**Transactions of the international Medical Congress : seventh session held in London, August 2d to 9th, 1881. Diseases of children / prepared for publication under the direction of the Executive Committee by Sir William Mac Cormac ; assisted by George Henry Makins and H. B. Donkin, R. W. Parker.**

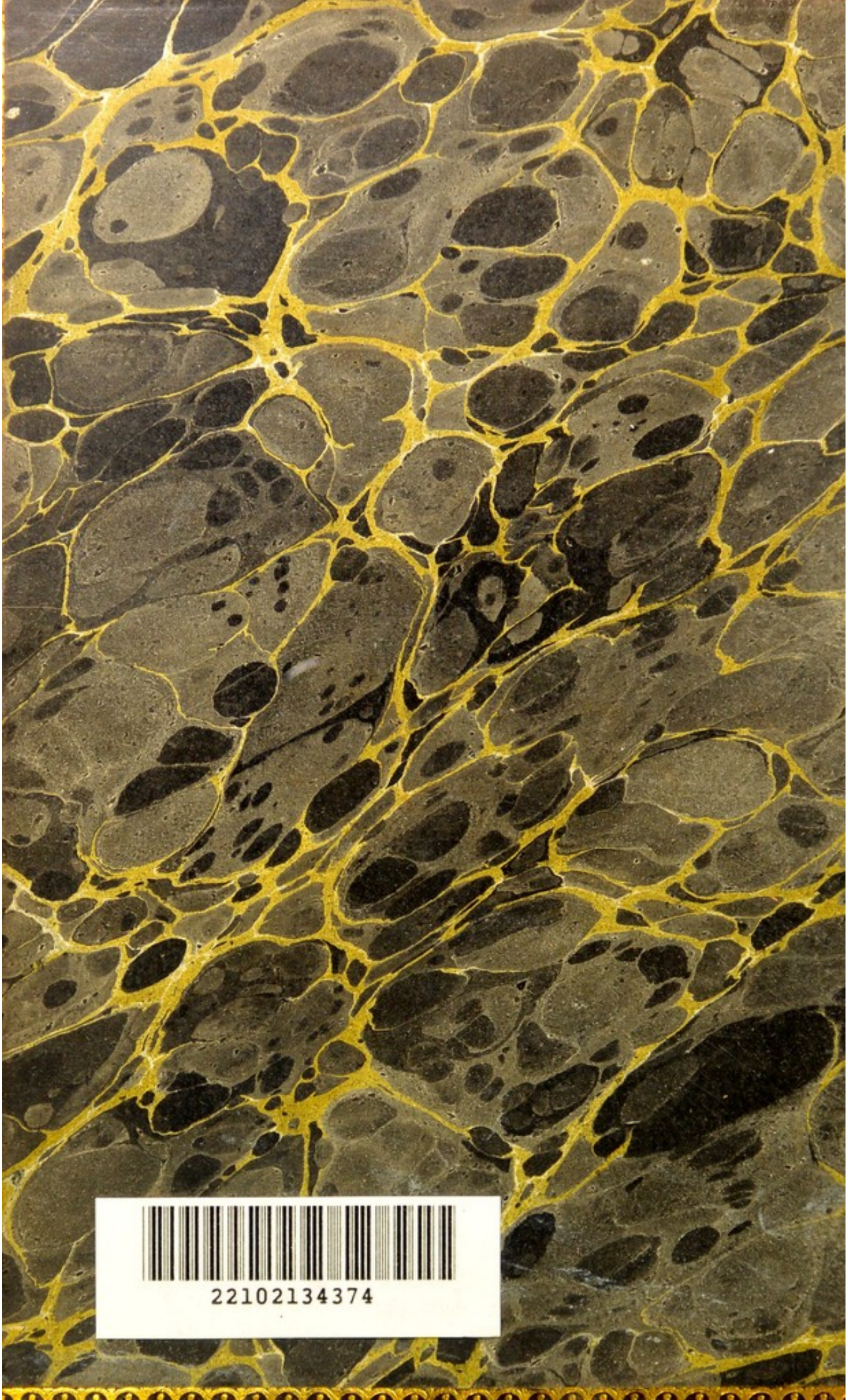
INTERNATIONAL
MEDICAL CONGRESS
1881.

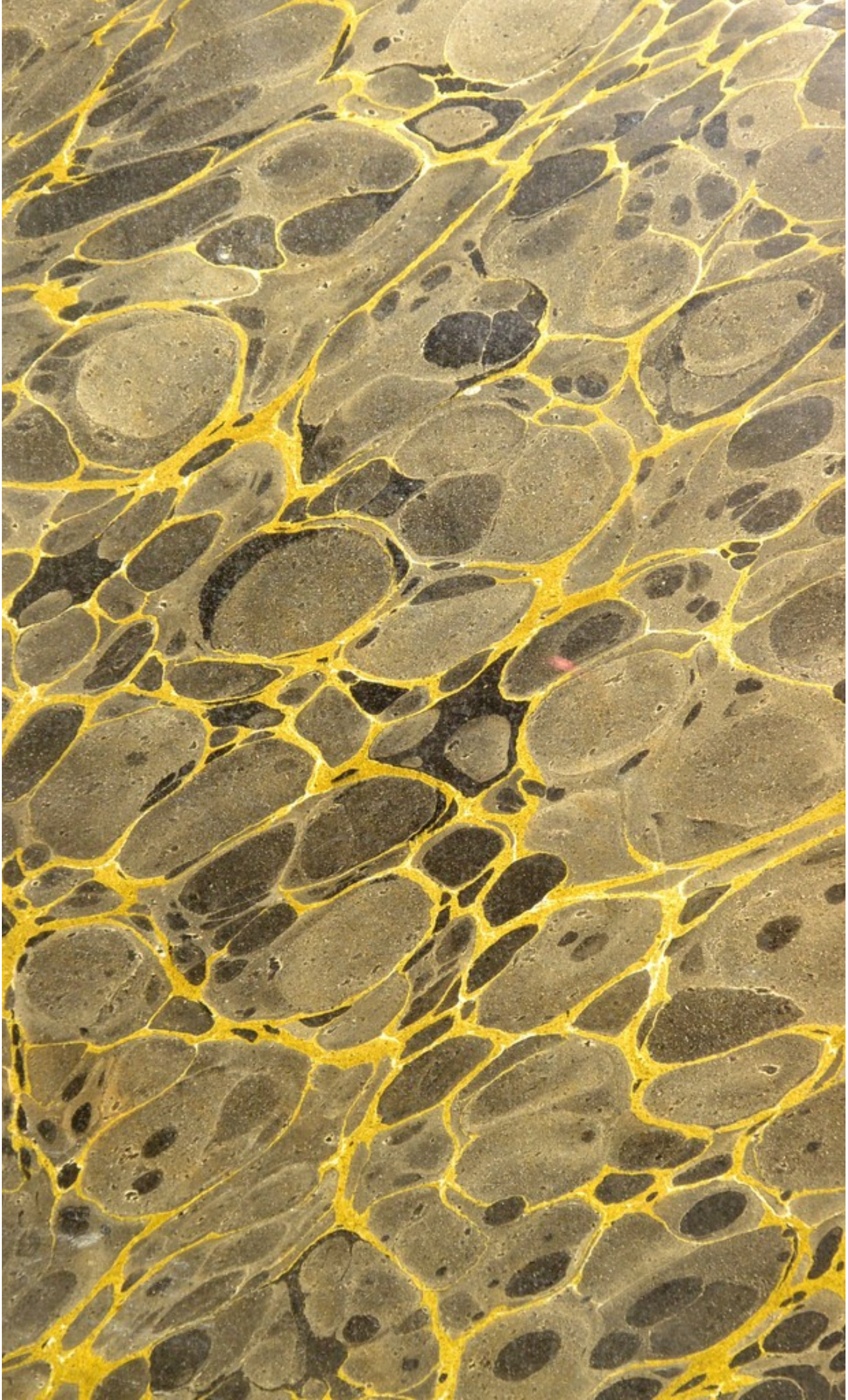
SECTION OF

# DISEASES OF CHILDREN

—

ROBERT WILLIAM PARKER ESQ
HONORARY SECRETARY.





13 Harley St.
April 25. 1881.

My Dear Brother Secretary:
Please to accept the accompanying
Souvenir of the Medical Congress of
1881. May it recal as many pleasant
recollections as I have of our work
together during that memorable
year -

Believe me, my dear Colleague,
most faithfully yours,
William Mac Cormac,
Secretary General.

R. W. Parker Esq

Presented by

The Executive Committee

of the

International Medical Congress

of

1881

To Robert William Parker, Esq.

in grateful recognition of his services as

Honorary Secretary

of the Section

Diseases of Children

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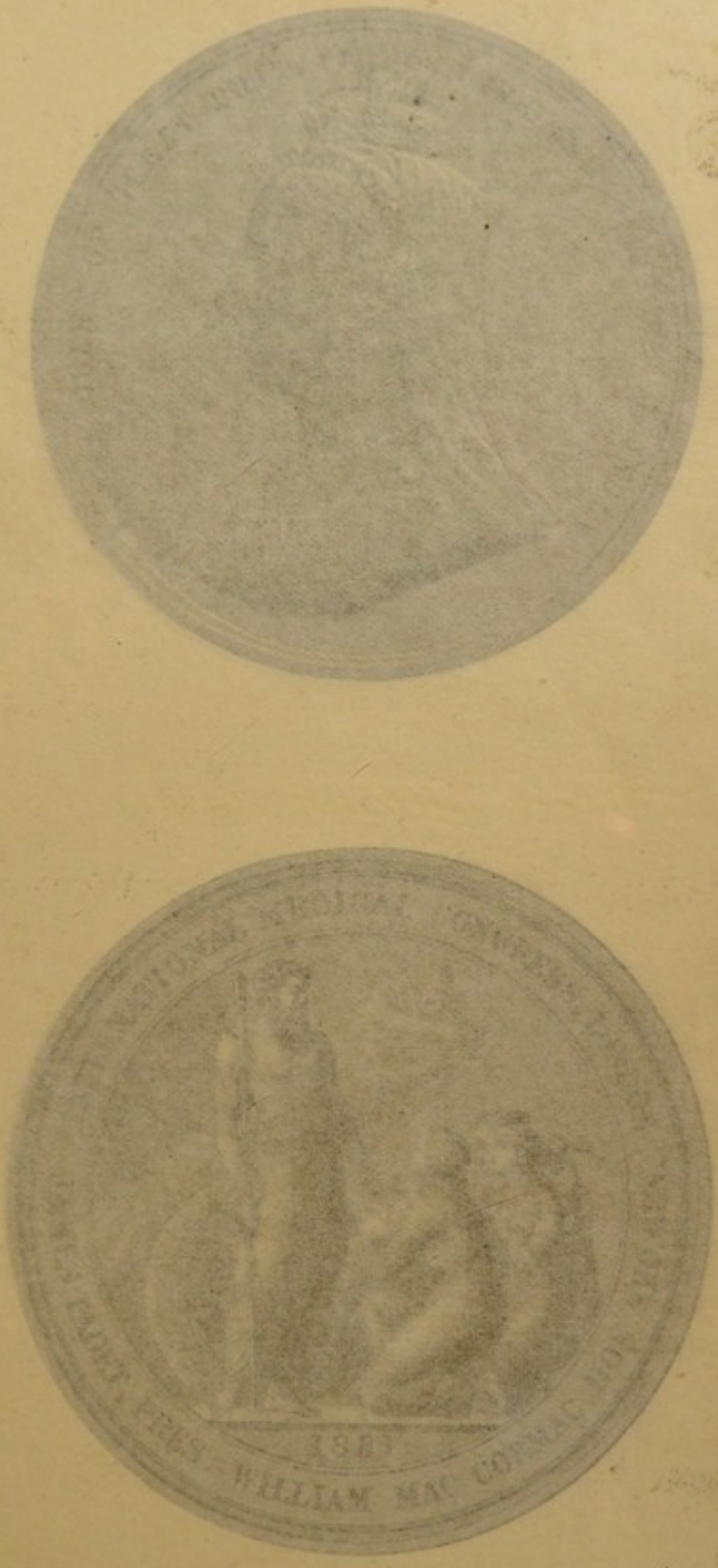


COMMEMORATIVE MEDAL

Presented by

the Executive Committee

of the

# International Medical Congress

of

1881

to

Robert Hilliers Parker Esq

in grateful recognition of his services as

Honorary Secretary

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Diseases of Children

Louis Lewis President of the Congress

Sir Risdon Bennett Chairman of the Executive Committee

A. Pearman Honorary Treasurer

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COMMEMORATIVE MEDAL.

# TRANSACTIONS

OF THE

# INTERNATIONAL MEDICAL CONGRESS

SEVENTH SESSION

HELD IN

London, August 2d to 9th, 1881

Prepared for Publication under the direction of the Executive Committee

BY

SIR WILLIAM MAC CORMAC

*Honorary Secretary-General*

ASSISTED BY

GEORGE HENRY MAKINS, F.R.C.S.

