by the local affection present at the time and not by the inherited deficiency in quality or quantity of the hair. It would be as logical to postulate heredity for loss of hair after typhoid fever because the patient's father was bald at an early age as to do so in these cases, all of which were sufferers from a local process which is generally acknowledged by all writers to be a potent and frequent cause of alopecia. In order to arrive at a satisfactory conclusion in regard to this question of heredity, I found it particularly important not to be satisfied with the bare statements alone of the patient, but to investigate personally and as far as possible into the family history. As a result of doing this, the accusation of responsibility for the alopecia brought against parents

was disposed of in all my cases except those already mentioned. Such conditions were frequently found as stated in the history of Case II—that is, the same local disease was present in parent and child as a cause, or the alopecia was due to some general systemic disease, condition, or dyscrasia, and post-dated the birth of the child by a number of years more or less, and not a few were fine examples of senile alopecia. In reality, I found by investigating the statements of the patients very slight basis of support for the claim of heredity, unless some such proposition was entertained as "post natal" heredity by means of which a child inherits from the parent a condition acquired by the latter years after the birth of the former—a certainly untenable proposition.

After carefully investigating, therefore, the parents' and the patients' histories, and excluding the twenty-four cases of alopecia due to some general systemic disease or condition, or which were of unknown cause, I would divide the remaining three hundred and twenty as follows: Absolutely no basis for accusing heredity as a cause, 290; heredity possible, but no alopecia until some time after the development of a local disease—eczema seborrhoicum—16; no definite information *pro* or *con* obtainable, but eczema seborrhoicum present on the patient, 10; heredity and no other factor existing, 4.

The result obtained from analyzing my cases, therefore, justifies me, I believe, in stating again that in my experience heredity as a cause of alopecia is possible and does exist, but it is exceptional and by no means the rule, as is generally believed and held.

In place of heredity it has, however, been my experience in the last two years and a half to again find that the most frequent cause of alopecia was that local disease representing some phase or grade of the process known to-

day as eczema or dermatitis seborrhoica, and in view of this renewed experience I may be allowed to refer briefly to certain features presented by the affection in its relation and bearings toward the individuals affected. In my former paper I stated that two hundred and seven out of two hundred and thirty-four cases of alopecia were the result of a seborrhoic eczema of variable duration—from a few months to a number of years—that is, 88·46 per cent. were the expression of the effects of the local disease. Much as that result was surprising, in view of the opinion generally held that heredity was the prime and most frequent cause, yet to day the percentage is still higher—91·86 per cent.—and three hundred and sixteen out of a total of three hundred and forty-four cases owe their origin and existence, in my opinion, to the local disease, and that after every other systemic, general, accidental, or other factor had been excluded by rigid investigation. It may, however, be advanced that notwithstanding the presence of the local disease it in reality had only a certain share in the production of the alopecia; there was something else—something more, something necessary for its own existence—and that it is this which is the real and important cause of the alopecia, the eczema seborrhoicum being only an accessory. There is no reason to object to the claim that there are some factors or conditions which render one individual more favorable for the development of the disease than another; there is no question but that the effects of the process may be very rapid in one, very slow in another, and nil in still another; that the affection may exist for years without producing any alopecia. There is no more doubt that these conditions exist in seborrhoic eczema than that they do in all other diseases of whatever nature, origin, or causation they may be, but they are the expression of certain unknown states of cell life; are indi-

vidual in the fullest sense of the term; are usually designated as predisposition, "nature of the soil," etc., without, however, being more nearly understood or defined; and can not certainly be regarded as determining or active causes of a condition or disease, but only as favoring or non-favoring factors, such as render the individual more or less liable or refractory to the development of a morbid process and to its effects. Consequently their interposition does not negative the claim that the active factor—the cause of the effect—is the local disease, which, it may be granted, may produce its effects easily or with difficulty, or not at all, according as the "soil" was more or less favorable. I must confess that so far as the production of alopecia by the disease eczema seborrhoicum is concerned, I could not estimate the rôle played by these possible but remote and indefinable factors. It was observed that the symptomatic evidences and effects of the disease existed irrespective of the systemic condition of the patients, was neither favored nor the opposite by any somatic state; it was seen that the alopecia persisted so long as the local process was allowed to exist, ceased with its removal, but returned with its reappearance, and under such circumstances the question certainly arises, By what should one abide? By the indefinable, remote, and unknown factors constituting predisposition, or by the objective, active, and determining cause—the local disease? That is the position in which I found myself placed after careful observation and investigation into the antecedents of my patients, the conditions existing at the time of consultation, and the effects of removing the local process; and in view of my experience I would unhesitatingly accuse some grade of eczema seborrhoicum as the direct and active cause of alopecia in the immense majority of cases.

The fact that this disease plays so important a part in

the production of alopecia is of more than ordinary consequence. It is a curable and preventable process, and its more general recognition would certainly lead to a great diminution in cases of baldness. In my experience many such are directly the result of neglect, owing to a misconception of the process at work, and to the ingrained belief that heredity is the primary, secondary, and only cause of alopecia, and hence irremediable.

There is no need to refer particularly to the clinical symptoms of eczema seborrhoicum,* as they have already received full description in other papers on the subject, and to do so would only involve repetition. I would rather call brief attention to some other features obtained from the present analysis of my cases, and then to take up the perhaps most important part of my paper.

The proportion in regard to sex is practically the same as previously found by me. There were then 55+ per cent. females and 44+ per cent. males, while now there are 64+ per cent. females and 35+ per cent. males. In other

Table of Ages.

1 to 10 years.....	1	2	3
10 to 20 "	11	31	42
20 to 30 "	59	99	158
30 to 40 "	31	44	75
40 to 50 "	8	21	29
50 to 60 "	1	8	9
	111	205	316

words, there is a decided preponderance in number in favor of the female sex, one, however, not to be taken as abso-

* Elliot. Eczema Seborrhoicum, *N. Y. Med. Jour.*, 1891. Alopecia Præmatura, etc., *N. Y. Med. Jour.*, 1893. Article, Dermatitis Seborrhoica, Morrow's *System of Genito-urinary Diseases, Syphilis, and Dermatology.*

lute, but as entirely relative, owing to the fact that women seek aid for their afflictions much oftener than men.

According to the table of ages, I may say that my former statements are substantiated by it, since the largest number of cases occurred in the decade twenty to thirty, and the next largest in the ten years thirty to forty. In 1892, 47+ per cent. occurred in the former and 31+ per cent. in the latter, while now the proportion is 50 per cent. and 23 per cent. respectively. More important, however, is the fact that two hundred and three cases, or 64+ per cent. of the whole, occurred before the age of thirty, or within those years in which alopecia may certainly be called premature, and also at a time when systemic and bodily health is at its best, and has not yet been taxed and strained to its utmost by the mode, conditions, and struggles of life. In other words, at the period of life when the recuperative powers of the body are most active, when the presence of systemic conditions sapping strength and nutrition are to the greatest extent wanting, it is then that the alopecia is most frequently initiated and sustained, instead of its having its origin later in life, when so many concomitant bodily factors may be present as adjuvants and aiders in the process. This seems to me to be certainly an additional argument in favor of the decisive influence of the local disease, as against those other remote and undemonstrable predisposing factors which are spoken of but not described, and I may also add that, in my cases at least, the condition of systemic health could not be appealed to as exerting any effect in the production of the loss of hair.

Of my patients, seventy-two per cent. were in good general and functional health, while the remainder furnished details pointing to gastric or intestinal dyspepsia of varying degree, to constipation, anæmia, rheumatism, gout, or some slight ailment of imagination or of fact, such as may

and does occur in connection with any skin disease. None, however, were in such condition as would preclude their coming to my office, or were in reality what might be called ill.

After reviewing carefully all my cases, after analyzing them with all possible rigidity, and after weighing all the *pros* and *cons* of the question during the last two years and a half, I am therefore compelled to repeat emphatically the assertion made by me in 1892—viz., that a local disease, eczema seborrhoicum, is in the overwhelming majority of cases the cause of alopecia.

The object of my paper was not, however, alone the reinvestigation of the causation of alopecia, but more particularly was it intended to bring forward a question in the pathology of eczema seborrhoicum, one of the very highest importance, and one to which attention is more particularly asked than to any other part of the subject.

Some years ago I had the pleasure of arousing interest in the question of seborrhoic eczema in the mind of my friend Dr. William H. Merrill, of Pepperell, Mass., and for several years he has devoted himself, as far as his time permitted, to the study of the bacteriology of the disease. The practical results of his work he has communicated to me from time to time, but, thinking that this was a fitting occasion for making his work and its results public, he has at my request kindly sent me the following preliminary report of his experiments and investigations:

BACTERIOLOGICAL STUDY OF ECZEMA SEBORRHOICUM, ETC.