*Under-Secretary*

AND

H. B. DONKIN, M.D. R. W. PARKER, M.D.

*Secretaries of the Section*

## DISEASES OF CHILDREN

LONDON

J. W. KOLCKMANN

1881

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## INAUGURAL ADDRESS

BY THE PRESIDENT,

### Dr. CHARLES WEST.

Gentlemen,—My first duty on taking this chair is a most pleasant one. It is to express my deep sense of the honour done me by my countrymen when they selected me as not unworthy to represent that department of medicine in England which we all assembled here more especially cultivate. The honour, too, was enhanced by the fact that at the time when it was conferred I was on the point of leaving London in search of what I am thankful to say I found—perfect health in a land of constant sunshine. That I have found there, too, a second home, I owe it to the kindness of you, my French friends, who received me so cordially and treated me so graciously. You did not regard me as a stranger, but as a fellow-member of that great *Société Internationale* which has for its object, not the upsetting of thrones nor the changing of Governments in quest of some grand social regeneration, to be accomplished in a few days by violence and bloodshed, but the improvement of mankind by gentle means. The one, like the thunderstorm and the torrent, does but lay waste; the other is like the silent dew, which falls unseen and fertilizes the land. But while I thank you most heartily for all your goodness to me in what I may now call my adopted country, you will, I am sure, find it but natural that I rejoice in returning

once more to my native land; in seeing again the old familiar faces; and revisiting the spots where I studied as a youth or where I laboured as a grown man.

"Cœlum, non animam, mutant,
Qui trans mare currunt."

And my French sympathies are not one jot lessened because I still feel myself altogether an Englishman.

With these words, gentlemen, I should have wished to stop, and to have invited you to pass at once to the business for which we are met. Some three weeks ago, however, I learned to my dismay that the Executive Committee desired that the President of each Section should open its meetings by a short address bearing on its special objects. Far away from my books, moving each day from place to place, I felt my utter inability to do anything worthy of the occasion. Moreover, there came to my recollection an anecdote which did not help to cheer me. Dr. Johnson and his friend Boswell dined one day with a gentleman by special invitation. The next day Dr. Johnson complained to his friend of the meagreness of the entertainment. "Well, sir," said Boswell, "but it was a good dinner." "Yes," replied Johnson, "a good enough dinner, but it was not a dinner to ask a man to." And so, how scanty and commonplace what I say is, pray remember, gentlemen, the entertainment is not one which, had I been left to myself, I should have thought good enough to ask you to.

One accusation which I have heard brought against a meeting like the present is, that it is apt to resolve itself into a mutual admiration society, each member praising what the other has done, all joining to extol what their own generation has accomplished; and that the gratification of personal vanities, not the promotion of science, is the chief outcome of the whole. But just as travellers on a long journey halt from time to time, and looking back on the road they have traversed, take courage to go further, so may we, with no feeling of undue self-gratulation, rejoice over what has been accomplished, even in our own day, as an earnest and as a pledge of further progress, an inducement to more unwearied effort. Thirty years ago, throughout the whole of England and America there was not a single hospital set apart for children. It was but rarely that one saw these little waifs and strays in the wards of our general hospitals; for the maxim "De minimis non curat lex" held good in medicine as in law. Germany, too, was in but little better case, and one was forced to go to Paris to study on a large scale those diseases which men like Guersant, and Blache, and Baron, and Trousseau and Roger, investigated with untiring zeal, and, in spite of the hospital arrangements most painfully defective, strove to cure. We all know how this is altered now. In London there are six separate children's hospitals, each, I believe, with its convalescent branch; and children's wards are to be found in every one of the large London hospitals. There are special children's hospitals in every large town in England; America and Germany have followed the same example; and everywhere throughout Europe the opportunities for the study of children's diseases are almost as numerous as for those of the adult. Nor has this wide field been without abundant husbandmen to till it, and we may count with satisfaction the fruit of their labours. The vague phraseology which served for years to conceal our ignorance, even from ourselves, has been to a great degree done away with. We talk no longer of worm fever, remittent fever, gastric fever, and so on, for under these

various names we recognise the one disease, typhoid fever, varying in severity, but marked always by its own characteristic symptoms. Half a page in a handbook was all that was to be found thirty years ago concerning heart disease in childhood, while at the present time the frequency of heart disease has been fully recognised, and it has been studied with as much care in the child as in the adult. The various inflammations of the respiratory organs are no longer looked on as a whole, but each is referred to its proper class, and we distinguish lobar and lobular pneumonia, bronchitis and capillary bronchitis, and assign to each its proper place and its characteristic symptoms. Nor have our therapeutics lagged behind. I remember the hesitation with which, some forty years ago, my dear friend and master, the late Dr. Latham, decided on tapping the chest of a boy eight years of age, who was received into St. Bartholomew's Hospital on account of a pleurisy which had terminated in empyema; and the delight—the wonderment almost—with which we regarded the successful issue of the operation in a child so young. A few months ago I communicated to the Medical Society of Nice the particulars of fifty cases in my own practice where paracentesis of the chest had been performed at my desire, and several of you gentlemen could relate as many cases or more. That once almost unrecognised disease, diphtheria, has been studied with the greatest care; its relation to membranous croup has been investigated; the close connection of the two has been demonstrated. I for my part should not hesitate to say their absolute identity has been established. Much light has been thrown on various diseases of the nervous system. That once enigmatical affection, the so-called essential paralysis of infancy and childhood, has been shown (in the first instance by the researches of my friend M. H. Roger, and his able coadjutor M. Damaschino) to be due to an acute inflammatory softening of the grey matter of the anterior columns of the spinal cord; and twenty-five recorded observations since that time attest the truth of their discovery. Though, strictly speaking, perhaps not a disease of the nervous system, the pseudo-hypertrophic muscular paralysis of Duchenne claims mention here as a new and important addition to our knowledge of the pathology of early life.

I fear to weary you by further enumeration, else it would not be difficult to increase largely the instances of new and most important knowledge added to our stores since my student-days. In estimating the value of these gains, too, it must not be forgotten that each truth established means an error exploded; so much base metal, so much counterfeit coin withdrawn from circulation; or, to put it differently, so much sterling gold substituted for inconvertible paper money. In this process surgery has, as everywhere, borne a large part. The treatment of hip-disease, the excision of scrofulous joints, the new modes of treatment of spinal curvature—some, indeed, still on their trial—the operation for the cure of genu valgum, which one cannot mention without a fresh tribute of thanks to Joseph Lister, who in this instance has rendered a proceeding safe and salutary from which, but a few years since, the common sense of the surgeon would have recoiled, are so many fresh instances of progress made during a period of little more than the half of my professional life. I take it, however, that the great use of meetings such as the present is to take stock far less of what we know than of what we do not know, or know at best but imperfectly. A few of these problems have been submitted to you in the list of subjects for discussion. To some it is probable that the combined experience of so many and such distinguished men as are here present may furnish definite and conclusive answers. Other questions are introduced in the hope of gaining fresh infor-

mation on points concerning which our knowledge is fragmentary; while there are many other problems still unsolved on which it is hoped that fresh light will be thrown during the time of our meeting here.

And now, with your permission, I will conclude with an old apologue, which tells how, when the fabled Arabian bird renewed each hundred years its vigour and eternal youth, the birds of the air all helped to build its nest. The eagle and the wren contributed alike to this labour of love and duty; each brought what he could, nor ceased till the task was done. And surely science and art, especially our science and art, are old and new, renewing day by day, and burning by a voluntary self-cremation old theories, half facts, hasty conclusions, and substituting more accurate observations, truer inferences, more solid judgments. To this great end we may all do something; but, labour as we may, our task will never be finished, for not once in a hundred years, as the fable runs, but every day and all day long the process goes on—a daily death, a daily renewal, as in our bodies' growth—a death of error, a development of truth.

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## *On the Existence of Two Distinct Forms of Eruptive Fever, usually included under the head of Measles, and the Relation to them of so-called Rubeola or Rötheln.*

Dr. W. B. Cheadle, London.

One of the main points laid down as distinctive of the group of contagious exanthemata, from the time of Cullen downwards, is that each disease occurs in the same individual but once in a lifetime—that is, that one attack of any of these eruptive fevers confers on the individual who suffers it immunity from a second; and the rule has been found to hold good, not, indeed, as absolutely invariable, but with exceptions so few and rare as not seriously to invalidate the general law.

One of the chief members of the group—viz., measles—has been found to respond to the test with striking uniformity whenever an outbreak has occurred under conditions favourable to exact and comprehensive observations; as, for example, in the two well-known epidemics in the Faroe Islands, when all who had had the disease in the first, without a single exception, escaped the second.*

Willan † had never met with a case of measles occurring a second time in the same person, and Thomas ‡ says that a second attack of measles is exceedingly rare, as rare as a second attack of smallpox or scarlatina. I might quote a large number of authorities to show that, although a few exceptions have been recorded, the law holds that one attack of measles affords almost certain pro-

* Dr. Panum, *Archives Générales de Médicine*, April, 1851, quoted by Sir T. Watson.

† Dr. Churchill ("Diseases of Children," p. 723, 3rd edition) sums up the evidence on this point to the following effect :—

Rosenstein never met with a second attack in forty years' practice.

Willan and others give the same as the result of twenty years' experience.

Dr. Baillie shows it may recur.

Dr. Dewees is doubtful, except where the first was rubeola sine catarrho.

Eberle mentions one example.

Rayer, three cases under his own observation.

Guersant and Blache give examples of the recurrence of the disease twice in same year.

Churchill doubtful, but gives one example in his own child.

Genovesi attended forty-six in Santa Cruz who had had the disease before, and Dubosq de la Roberdière prescribed in 1777 for persons he had treated in 1773.

‡ Ziemssen's "Cyclopædia," art. Measles.

tection against a second. My own experience was entirely in accord with this until a short time ago, when my confidence in this comfortable creed received a rude, although happily only a temporary, shock.

An epidemic of measles broke out in the district of London in which I live in December, 1878. Three of my own children caught the disease, and many other cases came under my observation at the time. The disorder exhibited all the symptoms of true measles. It was ushered in with incessant sneezing, profuse running at the eyes and nose, suffusion of the conjunctivæ, slight hoarseness, frequent but not severe cough, some diarrhœa, plentiful and well-marked crescentic eruption coming out on the morning of the fourth day, and a temperature of 102° to 103° or 103·5° during the stage of eruption. In a word, the majority of cases which came under my notice in the epidemic of 1878 were fairly typical examples of measles of more than average severity.

In November, 1879, or not quite a year later, measles broke out in the school which two of my boys attended, and appeared in a form far more serious than that of the previous epidemic. The boys in question were two of the three who had measles in the last outbreak, eleven months before, and I congratulated myself that they at least were safe from attack. But these two supposed protected ones forthwith took the disease, and transmitted it to my three other children, one of whom had also previously had measles in 1878.

Following immediately upon this startling recurrence of the disease in my own family came other instances, showing in an equally forcible manner the complete absence of any protective power exerted by the previous epidemic.

In two other families, numbering seven children in all, six had measles in the first epidemic, eleven months before, at the same time, and contracted from the same source, as my own children; the seventh child had had the complaint a year earlier. These seven children were all exposed in like manner to the contagion of the new epidemic; all took the disease again without exception—not one escaped. Again, three children of another family, under my immediate observation, who had had measles of severe type three years previously, were exposed to the contagion of the second epidemic, and from the same source as those above mentioned. All took the complaint and had it severely.

Thus it came about that, of the first fifteen cases of the second outbreak which came under my notice, no less than thirteen had had measles before, and nine of the thirteen under my own eye within the year.

Altogether I have been able to obtain a trustworthy history of thirty cases. Of these, eighteen came under my own observation at the time; the remaining eleven, although not seen at the time of eruption, occurred in individuals with whom I am acquainted, have been carefully investigated, and are thoroughly reliable. Out of this total of thirty cases of the second epidemic, twenty-two had certainly had measles previously. The protected individuals suffered equally with those who were exposed to the contagion for the first time. They took the disease just as readily; they had it just as severely.

At the outset of this second epidemic the character of the exanthem was one of grave and even dangerous type.* No instance in which the disease proved fatal to an individual previously healthy came under my own observation; but in three cases under my care the condition was for some time critical, and four deaths occurred in children suffering from other diseases at the time. Three were killed directly by the measles during the eruptive stage; the fourth died from the effects of broncho-pneumonia set up by it.†

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* The first twenty cases might be classed as severe; the remaining ten, all occurring at the close of the epidemic, were of milder type, and in some the constitutional disturbance was extremely slight.

† Three cases of empyema (two convalescent) and one of tetany.

The course and symptoms of the disease in this second epidemic of 1879 showed appreciable variations from those of the disorder which prevailed the year before, which might be taken as a typical example of the ordinary form. Yet it may be said, I think, that all the phenomena were such as have been described, at one time or other, as arising in the varying phases of true measles, and the outbreak was generally regarded, without question, as genuine measles of an unusually severe kind. If that were so, then in all cases previous attacks of measles absolutely failed to give the protection which experience proves them to confer, almost without exception, against a second attack of the same disease. It is impossible to accept this solution without question. The suspicion arises that the disease of the second epidemic, although at first sight apparently identical with measles, might in reality be as specifically distinct from it as typhus from typhoid or variola from varicella. Slight differences may indicate a generic distinction, masked by a general superficial agreement in more obvious characteristics, and a careful analysis of the phenomena of the second epidemic shows certain points of difference and some special features which, taken together, lend support to the theory of its specific distinctness.

I.—*The Period of Incubation.*—This was accurately ascertained in two cases where there was a single exposure to infection, limited to a few hours. In one,* the first symptoms showed themselves on the eighth day; the eruption on the eleventh. In the other,† the first signs were observed on the ninth day; the eruption was out on the morning of the twelfth day after exposure.

The period of incubation was ascertained approximately in five other cases. In two,‡ where the exposure lasted three days, this could not have been more than twelve days or less than ten, the period from eruption to eruption being ten days; and in the remaining three§ the period from eruption to eruption was uniformly twelve days.

No opportunity arose for testing the incubation period in the first epidemic, but I have ascertained it in two cases of ordinary measles where there was a single short exposure.‖ The first symptoms were observed on the tenth day; the rash appeared in both instances in the night between the thirteenth and fourteenth days. These conclusions are in exact correspondence with the observations of Panum in the Faroe Island epidemics, and are confirmed by other accurate records, as shown by Thomas in a very complete examination of the subject.¶

The period of incubation in true measles appears to have a duration of extreme constancy—viz., thirteen to fourteen days to the appearance of the eruption, and usually, but much less uniformly, of ten days to the first symptoms; so that in the epidemic we are considering the period of incubation was shorter than the normal one of true measles by from one to two days.

II.—*The Period of Invasion* was also shorter than normal by a day, although, as this appears to be liable to variation in true measles, much stress cannot be laid upon the difference in the eight cases in which this point was noted with exactness. In seven the rash appeared on the third day after the initial

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* L. M., exposed for a few hours, November 22, 1879. First symptoms, November 30. Rash, night of December 2–3.

† ———, child in Great Ormond Street Hospital, exposed January 7. First symptoms, January 16. Rash, night of January 18–19.

‡ W. W. C. and F. M. C., exposed to contagion November 11, 12, and 13. (Rash out in infector, 13th). First symptoms in two infected, 21st. Rash out in two infected, 23rd.

§ Ashwell, Coates, Parkins (empyemas). Eruption in infector, November 10; in infected, November 22.

‖ W. F., visited Brighton for a few hours. Exposed to measles contagion. Sickened tenth day. Rash thirteenth to fourteenth day.

Una B., slept one night with girl with measles. Sickened tenth day. Rash thirteenth to fourteenth day.

¶ Ziemssen's "Cyclopædia," art. Measles.

symptoms;* in the eighth† (one of empyema), not till the sixth day after access of febrile symptoms, but the twelfth after exposure.

III.—*The Catarrhal Symptoms and Affection of the Air Passages* exhibited certain special features. There was injection of the conjunctivæ; usually slight sneezing at the onset, which soon passed off; little running at the eyes and nose, in many cases none; no intestinal flux. On the other hand, the hoarseness and irritation of the throat were extreme, and the cough incessant, laryngeal, croup-like. In one instance this was harassing beyond anything I have ever seen. The patient (a boy of ten years old), could not be kept in bed, but stamped about the room in distress so intolerable that chloroform had to be freely given to relieve it.

In one case the respiration became difficult and stridulous; the tonsils, fauces, and soft palate were dusky red, much swollen, and covered with tenacious mucus. Enlargement of the glands at the angle of the jaw was observed in one or two instances. In one there was a film of coherent membrane on the tonsils, and albumen in the urine.‡ Marked bronchitis, evidenced by sibilant respiration and abundant fine rales at the bases, was present in all but the mildest cases. In two, broncho-pneumonia supervened.

The contrast between the two epidemics in regard to these symptoms was striking. In the first, incessant sneezing, coryza, lachrymation, comparatively slight hoarseness, frequent but not severe cough, some diarrhœa. In the second, little or no sneezing, coryza, or lachrymation, extreme hoarseness, incessant severe crouplike cough, marked implication of the larynx and bronchi, entire absence of catarrh of the intestinal mucous membrane.

IV.—*The Eruption* presented some points of divergence from the normal measles' rash. It was more raised, more coarse and papular; the grouping of the stigmata was not crescentic, but in irregular blotches, and of a darker, more purple hue. In the severest cases it was confluent on the face and backs of the wrists and hands, accompanied by much swelling. In one instance there were petechiæ and small purpuric patches on the extremities; and in this case the livid papular petechial rash and the swollen face and hands bore at first sight a strong resemblance to the condition in severe smallpox.

In two instances of more moderate intensity, but contracted from one of the more virulent cases, the confluent eruption on the face and limbs, in place of being purple and papular, was diffuse and rosy—closely simulating in these points the rash of scarlatina—elsewhere of the patchy, purple, measles form. In all these cases the eruption did not reach its maximum until the third day, the most intense not beginning to decline until sixty hours, or even more, after its first appearance—*i.e.*, at the close of the third or beginning of the fourth day.

V.—*The Eruptive Fever* ran high. The temperature went up to 103° as the rash came out, and continued to rise a little until the eruption reached its height, and ran up to 104° or 104·5° at the maximum. In the majority of instances the temperature began to fall on the fourth day of eruption; but in two, in which there was no complication, it remained at 103° to 104° for two days later, or the fall did not fairly commence until the sixth day.

The pulse ranged from 130° to 160° at the maximum. In one case (A. C.)

* W. and F. C., L. M., L. and M. F. (private cases). Coates, Parkins (Children's Hospital).

† Ashwell (Children's Hospital.) Exposed, November 10. First symptoms, November 17 (rise of temp. to 100·8°, continuing up to eruption).

‡ In an epidemic of measles which prevailed contemporaneously at Folkestone, and which probably was of the same generic form, the affection of the throat was equally prominent. I learn from Mr. Tyson of that town, who has kindly written to me with this information, that in three cases the laryngeal obstruction was so great that tracheotomy had to be performed; in another the operation was threatened, and that other cases of a similar kind had occurred.

without complication it remained 130° to 140° for forty-eight hours after the eruption reached its maximum, and was feeble and irregular.

During this period the tongue was much coated and became dry; there was night delirium; and, as the eruption came out, drowsiness extreme beyond all precedent, lasting for some days after the rash reached its height.

VI.—*Vomiting* was another salient feature present in all the severe cases. It was noted in thirteen, and generally showed this peculiarity:—In some instances it began with the first fever, and recurred at intervals until the eruption declined; but more usually it did not usher in the initial symptoms, as in most eruptive fevers, nor occur later in the stage of invasion *before* the appearance of the eruption, as observed sometimes in true measles, but *afterwards*, during the eruptive stage, and most commonly about the second day. This vomiting was not the result of the violent cough, but quite independent of it—a distinct effect of the measles poison.

VII.—Another symptom, present in all severe cases, with a constancy I have never observed before, was *earache*. It was noted in ten of the cases of which I had personal knowledge, and may have occurred in many more of which I have less complete records. It came on, with the greatest uniformity, about forty-eight hours after the eruption began to decline; lasted with much severity for several hours, and then disappeared. In no case was there any discharge from the external meatus, and in one only any persistent deafness afterwards.

Such were the symptoms observed in severe cases in the earlier part of the outbreak of 1879. Towards its close, the cases which arose were of exceedingly mild type. The period of invasion was extremely short, often not more than twenty-four or thirty-six hours, marked only by slight malaise, catarrh, hoarseness, and cough; little or no sneezing, or flux from the eyes and nose. The temperature rose to 101° or 102° for a short period as the eruption came out, with rapid defervescence. The patients, in marked contrast to those attacked at the outset, were hardly ill.

The special features observed in the epidemic of 1879, added to the evidence afforded by the absence of any protection conferred by previous attacks of common measles, confirm the view that the disease in this outbreak was specifically distinct. And if so, the question arises whether it is a new and previously unrecognised exanthem, or a severe form of so-called German measles, rubeola or rötheln.

At first sight the supposition that it might be rötheln would seem to be negatived by the great severity of the majority of cases.

As far as my researches go, authorities who recognise it at all are almost unanimous in regarding it as a disease of uniformly mild type.

It is said, indeed,* to be specially distinguished from true measles by its slightness and want of character.

Trousseau † estimates it as the mildest of the eruptive fevers, standing in the same relation to measles as chicken-pox to smallpox.

Vogel speaks lightly of it as without fever or catarrh.

And thus throughout the whole list of writers on the subject, ‡ except

* Bristowe's "Theory and Practice of Medicine," p. 153.

† "Clinique Médicale," Sydenham Soc. trans., vol. ii. p. 237.

‡ Wunderlich ("Temperature in Disease") says the elevations of temperature are generally sub-febrile, or at the most moderately febrile; and that although in isolated cases more considerable elevations of temperature may be met with, they depend no doubt either upon complications or on that peculiar mobility of temperature characteristic of very young children.

Thomas (Ziemssen's "Cyclopædia," art. Rubeola) states that in the majority of cases there is no fever, but that the course of temperature is a varying one. There are cases with fever and rapid initial increase, *with defervescence at or before* the disappearance of the eruption.

Aitken,* who states that fatal cases have occurred, and describes post-mortem appearances.

Now these descriptions of the character and symptoms of rötheln apply accurately enough to the slighter cases towards the close of the second epidemic, but to them alone.

On the other hand, there is an obvious resemblance between the severe cases and those described by authors as a malignant form of common measles—the rubeola nigra of Willan, the rubéole boutonneuse and ecchymotic measles noted by Trousseau,† and the similar variety graphically described by our President, Dr. West.‡

So that, in this epidemic in question, we have cases in the first fierceness of its outbreak corresponding closely with those ordinarily classed as abnormally grave and exceptional cases of common measles, and later others of less marked characteristics, directly bred from and lineally descended from the first, corresponding in all respects with those regularly recognised as rubeola or rötheln.§

Not only were the mild cases occurring at the close of the epidemic in West London generally regarded as rötheln, but rötheln was prevalent at the time, shortly before or shortly after, in various parts of the country, as appeared from numerous reports published in the medical journals at this juncture. ‖

Further, the severe cases agree in character with that which one would naturally expect an intensified rötheln to present:—Coryza slight or absent; papular non-crescentic rash, in some cases confluent on the face and extremities, sometimes scarlatiniform there; prominent throat symptoms; absence of intestinal affection; in some cases enlargement of glands at the angle of the jaw; with a shorter period of invasion and incubation than in ordinary measles. *The general features the same as in recognised rötheln, but certain of them increased and exaggerated.*

It seems impossible to avoid the conclusion that the disease in the second epidemic which I have described was that variously known as rubeola, epidemic roseola, or rötheln, which exists not only in the slight and unimportant form generally recognised, but as an eruptive fever of considerable severity, which may assume a dangerous, and even malignant type.

No other hypothesis will, to my mind, satisfactorily explain all the facts which I have adduced.

As rötheln is distinct from true measles, so, I believe, is it distinct from scarlatina. Eight of the cases recorded had, to my own knowledge, previously suffered from scarlatina, and it has been shown repeatedly by others that previous scarlatina possesses no more protective power against rötheln than a previous attack of measles.

No connection has been traced between rötheln on the one hand and scarlatina and measles on the other. The hypothesis that rötheln is a hybrid between the two rests on no better foundation than that it presents certain superficial points of resemblance to each of them.

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* Aitken, "Practice of Medicine," art. Rötheln.

† Trousseau, "Clinical Medicine," Sydenham Soc. trans., vol. ii. p. 216.

‡ "Diseases of Infancy and Childhood," 6th edition, p. 808.

§ L. M., a boy of six, caught the disease by a solitary exposure of three hours to the contagion of two of the most extreme cases under my observation; he had the exanthem severely, and of the same form as those from whom he took it. He communicated the disease to two sisters; in them the eruption was less severe, and had all the features of typical rötheln—*i.e.*, roseolous eruption on the face and extremities, elsewhere measles-like; no catarrh; red, swollen fauces; some tumefaction of the glands under the jaw.

‖ In Manchester, February 1880, Dr. Tomkins, *Brit. Med. Jour.*, May 29, 1880.
In Bradford, end of 1879 and beginning of 1880, Dr. Burnie, ibid., June 5, 1880.
In Bolton, spring of 1880, Dr. Robinson, ibid., June 19, 1880.
In Exeter, spring of 1880, Dr. Wordman, ibid., June 26, 1880.
In Wansford, autumn of 1879, Dr. Brown, ibid., July 3, 1880.