BY WILLIAM H. MERRILL, M. D., PEPPERELL, MASS.

The material used for the bacteriological experiments was taken from the scalps or faces of patients presenting all the necessary symptoms of seborrhoic eczema. The cases utilized were fifty in number, and in forty-eight of

these germ life of some kind was found. Two (Cases VI and VII, author's notebook) were sterile, it being impossible to obtain any growth from the scales by any method, and it is a most interesting fact to note that on the day previous to the collection of the material from both of these cases the eruption had been freely bathed with a solution of resorcin.

Of the forty-eight, only two (Cases III and XXXI, author) showed any growth in bouillon. In Case III it was produced by a large motile bacillus (not examined); in Case XXXI, by the *Staphylococcus pyogenes aureus.* In gelatin media, or in gelatin stiffened with agar, growth of some kind was invariably obtained.

In thirty-four cases the gelatin was liquefied to a greater or lesser degree, in fourteen it was not. In these latter the growths appeared only about the surface scales; but those thrust below the surface showed no development unless air gained free access to them through the needle puncture.

The cultures were always kept at ordinary room temperature. Of four cases examined in February the inoculated tubes were allowed to stand at night in a cold room (32° F.), and they remained sterile until the tenth day. Tubes inoculated in summer, however, would show a visible growth on the third day. On the fourth day (70° F.) signs of germination could generally be seen, and the time in the cases in which it began varied from the third to the sixth day after inoculation. Freezing temperatures were found to undoubtedly retard to a considerable degree the artificial growth of the germs occurring in eczema seborrhoicum. Pure cultures obtained from the experiments showed three distinct varieties of bacteria, which may be designated as Nos. 1, 2, and 3. In thirty-one cases all three were present; in seven, only Nos. 1 and 2; in two,

Nos. 1 and 3; in five, No. 1; and in one, No. 3 alone. In two cases (IX and XI, author's notebook) all three were present, and also a few colonies of the motile chromogenic *Bacillus fluorescens liquefaciens minutissimus* described by Unna and Tommasoli (Sternberg).

Variety I.—Small diplococci, single or in irregular groups. The parts forming each diplococcus are round or only slightly oval. The germs are aerobic, non-liquefying, and non-chromogenic. At 70° F. they grow rapidly. On gelatin plates the deep seated colonies remain about the size of an ordinary pin's head for weeks. The superficial colonies are round, white, with slightly raised surfaces, and smooth or somewhat irregular borders. In its growth the colony adheres very nearly to its circular form. After the first week the centre begins to turn darker, and with increasing age of the colony the whole surface, hitherto smooth, begins to be wrinkled and the edges become irregular, as though the evaporation of the water caused contraction. At the end of three weeks growth seems to stop, and the colony changes from its original white color to a dusky brown.

On agar agar the appearances closely resemble those of the gelatin colonies, except that it is slower in its growth and its surface has a whiter lustre. On potato the growth begins to be visible on the second day. On the fifth day it is cream white, smooth, raised about a tenth of an inch, and its edges are irregular and scalloped. At this time it covers about two thirds of the surface of a potato stick half an inch in diameter. After the first week the growth is slow and at the age of three weeks its size only equals that of the first week, but the colony itself is shriveled, dried, and dark in color.

In milk the culture had on the second day a slight greenish tinge, which by the fifth day had disappeared.

The upper quarter inch of the milk seems slightly thicker, but no other change is visible to the naked eye.

Variety II.—In appearance it is almost identical with Variety I, except that it seems more oval in form. This diplococcus is aerobic, non-liquefying, and chromogenic. As in Variety I, the ordinary changes of temperature, as occur from the rotation of the seasons of the year, retard or accelerate the growth of the cultures. On *Petri dishes* of *gelatin*, the minute, round, yellow colonies appear on the third or the fourth day. Those on the surface grow slowly, are slightly raised, and have smooth borders. After the first week's growth the centre shows a deeper orange color.

On *agar-agar*, the growth is slightly lustrous, thicker, and of a light orange color.

On *potato*, a deep golden layer develops, which is well raised and has irregular borders.

In *milk*, this diplococcus grows as Variety I does, except that after ten days the upper layer of the milk is thickened and has turned the same golden color mentioned.