Finally, as the outcome of these observations, I would venture to draw the following conclusions :—

1.—That rötheln is a specific contagious exanthem, distinct from either measles or scarlatina.

2.—That the period of incubation is from eleven to twelve days; the period of invasion from two to three days; but in mild cases this may not be more than twenty-four hours. On these points, however, more extended observations are desirable for their precise estimation.*

3.—That other features, which, not singly, but taken together as a clinical group, may serve to distinguish severe cases of rötheln from severe cases of ordinary measles, with which they are liable to be confounded, are—

The slightness or absence of sneezing and coryza.

The greater severity and frequency of the cough; its hoarseness and laryngeal character.

The more marked catarrh of the larynx and bronchi.†

The absence of intestinal catarrh, as evidenced by absence of diarrhœa.

The more papular character of the eruption; its more purple hue; the absence of any crescentic arrangement; its confluence on the face and extremities, where, in such case, it may be swollen, purple, purpuric, or scarlatiniform.

The higher range of temperature and its longer persistence.

The extreme drowsiness during the eruptive stage.

The occurrence of vomiting when the eruption approaches its maximum.

The occurrence of earache during its decline.

In addition to these more constant symptoms, there may be, in extreme cases, exudation on the fauces, and probably in the larynx, and albumen in the urine.‡

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## *Die wirkliche Stellung der sogenannten Rubeola, Rötheln oder "German measles" und die Verwandtschaft derselben mit Scharlachfieber und Masern.*

Dr. M. Kassowitz, Vienna.

Ich fühle mich in einer gewissen Hinsicht verpflichtet, mein Votum bei dieser von der Section für Kinderkrankheiten des internationalen-medizinischen Congresses aufgestellten These abzugeben, weil ich in einem vor 7 Jahren erschienenen Aufsatze (über einige seltenere Vorkommnisse bei Masern—österreichisches Jahrbuch für Pädiatrick, 1874, S. 79) die Erklärung abgegeben hatte, dass ich mich bis dahin noch nicht selber von der Existenz der Rubeolen als einer selbständigen, von den Masern unabhängigen Krankheitsform habe überzeugen können.

Die Gründe, die mich damals bewogen hatten, mich in dieser Frage so reservirt auszusprechen, waren im Grossen und Ganzen folgende :—

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* The incubation period of rötheln appears to be not clearly ascertained.
Bristowe ("Principles and Practice of Medicine") says about one week.
Aitken does not state it.
Thomas says it is uncertain, but probably two and a half to three weeks.

† Aitken ("Practice of Medicine," art. Rötheln) says the throat is sometimes so swollen that fluids regurgitate through the nose on swallowing, and that in fatal cases death occurs from coma or bronchitis.

‡ In addition to the case noted by me, Dr. Duckworth (*Lancet*, 1880 vol. i.) reports one in which there was transient albuminuria of probably three days.

1.—Ich hatte bis dahin ausser einigen isolirten Fällen, die ganz ausser Zusammenhang mit jeder Epidemie gestanden waren, und ausser einigen in jenem Aufsatze beschriebenen, welche nur mitten in einer heftigen Masern-Epidemie zur Beobachtung kamen, niemals eine Häufung von Erkrankungen, auf welche die von den Autoren gegebene Charakteristik der Rubeolen gepasst hätte, in einer gewissen Zeit, oder in einer Familie, ja nicht einmal bei 2 Mitgliedern einer Familie neben oder nach einander beobachtet. Ich gab damals ausdrücklich zu, dass dies auf einem Zufall beruhen könne; doch sei es immerhin auffallend, dass nicht nur mir selbst in den vielen Jahren, in denen mir das grosse Materiell des Kinderkranken-Instituts zur Verfügung steht, ein solcher Fall nicht vorgekommen sei, sondern darnach in den Berichten der grossen Wiener Kinderspitäler und der Ambulatorien die Diagnose "Rubeola" vollständig vermisst werde.

2.—Hatte ich bei dem Vergleiche der in den letzten Jahren über Rubeolen erschienenen Abhandlungen aus dem Grunde kein ganz bestimmtes Bild von dieser Krankheit gewinnen können, weil die Angaben der Autoren in einigen wichtigen Punkten stark differirten. So hatte, um nur ein Beispiel anzuführen, kurz zuvor Nymann sich dahin geäussert, die Contagiosität sei bei den Rötheln fast Null, und auch Steiner hatte sie für nicht contagiös erklärt, während alle übrigen Autoren dieselben den contagiösen acuten Exanthemen an die Seite stellten.

3.—Erschien mir eines der hauptsächlichsten Argumente der Vertheidiger der Selbständigkeit der Rötheln, nämlich die häufige Beobachtung, dass kürzlich Durchmaserte, an Rötheln Erkrankte, und Individuen, welche Rötheln überstanden haben, später wieder an Masern erkranken, aus dem Grunde nicht vollkommen beweisend, weil ich gerade in der damals von mir erwähnten Masernepidemie mehrere Fälle beobachtet hatte, in denen dasselbe Individium in ganz kurzen Zwischenräumen zweimal an ächten zweifellosen Masern erkrankt war, und ich es daher für umsoweniger auffallend halten konnte, wenn nach einer abgeschwächten Masernform bei demselben Individium nach einiger Zeit eine heftigere Erkrankung oder umgekehrt beobachtet würde.

Weit entfernt jedoch, etwa die Existenz der Rötheln zu leugnen, sagte ich damals wörtlich:—

"Es lasse sich aus allen diesen sehr genauen und offenbar höchst gewissenhaften Beobachtungen und Schilderungen nicht *mehr* mit Bestimmtheit abstrahiren, als dass es eine, den Masern in allen Punkten ähnliche, aber sowol bezüglich der Höhe des Fiebers und der dadurch bedingten Schwere der Allgemeinerkrankung, als bezüglich der Dauer des Fiebers und des Ausschlages, endlich hauptsächlich bezüglich der Intensität des Exanthems ausserordentlich abgeschwächte Krankheitsform giebt, die manchmal isolirt (d. h. ohne sichtbaren Zusammenhang mit anderen ähnlichen Erkrankungen), manchmal in kleinen Epidemien, in denen alle Fälle gleich leicht verlaufen, vorkommt, dass aber auch, wie Gerhardt ausdrücklich sagt, einzelne Angehörige der Epidemie öfter, manchmal auch die ganze Epidemie den Uebergang in ächte Masernformen zeigen.

Ich muss gestehen, dass mich namentlich der letztere Ausspruch Gerhardt's, eines Vertreters der Selbständigkeit der Rubeolen (Lehrbuch der Kinderkrankheiten, 2. Auflage, S. 76), stutzig gemacht, und mir hauptsächlich jene Bedenken eingeflösst hatte, denen ich in der eben citirten Weise Ausdruck gegeben habe; denn wenn sich wirklich solche Uebergänge in einzelnen Fällen und in ganzen Epidemien zeigen würden, dann wäre es ja eigentlich um die Selbständigkeit der Rubeolen geschehen.

Ich bin jedoch nunmehr in der angenehmen Lage, *aus eigener Beobachtung über eine sehr intensive Röthelnepidemie berichten zu können*, welche etwa im

December vorigen Jahres hier in Wien ihren Anfang nahm, und auch jetzt noch (Ende April) in etwas abgeschwächtem Maasse fortdauert, und ich gestehe sofort ein, dass ich nunmehr jene Bedenken zum grössten Teile fallen lassen kann, und zwar hauptsächlich aus dem Grunde, weil ich, trotzdem ich im Ganzen 64 Fälle in 23 Familien beobachtet habe, dennoch *nicht ein einziges Mal einen Uebergang in echte Masernformen* constatiren konnte.

Sämmtliche dieser Beobachtungen stammen aus der Privatpraxis, und ich muss das interessante Faktum constatiren, dass mir trotz der zweifellos sehr bedeutenden Extensität der Röthelepidemie in dem Ambulatorium des Kinderkranken-Institutes, welches jährlich von mehr als 3000 kranken Kindern frequentirt wird, auch nicht ein einziger Fall vorgekommen ist. Ich kann dies nicht anders erklären, als dass die ärmeren Leute wegen der so geringfügigen Veränderung in dem Allgemeinbefinden ihrer Kinder das Exanthem entweder ganz übersehen, oder es nicht für notwendig halten, ärztlichen Rat einzuholen. Ob diese Erklärung auch für das Fehlen der Beobachtungen in den früheren Jahren und in anderen Wiener Krankenanstalten ausreicht, oder ob diese Krankheit früher in Wien überhaupt noch nicht epidemisch aufgetreten ist, muss ich dahingestellt sein lassen.*

Bezüglich der Symptome und des ganzen Verlaufs der Krankheit habe ich den Angaben der neueren Autoren und speciell der monographischen Darstellung von Emminghaus im Gerhardt'schen Handbuche nichts Wesentliches hinzuzufügen. Höchstens könnte ich noch folgenden Daten Raum geben.

Das *Fieber* war in weitaus der grössten Zahl der Fälle sehr geringfügig; doch wurde eine Steigerung auf 37·5 bis 37·8° in der Achselhöle im *Beginne* der Eruption in keinem Falle, wo überhaupt eine Messung vorgenommen wurde, vermisst. In *einem* Falle betrug die Temperatursteigerung 38·6, in einem andern Falle 39·5 in den Abendstunden des ersten Krankheitstages. In allen Fällen war jedoch am zweiten oder am dritten Tage die Entfieberung eine vollständige.

Der *begleitende Catarrh* der Nasenschleimhaut und der Conjunction mit häufigem Niesen und mit ziemlich lästigem Brennen in den Augen fehlte in keinem Falle; dagegen war nur in sehr wenigen Fällen Hustenreiz zu beobachten. All diese Erscheinungen schwanden gewöhnlich am zweiten, spätestens am dritten Krankheitstage vollständig.

In etwa einem Dritteil aller Fälle waren sehr deutliche *Anschwellungen der Lymphdrüsen* am Halse und besonders hinter den Ohren mit ziemlicher Empfindlichkeit dieser Anschwellungen aufgetreten. Die Rückbildung derselben erfolgte häufig erst mehre Tage nach Ablauf aller übrigen Erscheinungen.

Das *Exanthem* war in der Mehrzahl der Fälle ein sehr schwaches, sowol was die Grösse der Flecken, als die Zahl derselben, und endlich auch ihre Verbreitung über die Körperoberfläche anlangte. Am dichtesten standen die Flecken gewöhnlich im Gesicht, welches dann auch häufig am ersten Tage wie gedunsen erschien. Am Stamme waren häufig nur vereinzelte rundliche rosarote Fleckchen zu sehen. In selteneren Fällen war aber der ganze Rumpf und die Extremitäten mit grossen dichtgedrängten, vielfach confluirenden, über das Niveau der Haut ein wenig prominirenden blass-rosaroten Efflorescenzen bedeckt, welche in diesen Fällen allerdings an und für sich betrachtet, und ohne Rücksicht auf den sonstigen Verlauf der Krankheit selbst und die gleichzeitig in derselben Familie beobachteten Fälle, einem Jeden als Masernexanthem mittlerer Intensität erscheinen müssten. In diesen Fällen zeigte sich oft noch 6–8 Tage nach Beginn der Krankheit, also mehre Tage nach eingetretener vollständiger Gene-

* Nach Abschluss dieser Schrift erschienen an einem Tage 2 Rubeolenkranke Kinder im Ambulatorium.

sung, eine deutliche Marmorirung der Haut, insbesondere an den unteren Extremitäten.

Isolirte rote *Flecken an der Schleimhaut des harten Gaumens* beobachtete ich in einer geringen Anzahl von Fällen am ersten Tage der Erkrankung.

Die *Contagiosität* war eine zweifellose und sehr bedeutende, und lässt sich durchaus jener der Masernkrankheit an die Seite stellen. Nur in einer einzigen Familie, in welcher der *Paterfamilias* von aussen her bei einer genau constatirten Begegnung mit einem ambulanten Röthelkranken inficirt worden war, blieben die übrigen Familienglieder, inclusive die Kinder, verschont. In allen übrigen Familien folgte auf die erste Erkrankung nach einer bestimmten Incubationsdauer, welche zwischen 14 und 20 Tagen schwankte, aber innerhalb dieses Spielraums sich viel häufiger der niederen Zahl näherte, *jedesmal* eine Erkrankung eines oder mehrer anderer Familienglieder. Nur ein einziger Fall blieb noch isolirt, wo nämlich ein einziges 4 Monate altes Kind erkrankt war, welches noch niemals die Wohnung verlassen und auch sicher keinen Besuch von einem Rubeolenkranken erhalten hatte. Ich halte es für möglich, dass in diesem Falle *ich selbst* es war, der, ohne selbst erkrankt gewesen zu sein, bei einem früheren Besuche den Krankheitskeim eingeschleppt hatte.

Unter den 64 Fällen betrafen 5 Fälle Erwachsene. Letztere scheinen sich demnach einer gewissen relativen Immunität zu erfreuen. Sehr häufig kam es vor, dass in einer Familie sämmtliche Kinder und nicht eines der erwachsenen Familienglieder erkrankte.

Wenn ich mich nun zu dem zweiten Teile der These, nämlich zu der *Verwandtschaft dieser Krankheitsform mit Scharlach oder Masern* wende, so kann, wenigstens so weit meine eigenen Beobachtungen reichen, und soweit mir die Schilderungen anderer Beobachter bekannt sind, von einer Verwandtschaft mit Scharlach kaum ernsthaft die Rede sein. Es fehlt nicht nur der Charakter des Scharlachexanthems, sondern auch die Neigung zu den, den Scharlach charakterisirenden Erkrankungen der Rachengebilde und der Nieren. Dagegen lässt sich sowol bezüglich des Hautausschlages, als auch insbesondere in Bezug auf die concomitirenden Erscheinungen, eine *sehr bedeutende Aehnlichkeit mit der Masernkrankheit* niemals verkennen; und wenn nun auch der Umstand, dem eine bedeutende Häufung ähnlicher Erkrankungen ohne Uebergang in wirkliche wolcharakterisirte Masern stattgefunden hat, die Annahme einer eigenen Krankheitsspecies gerechtfertigt erscheinen lässt, so wird man andrerseits nicht übersehen dürfen, *dass die Rötheln den Masern viel näher stehen, als jeder anderen uns bekannten Krankheit.* Vielleicht wird sich, wenn man einmal dahin gelangt sein wird, jede dieser beiden Krankheiten auf einen bestimmten Mikroorganismus als Krankheitserreger zurückzuführen, der Schlüssel zu dieser Aehnlichkeit in einer nahen Verwandtschaft dieser beiden Organismen finden lassen. Man wird möglicher Weise constatiren können, dass die eine Species sich allgemach aus der anderen entwickelt und sich endlich so weit von ihrer ursprünglichen Stammform entfernt hat, dass sie nunmehr selbst unter günstigen Bedingungen im menschlichen Körper nicht mehr die ursprüngliche, sondern immer wieder nur die modificirte Krankheitsform hervorbringen kann, und dass die eine Krankheitsform nicht einmal mehr befähigt ist, die Immunität gegen die andere zu gewähren.

Es scheint mir zwischen den Masern und den Rötheln ein ähnliches Verhältniss zu bestehen, wie zwischen *Variola und Varicella.* Auch hier auf der einen Seite eine geradezu frappante Aehnlichkeit in der Form und noch mehr in der Anordnung der Eruptionen auf der Haut und den Schleimhäuten; dabei die concomitirenden Erscheinungen bei der Varicella in hohem Grade abgeschwächt, aber manchmal noch teilweise erkennbar;—auf der andern Seite aber

keine gegenseitige Immunitätswirkung zwischen Variola—Vaccine und zwischen Varicella, und endlich, wenigstens in der überwiegenden Mehrzahl aller Beobachtungen, auch keine Uebertragung der Varicella als Variola auf ein anderes Individium oder umgekehrt. Allein gerade in Bezug auf den letzteren Punkt gebieten mir meine eigenen Erfahrungen eine gewisse Vorsicht. Ich habe nämlich Beobachtungen verzeichnet (*Jahrbuch für Kinderheilkunde*, VI. Band, 1871), in welchen einmal von 2 Geschwistern das eine an Varicella, und nach Verlauf von 12 Tagen das andere ungeimpfte an wolcharakterisirten Variolen erkrankte, und in einem zweiten Falle auf die Varicellenerkrankung eines Kindes nach 14 und 16 Tagen eine zweite Varicellenerkrankung des Bruders und eine Variolenerkrankung der Mutter gefolgt waren. Auch andere Beobachter haben vereinzelte ähnliche Beobachtungen verzeichnet. Dem gegenüber will ich keineswegs in Abrede stellen, dass auch ich in der weitaus überwiegenden Anzahl meiner Beobachtungen aus Varicella wieder nur Varicella, auch bei nicht geimpften Kindern, habe entstehen gesehen, und ich stehe daher nicht an, auch für die Varicella einen gewissen sehr bedeutenden Grad von Selbständigkeit gegenüber der Variola zu zugestehen; aber ich glaube dennoch, dass man die Acten über diese Frage noch nicht schliessen darf. Auch für die Rubeolen kann ich ja nur mit Rücksicht auf die hier mitgeteilte, immerhin doch beschränkte Zahl von Beobachtungen constatiren, dass *ich* eine Uebertragung von Rubeola auf ein anderes Individium in der Form von echten Masern nicht gesehen habe. Dass aber eine solche Uebertragung niemals stattfindet, möchte ich, insbesondere mit Rücksicht auf die oben citirte Aeusserung von Gerhardt noch nicht behaupten.

Es mag also vielleicht für die klinische Seite der Frage von Wert sein, die Trennung ganz absolut hinzustellen; vom Standpunkte des Naturforschers möge man aber den Faden zwischen Rubeola und Morbilli, und zwischen Variola und Varicella vorläufig noch nicht durchschneiden.

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## *Contributions to the Study of Rötheln.*

Dr. J. Lewis Smith, New York.

At the close of the epidemic of rötheln which visited New York in 1873–74, I read before one of the medical societies, and afterwards published, statistics which I had collected, together with such observations as seemed to me useful. The paper then prepared is made the basis of the following communication to the Pædiatric Section of the International Congress, with such emendations and additions as I thought proper to make after having recently witnessed another and very similar epidemic.

The disease known as rötheln has heretofore been rare in America. In the Eastern continent, on the other hand, it appears to have been known for many years, and American physicians frequently designate it German or French measles. Meagre and imperfect descriptions of this malady have appeared in some of the British journals, and cases, quite fully detailed, have been published by British physicians.

Rötheln is not entirely a new disease in this country, though most American physicians never saw a case of it until within the last decade. Cases occurring in and about Boston were described by Dr. Homans, sen., in 1845; and at a later date, namely, in 1853 and 1871, B. E. Cotting, M.D., Harvard, saw cases and described them in papers read before local societies (see *Boston Medical and Surgical Journal*, March 15, 1873). In 1874, Dr. Caleb Green of Homer,

Cortland County, New York, an accurate and intelligent observer, also witnessed an epidemic.

This hitherto rare and interesting malady occurred in New York city as an epidemic in 1873 and 1874, attaining its maximum prevalence in March and April of the latter year, after which it declined, occasional cases occurring throughout May. This, so far as I can learn, was the first occurrence of rötheln in this locality. In a general practice of more than twenty years, extending over a considerable portion of this city, I had previously seen nothing like it; and other older physicians, having a large general practice, have informed me that they consider it an entirely new disease with us. Those who believed that they had occasionally observed isolated cases of it previous to the epidemic probably referred to roseola.

The first case which I met with occurred in the middle of December, 1873, in West Seventy-first Street, in the northern suburbs of this city. A few weeks later cases were so numerous in the more thickly populated sections of New York as to attract the attention of many physicians. It was evident that a disease had appeared with which we were not familiar, and as the eruption occurred in points and small circumscribed patches, it was usually designated by the physician, in want of a more accurate name, epidemic roseola, or was spoken of as a spurious measles. Those physicians who were familiar with foreign medical literature saw the resemblance between these cases and those of rötheln as described by British and Continental writers; but in certain, at least, of the foreign cases the duration of the rash was said to be seven days (Liveing, London *Lancet*, March 14, 1874, and *Medical News and Library*, May, 1874), whereas, in the cases in New York, it commonly disappeared by the fourth day. This discrepancy, however, was not sufficient to invalidate the belief in the identity of the New York disease with the foreign rötheln. It was readily explained by the difference in the seasons in which the cases occurred, for Liveing observed his cases in June and July, and, as we shall see, the greater the external heat the longer the duration of the eruption.

Between the middle of December, 1873, and May 1, 1874, I had observed and treated this malady in eighteen families. Cases occurred in three other families living in the same houses with some of those which I attended, and as they were fully and clearly described to me, so that there could be no doubt as to their nature, I have included them in my statistics. The total number of cases in these twenty-one families was forty-eight. During May, when the epidemic was declining, I saw six additional cases, occurring singly, making a total of fifty-four. Their ages are given in the following table—

| AGE. | CASES. |
|---|---|
| From 8 months to 1 year | 2 |
| „ 1 year to 2 years | 4 |
| „ 2 years to 5 „ | 16 |
| „ 5 „ 10 „ | 23 |
| „ 10 „ 15 „ | 3 |
| „ 15 „ 30 „ | 6 |
| Total number of cases | 54 |

The age of the youngest patient was eight months, and that of the oldest thirty years. Seventy-two per cent. of the total number were between the ages of two and ten years, so that rötheln is pre-eminently a disease of childhood. Individuals in and beyond the middle period of life seem to have nearly an immunity from it. The age of the oldest patient of whom I was informed, in the epidemic of 1872 and 1873, was about forty years.

On March 25, 1873, during my attendance in the New York Foundling Asylum, rötheln appeared in a boy of four years. In the following month about thirty more cases occurred in this institution, all children, while among the large number of female nurses and employees, who were chiefly between the ages of twenty and thirty years, all but three escaped.

From 1874 to 1880 rötheln did not occur in New York, unless now and then an isolated or sporadic case, the nature of which was not recognised, and which was supposed to be roseola. On August 9, 1880, two cases appeared in different wards of the New York Foundling Asylum, when it was remembered that two weeks previously these children had been exposed to a patient in the hospital attached to the institution who had what the physician in attendance supposed at the time to be roseola.

Commencing with these two cases, an epidemic occurred in the Asylum, mild in type, affecting only a few at a time, but extending over several months, until about sixty inmates, chiefly children, were attacked.

Toward the close of 1880, rötheln began to appear in the northern part of the city, in which the Asylum is located, and over which my practice extends. Its maximum prevalence was attained in the latter part of March and April 1881, when it particularly attracted the attention of physicians. A large proportion of the children attending certain public and private schools were attacked. It occurred in seventeen families in my practice, in which there were forty-two cases. Their ages are given in the following table :—

| AGE. | CASES. |
|---|---|
| From 1 to 2 years. . . . . | 3 |
| „ 2 „ 5 „ . . . . . | 8 |
| „ 5 „ 10 „ . . . . . | 18 |
| „ 10 „ 15 „ . . . . . | 11 |
| There were 2 cases over 15 years, aged respectively 22 and 42 years . . . . . | 2 |
| Total number of cases . . . . | 42 |

*Premonitory Stage.*—Premonitory symptoms are, in most instances, absent, or so mild as to attract but little attention. It not infrequently happened in the New York epidemics that the parents or the teachers in the schools were first made aware of the illness of the children by observing the eruption. In some instances children were sent from school, not because they felt too ill to remain, but on account of the unusual appearance of the skin. Sometimes, however, in those old enough to express their sensations, a premonitory stage of some hours or a day, or even of longer duration, was present, consisting of such symptoms as usually occur when one has taken a severe cold, as languor, pain in the head, trunk, or limbs. The resident physician of the New York Foundling Asylum was so ill with rötheln that he was confined to his bed during the first day of the disease. Now and then patients experience nausea previous to the eruption, and in the first and second days of the eruptive stage. In only one instance did I observe grave prodromic symptoms. A boy aged eight years was suddenly seized with clonic convulsions; and while in a warm bath for the relief of these, the rash appeared upon those parts of his body which were immersed in the water.

*Symptoms.—Tegumentary System.—(a.) The Skin.*—The eruption commonly commences upon the forehead, around the ears, and along the neck, as in measles. Occasionally it may appear first upon the back or chest, as in the above-mentioned case, in which the hot water accelerated its appearance. Commencing above, the efflorescence travels downward, appearing after some hours

upon the lower part of the trunk and on the legs, resembling in this respect that of scarlet fever and measles. It occurs on all parts of the integument except the scalp and palmar and plantar surfaces. In the majority of the cases which I have seen it gradually faded away, disappearing by the fourth day; but on children who were kept warm in bed or in warm apartments it remained longer than on others. In many instances traces of the rash were still visible several days after recovery, when the patients were heated by exercise or excitement. It reappeared at times, though indistinctly, on a girl of thirteen years for three weeks. In most of the cases in the New York epidemics, the eruption commonly occurred in points and circular spots somewhat smaller than those of measles. These points and spots were numerous and thickly set, covering at times at least half, often more, of the surface, while between them the skin presented nearly or quite its normal appearance. The general aspect in most cases was more like that of measles than that of scarlatina; but in exceptional instances the skin between the points and spots had a redness similar to that of erythema, and the resemblance was very like the scarlatinous efflorescence. Thus, in a boy of three years, the eruption so closely resembled the scarlatinous over the trunk, that, were it not that the temperature was constantly below 100°, and all febrile movement had ceased within three or four days, I would probably have considered the malady a mild scarlatina. In certain patients the eruption, beginning in circumscribed spots like that of measles, becomes in two or three days confluent, so as to resemble that of scarlatina, while over other parts the spots remain discrete. This was the character of the eruption upon the third and fourth days on the extremities of a little boy in the Foundling Asylum. The rash is attended by considerable itching, from which, indeed, many patients suffer more than from all other symptoms. The eruption disappears on pressure, produces a slight roughness of the surface, as ascertained by passing the fingers gently over it, and usually fades away without desquamation. Exceptionally there is a slight branny exfoliation, and in one of my patients this was as considerable over the abdomen as in cases of scarlatina.