In *stab* cultures of Varieties I and II the growth adheres pretty closely to the puncture line, gradually spreading down it and over the surface.

Variety III.—A bacillus with rounded ends, single, in pairs, or in short or long chains. It is aerobic and anaerobic, motile, liquefying, and non chromogenic.

In gelatin tubes, a grayish-white growth commences on the second day. In smear cultures, a pit of liquefied gela tin is formed, and, remaining of the same irregular shape as the smear, it gradually deepens and contains at the bottom a whitish sediment.

In stab cultures the resulting pit is the shape of the puncture and contains the same white sediment.

On agar-agar the growth is whitish, its surface raised, without lustre, and its border indented.

Dr. Unna has described three varieties of diplococci as present on the skin of patients suffering from eczema seborrhoicum (Sternberg). They all differ somewhat from the two varieties described here. Unna's two first are liquefying diplococci, while Nos. I and II are non-liquefying—a fundamental difference. His third variety, the *Diplococcus albicans tardus*, resembles No. II somewhat, but in certain aspects of its growth on the various culture media it differs decidedly. In addition to the diplococci, he has also described four varieties of bacilli as being often present in the disease. No. III seems to differ from them all. It resembles most closely the *Bacillus fluorescens liquefaciens*, being, however, larger and non-chromogenic. These differences are enough to warrant separate classification.

Inoculation experiments were attempted in twelve cases. The site of inoculation chosen was the hairy scalp, or over the sternum.

After thoroughly sterilizing the skin, two or three hairs were pulled out and the skin slightly abraded, as in vaccination; portions of an actively growing culture were then rubbed in with a sterilized platinum needle.

With No. III two attempts were made and both failed.

With No. I five attempts were made. Of these, one was a failure. In the four others the edges of the inoculation spots began to grow slightly reddened from the fourth to the sixth day, and small scales formed on the surface. By the seventh to the tenth day the spots had increased in size and were covered with dry white scales. Scales taken from these spots and placed in suitable culture media in each case gave rise to pure cultures of diplococcus No. I.

Variety No. II was used once. On the sixth day yellowish scales appeared over the surface. They grew

slightly more marked on the tenth day, and the lesion then closely resembled certain typical forms of seborrhoic eczema. Diplococcus No. II was found in the cultivations from these scales.

The last four inoculations were made with both No. I and No. II. Of these, one was a failure. Another showed a small spot covered with a few branny scales, too small to allow of any conclusions being drawn. In the other two a change began on the fourth day. The bases began to redden, and typical crumbly, greasy scales began to cover the surfaces and pile up in the centres. On the eighth day the spots were an eighth of an inch across and represented patches of seborrhoic eczema. Both Nos. I and II could be cultivated from these scales.

It is possible that Variety No. II may have given the yellow color to these scales, as it was absent in the successful cases using No. I.

The result of the twelve inoculation experiments, therefore, are: Five failures. Seven cases in which definite lesions were produced.

The percentage of successes is certainly large enough to show that either No. I or No. II, or both, bear some definite causative relation to the disease, but the number of cases is too small and the experiments too incomplete to draw any general conclusions. Certain probabilities may, however, be fairly deduced:

1. That eczema seborrhoicum is caused by a specific germ or germs.

2. That this germ is a diplococcus, or that they are diplococci.

3. That the yellow color often seen in the eruption is due to the chromogenic faculty of a germ.

4. That the wide prevalence of the disease is due to the ability of these germs to develop at the ordinary temperatures of the varying seasons of the year.

According to Dr. Merrill's report, we thus see that following the necessary steps and observing the prime requirements by which it may be judged whether micro-organisms found in a disease are pathogenic *per se*, and pathogenic in connection with the disease from which they were obtained, he determined:

1. The constant presence of certain micro-organisms in all cases of the disease examined, except in two, and in these the negative result was in all probability due to the factor he mentioned—the prior use of resorcin.

2. He cultivated these germs upon various media and obtained each in a state of pure culture.

3. He made inoculations with these pure cultures, and with two of them (Nos. I and II), singly and together, he produced lesions characteristic of the disease.

4. From the lesions produced he reobtained pure cultures of the germs.

I believe that these are the crucial tests for determining the pathogenetic influence of micro-organisms *per se* and in the production of a disease, and I think, therefore, that Dr. Merrill's work is of the very greatest importance and worthy of the highest consideration.