(*b.*) *The Mucous Membrane.*—In connection with the cutaneous eruption, a mild inflammation also occurs upon the mucous membrane covering the fauces, buccal cavity, and nostrils, and upon reflections of this membrane over the eyes and eyelids—*i.e.*, upon the conjunctivæ. In certain patients this inflammation is scarcely appreciable, but in the majority it arrests attention at once. It produces a suffused, reddish, or weak appearance of the eyes, with a moderately increased lachrymation. On everting the eyelids, the palpebral conjunctivæ are seen to be injected. In certain patients a moderate puriform secretion collects at the inner angle of the eyelids. In occasional cases the conjunctivitis causes œdema of the lids, usually slight, and likely to be overlooked by the physician; but in three instances, which I now recall to mind, the mothers of the children directed my attention to the swollen state of the lids. In one of these, an infant of twenty-three months, the tumefaction was so great, commencing about the time the eruption began to fade, that light was totally excluded from the eyes, and it was impossible to ascertain their condition. The skin over the eyelids retained nearly its normal appearance, and a puriform secretion appeared between the lids. In three or four days the œdema of the lids and the hyperæmia of the conjunctivæ rapidly declined. The coryza is, in most cases, sufficient to cause an unpleasant sensation in the nostrils and provoke sneezing; but the flow from the nostrils, though present, was, in no instance under my observation, as abundant as in ordinary cases of scarlatina, or even of measles. The fauces present an injected appearance, and in severe cases there is moderate swelling of the tonsils. The same catarrhal hyperæmia is also seen in spots or patches, more or less diffused upon the buccal surface. Both the faucial and buccal

catarrh are less in degree, however, than in cases of measles and scarlatina, having an equal intensity of cutaneous eruption; and this fact has aided me in differential diagnosis.

*The Respiratory System.*—In both the epidemics which I have witnessed, the mucous membrane of the larynx, trachea, and bronchial tubes participated only slightly in the inflammation, which involved the nasal, buccal, and faucial surfaces. Many of my patients had no cough, but others had a mild cough lasting for a few days, but with normal respiration. It was, apparently, due to a very mild catarrh of the respiratory tract at the time when the nasal and conjunctival surfaces were the most affected. It subsided in a few days without treatment. In no case do I recollect that there was any hoarseness.

*The Digestive System.*—The tongue in rötheln is moist and of normal appearance, or covered by a slight fur. The appetite may be impaired, but is not wanting in uncomplicated cases. The parents sometimes say that it is nearly the same as in health. The thirst is slight and the bowels are regular. Nausea is not infrequent, and vomiting was, in several cases in my practice, one of the initial symptoms. In certain patients it also occurred on the first or second day of the eruption. In others there was no nausea, as far as I could learn, either immediately before or during the prevalence of the disease. This system is less frequent in rötheln than in scarlet fever, but is as common, apparently, as in measles. I have never found albumen in the urine, though I have examined that passed by several patients. This secretion did not appear to be abnormal except that it contained urates, so common in febrile states.

*The Pulse and Temperature.*—The largest number of accurate daily observations relating to the temperature was, I think, that of Dr. Reid in the New York Foundling Asylum during the month of March, 1874. He has kindly furnished me with his statistics relating to this symptom as follows:—"The number of closely observed cases in which the temperature was taken was twenty-four. In seventeen of the cases the temperature ranged from 97° to 99°; in six it reached 100°, 100¼°, and 100¾°; in one it reached 103¼° on the second day of the eruption, but remained so elevated only one day." In certain patients Dr. Reid observed what he designates "a tendency to the development of an ephemeral fever." These observations correspond closely with those made by myself during the same epidemic. Thus, in sixteen cases, I found the axillary temperature, taken each day, to be constantly between 98° and 100°, with a pulse under 110°, except in one case, in which it numbered 124°. In certain other patients a more decided febrile movement, lasting from one to two or three days, occurred, usually in the commencement of the malady. Thus, a girl, aged three and a half years, had a temperature of 101¾° and a pulse of 128. In another instance the pulse was 124 and the temperature 102. In another, a girl of three and a half years, there was active febrile movement, occurring without apparent cause, on Saturday night, but abating on the following day. She seemed well until the following Tuesday, when the febrile movement returned and the eruption appeared. On Thursday the temperature from 102° to 103° fell to 99½°, and within a day or two she was convalescent. In two other patients, from two to four days after the disappearance of the eruption, an accession of fever occurred, lasting about one day, and attended by pain and distress in the epigastric region, but without vomiting or diarrhœa. In one of these the temperature was 103¼°, and the pulse 130 per minute. In the other case the temperature and pulse did not seem to be under these figures, but were not accurately ascertained. Occasionally the febrile movement is due more to complications than to the primary disease. Thus, in two of my patients the febrile movement was mainly attributable to diphtheritic inflammation, which had attacked the fauces. But while the febrile movement in rötheln is

ordinarily of short duration, in certain patients temporary exacerbations may occur, in which the temperature is as high as in scarlet fever or measles.

*Complications, Prognosis.*—The only complication which occurred in cases in my practice has already been alluded to, namely, diphtheria, which, when prevalent, is apt to attack surfaces already inflamed. In the Foundling Asylum, varicella complicated one case, and pneumonia another; and in a third, pneumonia occurred about three days after the disappearance of the eruption. The prognosis in uncomplicated rötheln is always very favourable, and there is no more liability to sequelæ than in mild catarrhal inflammations of a non-specific character. The duration of rötheln is short, not ordinarily extending beyond three to five days.

*Nature, Incubative Period, Contagiousness.*—Is rötheln a distinct malady or one with which we are familiar, but the form and character of which are modified by unusual meteorological conditions? Is it roseola, assuming at certain periods an epidemic character, and appearing to be contagious? Or is it at all times infectious, possessing a specific principle, and, like other infectious diseases, self-propagating? Should it, in nosological classification, be placed among the non-contagious and local, or among the constitutional and infectious maladies? Let us consider the facts observed in the New York epidemics.

The first cases of rötheln in this city were often designated roseola by the physicians called to treat them, since they seemed to resemble more closely this disease than any other with which they were familiar. Rötheln, however, differs widely from the peculiar form of dermatitis known as roseola. The successive occurrence of the eruption over the upper and then the lower parts of the body, but covering the whole surface, and the definite duration of from three to five days, are points of difference. Moreover, roseola would not, without so great a change in its character as to become virtually a distinct disease, occur in the cool months without any appreciable dietetic cause as an epidemic over a certain area, and for a limited time, affecting whole households of children and sparing other households as well as individuals of a certain age. We therefore consider it distinct from roseola.

Most of the cases in the New York epidemics bore considerable resemblance to measles, both as regards the appearance and duration of the eruption and the catarrh of the mucous surfaces. Parents often diagnosticated measles before the arrival of the physician, and the physician himself, at first glance, sometimes made the same diagnosis. But in rötheln, the shortness and mildness of the stage of invasion, the absence of cough, or the presence of one trivial and scarcely noticed, appetite good or but slightly impaired; in fine, symptoms that are transient or slight, afford a striking contrast with the graver symptoms of measles. But the decisive proof that rötheln is not a modified measles is found in the fact that one does not prevent the other. Of the forty-eight cases observed by myself prior to May 1 in the epidemic of 1874, nineteen at least had had measles, and one who had rötheln took measles subsequently. I have already stated that in the New York Foundling Asylum rötheln, in 1873 and 1874, closely followed an epidemic of measles. A considerable number of the children attacked by the former disease had recently recovered from the latter. During the epidemic of 1880 and 1881 the same fact was observed, namely, that a previous attack of measles as well as scarlet fever afforded no protection from rötheln. Dr. Chadbourne, the resident physician, writes of the cases in the Foundling Asylum in 1880 and 1881:—"Eight children had rötheln who had had both scarlet fever and measles within six months under my observation, while certain others had had these diseases at some previous time." Of the cases observed by myself in family practice in the same epidemic, it is

stated in my notes that ten had had measles. These statistics are sufficient to show that rötheln is a distinct disease from measles, however close the kinship.

That rötheln is not a form of scarlet fever is evident from the fact that, as regards at least the New York epidemics, the rash was in most instances quite different from the scarlatinous efflorescence, occurring, as we have seen, in small more or less circular points and patches. Moreover, as we have remarked above, there is in rötheln a slight febrile movement and general mildness of symptoms which contrast with the high fever and other pronounced symptoms of scarlatina, or if there be considerable febrile movement its duration is brief. But the conclusive proof of an essential difference between these two diseases is found in the fact already stated in reference to measles, that the attack of the one malady does not prevent the occurrence of the other. There are, it is true, cases in which it is difficult at first to make the differential diagnosis between rötheln and mild measles or mild scarlet fever, but when the course of the malady has been closely observed for three or four days, it will rarely happen, I think, that we will be unable to make out its character.

Those cases of an epidemic which arise when the causes or conditions from which it is developed are most strongly operative, and which at this time are apt to be typical, obviously afford the best data for studying its nature. Such were the forty-eight cases which I saw in the epidemic of 1873 and 1874, and the forty-two in that of 1880 and 1881. As regards the former epidemic, in thirteen of the twenty-one families embraced in my statistics, the first cases were children who up to the time of the seizure were attending public or private schools, and in certain instances those who were nearly simultaneously attacked, living perhaps in streets widely separated, were attending the same school. During the epidemic of 1880 and 1881 the first patients in thirteen of the eighteen families in which rötheln occurred were school children between the ages of six and twelve years, and in most, if not all, the different schools which they attended, rötheln was at the time prevailing as an epidemic, as I ascertained on inquiry. It therefore seemed probable that these had contracted it from others.

In both the New York epidemics during the time that rötheln was at its maximum prevalence, in most of the families containing two or more children the cases were multiple, not occurring simultaneously, but in succession, as if the malady were contracted from those first affected. This is what we daily witness in the spread of exanthematic fevers. Thus in Mr. E.'s family, a girl attending one of the public schools took rötheln in the middle of December, 1873; the two remaining children sickened with it one week and two weeks later. A niece visiting in the family at the time when the first child was sick, but returning home to another street, also had the eruption on December 27th. Alice R., aged ten years, a frequent visitor at Mr. E.'s, living in the same street, and several times exposed to his children during their illness, also took rötheln about January 4th. West Seventy-first Street, where these cases occurred, is thinly settled and suburban, and I could learn of no other cases in the vicinity. A child of Mr. P., aged five and a half years, had been in the habit of playing with two children two doors away who became affected with rötheln in the beginning of April 1881. On April 14th he was supposed to have a mild coryza from taking cold, as he sneezed often, but in a few hours the efflorescence appeared. Four days subsequently, on the 18th, an infant was affected in the same way, and thirteen days later another child in the family, aged twelve years. In a similar manner rötheln occurred in the families of two brothers, living in adjoining houses in West Fifty-first Street. The first patient was a boy of twelve years. It appeared successively in the children of these two families until ten had been affected. In a family in West Forty-sixth Street,

the first case was a boy attending a school in which rötheln was prevalent. Within twenty days, namely, between March 31st and April 20th, four other children were attacked in succession.

These facts and cases seem to demonstrate the contagiousness of rötheln, at least during the time in which the conditions are most favourable for its development, or during the time in which the epidemic influence is most pronounced. In the declining period of both the New York epidemics, the cases which I observed occurred for the most part singly, although there was no attempt to isolate the patients; so the contagious character, if present, must have been very slight.

Rötheln is, in my opinion, an exanthematic fever, mildly contagious. It resembles varicella in general mildness of symptoms, in the absence of dangerous complications or sequelæ, and in the uniformly favourable prognosis, while its symptoms and history show a resemblance to measles and scarlet fever.

If the above view be correct, rötheln must possess an incubative period, which, in the cases observed in both epidemics, apparently varied between seven, or perhaps less than seven, and twenty-one days. Its incubation, therefore, resembles that of scarlet fever, which, as is well known, varies in different patients. In the cases which came under my notice the incubative period, when it could be accurately ascertained, was more frequently about two weeks than a longer or shorter period. The resident physician of the New York Foundling Asylum, when the epidemic was prevailing in that institution, returned to his home in the State of Maine, to a locality where rötheln was unknown. Fourteen days from the date of his departure he was himself affected with the disease in its typical form. No other case occurred at his home, where probably the atmospheric conditions were unfavourable. Minnie B., attending a school in which there were many cases, had the rash on April 5th. On the 23rd of the same month, eighteen days afterwards, it appeared upon the servant who was frequently in Minnie's room. Elizabeth C., attending a school in which rötheln was prevailing, had the eruption on April 17th. It commenced upon her sister thirteen days, and her mother fourteen days subsequently. Other cases might be cited of an apparently shorter as well as longer incubative period. The following note from Dr. Chadbourne, of the New York Foundling Asylum, bearing upon this subject, is interesting:—"I am led to believe, from my observations, that the period of incubation was in the majority of the cases from twelve to fifteen days. The disease has been very feebly contagious. In some cases one child would have rötheln, while the other, nursed by the same woman, would escape. In two instances nursing women had the disease, and though each suckled two infants, the latter escaped."

Rötheln requires no treatment.

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## *The Real Position of the so-called Rubeola, Rötheln, or German Measles, and its Relation to Scarlatina and Measles, as Illustrated in the History of Thirty-one Observed Cases.*

Dr. G. E. Shuttleworth, Lancaster.

Having, in my position as medical superintendent of a large institution for imbecile children, had the opportunity of closely observing over thirty cases of the so-called German measles, and of following the history of the patients,

both previous and subsequent to the attack, I venture to offer my experience as a slight contribution to the discussion of the subject.

My first series of twenty-seven cases occurred in the summer of 1874, and was briefly described by me at the British Medical Association meeting in the following year. My second series of four cases occurred during 1880. I trust I need not apologize for reverting to what, as regards the first series, may be designated old material, inasmuch as the value of my observations in 1874, in connection with the present discussion, is much enhanced by the knowledge of the subsequent history of the patients.

To proceed then to a sketch of the leading features of the epidemic of 1874. I may say, in the first place, that the Royal Albert Asylum (which then contained over two hundred children) had, from its opening in 1870, been up to that date entirely free from epidemic disease. The first case of rötheln which came under observation arose in the person of a nurse, who on the evening of June 15th (a very hot day) consulted me with regard to a rash which had just appeared on the neck and face, consisting of a few brownish-red elevated papules isolated from each other by clear skin. The fauces were slightly reddened. The temperature at 9 P.M. was barely 99°, and the pulse but 84. There was no constitutional disturbance, but as a matter of precaution the girl was at once isolated. An aperient was given. Next morning at 9 A.M., the temperature had risen to 101·8°, and the pulse to 116; there was slight swelling of the tonsils, and a raw appearance of the tongue at tip and edges. The constitutional disturbance was still trivial, and as the menstrual period was approaching, it was thought that possibly the symptoms might have some connection therewith. The rash, still of the same hue and character, had extended over the body and legs. On the morning of the third day the temperature had fallen to 99°, the pulse to 84, and the rash was fading from the face and neck. On the fourth day the pulse and the temperature were normal and the rash had well-nigh disappeared. The nurse was kept isolated for ten days and then allowed to visit her friends in the country for a week, returning to her duties at the institution on July 3rd, it having been previously ascertained that she was entirely free from desquamation. On the 7th July, four days subsequent to the nurse's return, an imbecile girl of twelve (one of the nurse's charge) displayed a rash and other symptoms in all respects resembling those described in the first case, except that the initial pulse and temperature were higher (103° and 120° respectively), falling next day to 100·5° and 112°, on the third day to 99·5° and 108°, and on the fourth becoming normal. In this case there was more noticeable injection of the fauces and swelling of the tonsils, and there was something of the "strawberry" character of the tongue. The rash in colour and arrangement was more like that of measles than of scarlatina; the throat symptoms and tongue more like those of the latter. On the 8th and 9th July, the days succeeding that on which the girl's rash appeared, two fresh cases occurred, these patients belonging also to the class of which the nurse was in charge. In both the rash was slight, and in the second case there was scarcely any redness of fauces. Isolation was all along resorted to, and from the 9th to the 18th July there were no fresh cases. On the 18th a boy, from a different department, but attending school with the girls, was attacked; and the next nine days furnished seven fresh cases. These were all extremely mild, the temperature not rising above 100°, and the rash not lasting for more than three days. Eight days elapsed and three more children were affected; and after two more similar intervals the total of twenty-seven was made up, the last case occurring on August 31st. The last two cases were notable as displaying certain marked differences in the characters of the rash, which had indeed to a less degree been observed throughout the series. Case 26 presented a diffuse and bright rash resembling that of

scarlatina, but there were no throat symptoms beyond redness of the velum palati, and the maximum temperature (that of the first day) was 100°. In Case 27, on the other hand, the rash, both in colour and arrangement, simulated measles, and there was considerable conjunctival injection, though but little lachrymation. In this case the initial temperature, which was the maximum, never exceeded 100°, and it fell to 99° on the third day.

I trust that the brief description just given, together with the table which I present, will suffice to give a general idea of the epidemic of 1874. I may add that the diagnosis of the cases caused me no little anxiety, the more so as medical men who saw only individual cases were inclined in some to diagnose measles, and in others scarlatina. Looking, however, at the whole series, I could not accept these diagnoses, and was greatly relieved to find that the descriptions given by Babington, Squire, Liveing, Tilbury Fox, Vogel, Von Ziemssen, and others of rötheln tallied with what I had observed. My diagnosis was confirmed by the absence in every case of any serious sequelæ; there was no albuminuria; and desquamation, when it occurred in two cases, was of the slightest furfuraceous character. I may perhaps be allowed to quote from the brief notice published in the *British Medical Journal*,* in August, 1875, my conclusions as to the pathology of the affection. "There was no doubt that the twenty-seven cases bore a homogeneous relation to each other; they were not mere summer roseola, for the weather was even cold at one period of the epidemic. They were not cases of *scarlatina*, for in no case were there the full characteristic signs or sequelæ of this disease; they were not measles simply, for there was an almost universal absence of catarrhal symptoms, and more than half the patients had previously had measles; and there was no reason to suspect that the rash depended upon any error of diet or hygiene. It was argued (both from negative and positive characteristics) that the cases were best described under the name of *rubeola notha;* and it was urged that the diagnosis of the affection, as entirely distinct on the one hand from scarlatina, and on the other from measles, might often be a matter of great moment to the practitioner."

It is noteworthy that of the twenty-seven patients in this series, fifteen were recorded to have had measles and four also scarlatina. The sequel enables me to supplement this statement by more recent experience with the same patients.

In 1877 the Asylum was visited by an epidemic of scarlatina, extending to fifty-six cases, and in the early part of 1880 by measles, extending to seventy-seven cases, the last occurring on March 23rd. In the summer of the same year occurred a series of six cases of scarlatina, the first occurring on the 25th May, the last in July.

Whilst these cases of scarlatina, of the nature of which there could be no doubt, were going on, four cases of anomalous rash occurred. On the 23rd June, a boy (D. B.), from an entirely different department from that in which the scarlatina had arisen, displayed a papular eruption on the face and chest, somewhat resembling measles, but the papules were discrete and not arranged in crescentic patches. In the morning (at 8 A.M.), when these symptoms were observed, the temperature and pulse-rate were normal; there was very slight conjunctival injection, and no coryza. In the course of the day the rash extended all over the body, and its resemblance to that of measles became greater. The temperature at 7 P.M. was 100°; at midnight 101·5°. There was little constitutional disturbance, and the appetite was as good as usual. On the second morning the temperature was 100°, the rash still out; on the third, the rash had entirely disappeared, and the temperature was nearly normal. This boy was reported to have had both scarlatina and measles in infancy. On the

* *British Medical Journal*, No. 764, p. 229.

# CASES OF "RUBEOLA NOTHA," OBSERVED AT ROYAL ALBERT ASYLUM,

| No. | Initials of Patient and Sex. | Age. | Date of Appearance and Duration of Rash. | CHARACTER OF RASH. | State of Fauces and Tongue. | Range of Temperature and Pulse. Days of Rash. I. | II. | III. | IV. | V. |
|---|---|---|---|---|---|---|---|---|---|---|
| | SERIES I. | | 1874. | | | | | | | |
| 1 | I. A. ... (F) | 20 | June 15 to 18 | Brownish-red elevated discrete papules; first on neck and face, then over body generally | Fauces and Tonsils slightly red and swollen. Tongue raw at tip and edges | T 99<br>P 84 | 101·8<br>116 | 99<br>88 | 99·1<br>84 | 98·4<br>84 |
| 2 | M. W. ... (F) | 12 | July 7 to 10 | Resembling above ... ... | Similar to above. Tongue somewhat "strawberry-looking" | T 103<br>P 120 | 100·5<br>112 | 99·5<br>108 | 98·4<br>100 | ... |
| 3 | A. B. ... (F) | 17 | July 8 to 11 | Very slight rash on face. Some injection of conjunctiva. Diffuse redness of buttocks: elsewhere rash discrete | One Tonsil enlarged. Tongue "strawberry-like" | T 99<br>P 84 | 98·5 | 98 | ... | ... |
| 4 | A. F. ... (M) | 10 | July 9 to 10 | Very slight rash on chest and face: more about buttocks | Scarcely any redness | Norm. | Norm. | Norm. | ... | ... |
| 5 | J. T. S. (M) | 9 | July 18 to 20 | Slight intermittent papulated rash | Slight redness | T 99<br>P 96 | 98·4 | ... | ... | ... |
| 6 | W. J. ... (M) | 10 | July 21 to 24 | Considerable redness of face, somewhat diffuse, but rash discrete on body. Slight conjunctival injection | Some redness and enlargement of Tonsils | T 99<br>P 96 | 99<br>96 | 98·4<br>84 | ... | ... |
| 7 | J. T. ... (M) | 8 | July 22 to 24 | Slight discrete rash ... ... | Slight redness | Norm. | Norm. | Norm. | ... | ... |
| 8 | J. D. ... (F) | 16 | July 23 to 27 | Brownish-red papular discrete rash all over body | Some redness | T 99<br>P 96 | 99<br>96 | 98·6<br>96 | 98·4<br>84 | ... |
| 9 | H. G. ... (M) | 13 | July 23 to 26 | Very slight rash. Injected conjunctiva | Scarcely any redness | T 100<br>P 96 | 99<br>96 | 99·5<br>96 | 99<br>90 | 99·5 |
| 10 | J. B. F. (M) | 11 | July 23 to 25 | Very slight rash on face and chest | Very slight redness | Norm. | Norm. | Norm. | ... | ... |
| 11 | H. W. ... (M) | 10 | July 23 to 25 | Do. do. | Do. | T 99 | Norm. | Norm. | ... | ... |
| 12 | F. R. ... (M) | 15 | July 23 to 25 | Do. do. | Do. | T 99 | Norm. | Norm. | ... | ... |
| 13 | J. T. ... (M) | 6 | July 24 to 26 | Do. do. | Do. | T 98·9 | Norm. | Norm. | ... | ... |
| 14 | J. F. ... (M) | 15 | July 24 to 26 | Do. do. | Do. | Norm. | Norm. | Norm. | Norm. | Norm. |
| 15 | G. B. ... (M) | 8 | July 25 to 27 | Do. do. | Do. | Norm. | Norm. | Norm. | ... | ... |
| 16 | F. W. ... (M) | 10 | July 25 to 27 | Do. do. | Do. | T 99 | Norm. | Norm. | Norm. | ... |
| 17 | C. B. ... (M) | 11 | July 26 to 29 | Do. do. | Do. | T 99·5 | Norm. | Norm. | Norm. | ... |
| 18 | S. L. ... (F) | 9 | July 26 to 29 | Slight rash: most on face ... | Red Uvula | T 98·6 | Norm. | Norm. | ... | ... |
| 19 | J. P. ... (M) | 12 | July 27 to 29 | Very slight rash on face and chest | Very slight redness | T 98·6 | Norm. | Norm. | ... | ... |
| 20 | J. S. ... (M) | 10 | Aug. 5 to 7 | Diffuse red rash on face. Papules on body | Slight redness | T 100·5<br>P 100 | 99·5<br>96 | 98·4<br>96 | ...<br>84 | ... |
| 21 | E. P. ... (M) | 8 | Aug. 9 to 12 | Like No. 6, above described | Very slight redness | T 99·5<br>P 90 | 98·4 | ... | ... | ... |
| 22 | R. R. ... (F) | 7 | Aug. 9 to 12 | Slight rash ... ... ... | No affection | Norm. | Norm. | Norm. | Norm. | Norm. |
| 23 | L. D. ... (F) | 6 | Aug. 16 to 19 | Slight rash ... ... ... | No affection | T 99 | Norm. | Norm. | ... | ... |
| 24 | I. B. ... (M) | 21 | Aug. 16 to 19 | Well-marked rash, like No. 1 | Slight redness | T 99 | Norm. | Norm. | ... | ... |
| 25 | C. B. ... (M) | 30 | Aug. 20 to 23 | Slight: limited to chest and back | No redness | T 98·8 | Norm. | Norm. | ... | ... |
| 26 | W. McA. (M) | 14 | Aug. 25 to 28 | More diffuse and brighter rash than the others | Redness of Velum Palati. Tongue not "strawberry-like" | T 100<br>P 108 | 99·8<br>96 | 98·4 | ... | ... |
| 27 | M. S. ... (F) | 20 | Aug. 31 to Sept. 4 | Considerable conjunctival injection: somewhat crescentic arrangement of papules, resembling rash of measles | Considerable injection of Velum Palati: some redness of Tonsils | T 100<br>T 96 | 100<br>96 | 99<br>84 | 98·8<br>84 | 98·4<br>84 |
| | SERIES II. | | 1880. | | | | | | | |
| 28 | D. B. ... (M) | 12 | June 23 to 26 | Papules discrete: brownish-red rash universal | Palate and Tonsils injected | T 98·8<br>100<br>101·5<br>P 100 | 100<br>96 | 98·8<br>84 | Norm. | ... |
| 29 | J. M. H. (M) | 14 | June 27 to 30 | Similar to preceding, but brighter | As above | T 101·4<br>P 100 | T 100·5<br>87 | 99·4<br>86 | Norm. | ... |
| 30 | T. H. ... (M) | 13 | June 28 to July 1 | Similar to preceding, but less extensive | Slight injection of Fauces | T 100<br>P 96 | 99·8<br>90 | 98·4<br>84 | ... | ... |
| 31 | D. K. ... (F) | 15 | July 5 to 8 | Brownish-red rash, first seen on face and neck, afterwards extending to limbs | ... ... | T 101·2<br>P 100 | 100·6<br>98 | 98·6<br>90 | Norm. | ... |