There were only seven out of twelve inoculations, however, which were successful, and it may in consequence be maintained that his experiments were not so very decisive, or there would have been uniform results. That would appear to me, however, to be hypercriticism, as positive and negative results occur in every series of experiments in which inoculations of bacteria are made, the one inoculated being refractory or immune, or the surface not at the time in a condition suitable for reaction toward the pathogenic element. Such states are allowed and claimed for diseases in general and of all kinds, and I do not see, therefore, why the same claim can not be postulated in seborrhóic eczema.

To myself personally the results of Dr. Merrill's work are particularly gratifying, as they corroborate so decidedly clinical observation. I have in every paper on the subject asserted my belief in the bacterial origin of the disease, basing my opinions, however, only upon clinical facts relating to its development, course, and general behavior, and especially upon the many apparently conclusive demonstrations of its contagiousness. This latter, as mentioned in my previous paper, was shown by its evident transmission from one person to another by means of direct contact in family life, by the use of hair brushes, etc., and the suggestion entertained upon these grounds that the disease was a parasitic and contagious one was, moreover, very decidedly strengthened in the last two years and a half by the clinical fact that a hundred and forty seven of my three hundred and sixteen cases occurred in several or more individuals in families. From two to six persons in the same family have been treated by me, all having the same disease, and it was repeatedly possible to trace the transmission of the process from one member to another. Of course, such evidence was only suggestive, not conclusive, and positive proof of the parasitic nature of the disease had to wait until such work had been done as is contained in Dr. Merrill's paper and a definite micro-organism had been isolated. He has unquestionably demonstrated that the disease is a parasitic one, and he is the first who has by inoculation of pure cultures of his diplococci produced lesions characteristic of those seen clinically in eczema seborrhoicum.* Unna himself by his inoculations only produced vesicles on

* In a letter to me Dr. Merrill has further stated: 1. That he did not obtain any of Malassez's flask bacilli in his experiments. 2. He also produced the lesions over the sternum by using cultures of his diplo cocci obtained from the scalp. 3. All successful inoculations on the scalp were accompanied by alopecia over the affected area.

human subjects and redness with alopecia on rabbits, but no lesions distinctly those of the disease in question.

There is in Dr. Merrill's investigations one peculiarly interesting fact brought out by the inoculations, and shown in the difference of lesion produced according to the variety of diplococcus made use of. For instance, when No. I (a *non-chromogenic diplococcus*) was inoculated, a lesion having only dry, white pityriasic scales resulted. When No. II (a *chromogenic diplococcus*) was used, then the scales were yellowish and greasy. When both I and II were inoculated together, greasy, crumbly, yellow crusts were formed. These results are most certainly particularly suggestive when it is considered that precisely similar clinical phenomena are daily seen representing the disease, and they lead to the idea that the clinical differences presented by the lesions occurring in eczema seborrhoicum are probably due to the presence of diplococcus I alone or of diplococcus II alone, or of both at the same time. The question is of great importance and will undoubtedly be solved later on. Dr. Merrill's report is only a preliminary one, and after further work he may be able to settle definitely that point and many others which are of importance in the disease.

The demonstration that the affection is a parasitic one and inoculable will also hasten the recognition of the true pathological nature of those phases of cutaneous disease which make up the process eczema seborrhoicum. Previous to Hebra they were regarded as inflammatory in nature, but he swept them all into the category of disturbances in secretion of the sebaceous glands, and to them and other clinical phenomena he gave the name of seborrhœa sicca. This has been their position ever since, and only after Unna's first attack upon their misconceived pathological position has a conception of their real nature begun to make headway. He first pointed out that they were pri-

marily inflammatory and not functional disorders in the quality of the secretion of the sebaceous glands, and subsequently in this country I have demonstrated that the various phases studied by me belonged absolutely in the class of inflammations. Now that micro organisms have been found which produce, when inoculated, the clinical phenomena of "seborrhœa sicca," it is probable that the disputed nature of the disease may finally be settled and properly understood.

The conclusions which I would make from the present series of cases of alopecia præmatura are not different from those made by me in 1892:

1. Heredity may be a cause of alopecia, but is an exceptional one.

2. Its most frequent cause is some form or grade of eczema seborrhoicum.

To these I would add:

3. That this disease, suspected and alleged to be parasitic in nature, has been demonstrated to be so in reality by Dr. Merrill's experiments and successful inoculations.

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