| dition as to quamation. | Condition as to Sequelæ, Albuminuria, &c. | Duration of Isolation. | Possible Source of Infection. | Exanthems previously undergone. | OTHER PARTICULARS. | Sequel of History. | No. |
|---|---|---|---|---|---|---|---|
| ıe, though arefully rched for | None | June 15 to July 2 | ? | Measles | Was completely isolated from June 15 to June 24. Sent home into country from June 24 to July 2, and slept with younger sister: no spread of disease there | Left Institution 1875 | 1 |
| None | None | July 7 to Aug. 31 | No. 1 | None | ... ... ... ... | Died 1877 | 2 |
| None | None | July 8 to Aug. 31 | No. 1 | Doubtful | ... ... ... ... | Had { Scarlatina, 1877 / Measles, 1880 | 3 |
| None | None | July 9 to Aug. 31 | No. 1 | Measles | (Some little difficulty in thermometry in this case) | Still in Institution | 4 |
| None | None | July 18 to Aug. 31 | ? No. 4 (Meets at School) | Measles | ... ... ... ... | Had Scarlatina, 1877; Left Institution Jan. 1880 | 5 |
| None | None | July 21 to Aug. 31 | No. 4 | Measles and Scarlatina | Medical man confirms friends' statement of his having had Scarlatina about three years before | Left Institution in 1879 | 6 |
| None | None | July 22 to Aug. 31 | No. 4 | None | ... ... ... ... | Died 1879 | 7 |
| None | None | July 23 to Aug. 31 | No. 4 | Doubtful | ... ... ... ... | Died 1880 | 8 |
| None | Persistent high temperature (99·5) | July 23 to Aug. 31 | No. 4 | Measles | Persistent high temperature proved symptomatic of tuberculosis, of which he subsequently died | Died 1874 | 9 |
| ry slight branny. gust 4th. | None | July 23 to Aug. 4 | No. 5 | None | Went home to visit friends Aug. 4. No spread of disease there | Left Institution, Jan. 1880 | 10 |
| None | None | July 23 to Aug. 31 | No. 5 | None | ... ... ... ... | Had { Scarlatina, 1877 / Measles, 1880 | 11 |
| None | None | July 23 to Aug. 4 | No. 5 | Measles | Visited home, Aug. 4. No spread of disease there | Left Institution, 1879 | 12 |
| None | None | July 24 to Aug. 31 | No. 6 or 9 | Measles | ... ... ... ... | (Left 1880, Oct.) | 13 |
| None | None | July 24 to Aug. 31 | No. 5 | Measles | ... ... ... ... | Left Institution, 1878 | 14 |
| None | None | July 25 to Aug. 31 | No. 6 or 9 | Measles | ... ... ... ... | Still in Institution | 15 |
| None | None | July 25 to Aug. 31 | No. 6 or 9 | None | ... ... ... ... | Had { Scarlatina, 1877 / Measles, 1880 | 16 |
| None | None | July 26 to Aug. 31 | No. 5 | Measles | ... ... ... ... | Died, 1875 | 17 |
| None | None | July 26 to Aug. 31 | No. 8 | Measles and Scarlatina | This girl helped to make beds of several patients who had rash | Had Scarlatina, 1877 | 18 |
| None | None | July 27 to Aug. 25 | No. 6 or 9 | None | Went to Blackpool, Aug. 25. No spread of disease there | Had Scarlatina, 1877, died, 1878. | 19 |
| None | None | July 26 to Aug. 31 | No. 5? | Measles | Came from same department as No. 5, and had no communication with intervening cases | Had Measles, 1880 (severe) | 20 |
| None | None | Aug. 9 to 25 | 10, 11 or 12 | None | Visited friends, Aug. 25. No extension of disease amongst younger children at home | Had { Scarlatina, 1877 / Measles, 1880 | 21 |
| None | None | Aug. 9 to 31 | No. 18 | Scarlatina | "Has certainly had Scarlatina; not Measles" | Died, 1879 | 2 |
| None | None | Aug. 16 to 31 | No. 22 | None | There was unusual irritability of temper for two days previous to manifestation of rash | Had Measles, 1880 | |
| None | None | Aug. 16 to 31 | No. 20 (probably) | Measles | ... ... ... ... | Still in Institution | 24 |
| None | None | Aug. 20 to Sept. 14 | No. 24 (certainly) | Doubtful | ... ... ... ... | Had { Scarlatina, 1877 / Measles, 1880 | 25 |
| ry slight ranny. | None | Aug. 25 to Sept. 14 | No. 24 or 25 | Measles and Scarlatina | Symptoms resembled those of Scarlatina | Died, 1877 | 26 |
| None | None | Aug. 31 to Sept. 22 | No. 22 or 23 (certainly) | Measles and Scarlatina | This case resembled Measles more than did any of the others | Still in Institution | 27 |
| None | None | About a month | ? | Measles and Scarlatina | Measles had prevailed in Institution four months earlier, and six cases of Scarlatina occurred in May, June, and July, 1880 | Still in Institution. | 28 |
| None | None | Do. | No. 28 (?) | Measles and Scarlatina | Do. do. do. | Do. | 29 |
| None | None | Do. | No. 29 | Measles | Do. do. do. | Do. | 30 |
| None | None | Do. | Preceding Cases. | Measles, Feb. 1880 Scarlatina, June 18 80 | Was convalescent from Scarlatina when rash of Rubeola Notha appeared | Left Institution, July 1881 | 31 |

27th June, J. M. H., a boy from the same department as the preceding, came under observation. A rubeoloid rash, lasting three days, with a maximum temperature of 101·4° on the evening of the first day, and unattended with any constitutional disturbance, was the principal feature in this case. There was slight reddening of fauces and conjunctival injection, but no catarrhal symptoms. This boy was also reported to have had both scarlatina and measles in infancy. On June 28th, T. H., a lad from same department as preceding, had similar though less severe rash: he had had measles but not scarlatina in infancy. These three boys were placed, for the sake of isolation, in the lower rooms of a detached wing of the Asylum, the upper floor of which was occupied by cases of scarlatina. One of these, a girl aged fourteen, who had sickened with scarlatina on the 26th May, and (after a pretty severe attack) was making a satisfactory recovery, surprised her nurse by displaying on the evening of July 5th (the forty-second day) a brownish-red rash on face and neck, afterwards extending to trunk and limbs, and accompanied by an elevation of the temperature to 101·2° on the first evening. The rash remained visible during the second day, and gradually faded on the third. There seems every reason to believe that in this case infection had spread from the cases downstairs, which, it will be remembered, occurred from June 23rd to June 28th. Special interest attaches to this case, inasmuch as the patient had had in the preceding February an unquestionable attack of measles, and in the preceding month an unquestionable attack of scarlatina.

A review of the history of the several epidemics to which reference has been made will, I trust, tend to throw some light upon the relations of rubeola notha with scarlatina and measles respectively. Of the twenty-seven patients who are recorded to have had rubeola notha in 1874, thirteen remained under my observation in the institution during the epidemics of scarlatina in 1877 and 1880, and during the epidemic of measles in 1880. "Of these thirteen patients, no fewer than *five* had, in the most unmistakable way, both scarlatina and measles in the successive epidemics; whilst of the rest, *two* had measles only, and one scarlatina, in addition to rötheln. Thus more than half of the patients remaining under observation from the rötheln epidemic were subsequently attacked with scarlatina or measles, the majority with *both* of these exanthems. It is noteworthy that of the seven patients who had measles in 1880 only one was said to have had that disease *previous* to the rötheln epidemic. The other six patients, who had rötheln in 1874 and escaped the measles in 1880, are reported to have had the latter in infancy." *

I have advisedly left out of consideration the fourteen cases making up the twenty-seven tabulated, several having died, and others living at a distance with their friends. But I trust I have advanced facts enough to justify me in urging upon the Section the following conclusion:—*That rubeola notha is a disease sui generis, and is entitled to a distinct place in Pathology.*

My experience leads me to dissent from the view that the cases properly designated as rubeola notha are nothing more than mixtures or modifications either of scarlatina or measles. My cases show, so far as so small a number of cases can show anything, that the patients who have had rubeola notha are in no degree protected from subsequent attacks either of scarlatina or measles; that cases of rubeola notha never communicate the infection of undoubted scarlatina or undoubted measles; and that the characteristic sequelæ of these two latter diseases never follow cases of rubeola notha. In opposition to the theory that rubeola notha is merely secondary measles, there is the fact that six (if not seven) of my patients had measles, not *previously*, but *subsequently* to a characteristic attack of rubeola notha; and the case of the

* *British Medical Journal*, No. 1019, p. 49.

girl who had all three disorders within six months, under my own observation, furnishes perhaps the most striking evidence that could be advanced in favour of their distinct pathological character.

The confusion existing in the minds of the profession as well as of the public with regard to the "so-called rubeola, rötheln, or German measles," is much to be deplored. That an epidemic which is, in truth, a compound of scarlatina and of measles, and combines the dangerous elements of both disorders, should pass by the same name as the comparatively innocent affection which I have described, is an anomaly which cannot fail to be detrimental to the public safety. The term "German measles," applied to such a compound disease (I mean the concurrence of scarlatina and of measles), albeit it has sanction of high medical authority, gives a false sense of security, and leads to a neglect of precautions which the public are now learning to adopt with regard to the later stages of scarlatina. Quite recently it has fallen to my lot to visit a village decimated (as regards number of cases) by this compound disease, in which the scarlatina rash and the rash of measles appeared separately, and the characteristic symptoms of each disorder were distinguishable. Severe throat symptoms, nephritis, and free desquamation were observed in many of the cases; and in these there seems no reason to doubt that scarlatinal poison would be diffused, either separately or in conjunction with the infection of measles. The International Medical Congress will confer a benefit upon society at large if, by the discussion of this question, it fixes the legitimate application and obtains the authoritative definition of the "so-called rubeola, rötheln, or German measles," thus at last securing in the bonds of science this very Proteus of Pathology.

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## *On Rubella; Rubeola sine Catarrho; Rötheln, or German Measles.*

Dr. William Squire, London.

This exanthem, popularly known among us as "false measles," has been mentioned by various writers under the above names, or as rubeola notha, spuria vel incocta, and sometimes as epidemic roseola, for the last hundred years.

A century was required to complete the separation of measles from small-pox. Another century passed from Sydenham to Withering before scarlet fever was finally distinguished from measles. The disease in question has but a superficial resemblance to scarlet fever; its relations are clearly and closely to measles or rubeola as defined by Sauvages and Cullen. The century is fulfilled that should give autonomy to rubella.

Whether introduced by the Moors into Spain and Italy in the eighth century, or only first described by the Arabian writers, is uncertain. Their name of Hrasbah included both measles and scarlet fever, and gave origin to our comprehensive word "rash." It was included with scarlet fever by Sydenham, and consequently considered a slight ailment. We know that it was so included by Richard Morton (Πυρετολογιας, 1694), where scarlatina is spoken of as a mild form of scarlet fever, and a case of scarlet fever is given as "confluent measles." Hildebrand imagined it to be a true hybrid between scarlet fever and measles, in a way which those who now heedlessly adopt his view would hardly contend for. Bucholz mentions the long incubation. Heberden speaks of measles without fever or precedent catarrh. Willan named it *rubeola sine catarrho*, noticing that it did not protect from measles. Bateman confirms this (Synopsis, Lond., 1813), and makes it a non-febrile variety

of rubeola. The name *rubeola sine catarrho* is objected to by Mason Good, as the genuine measles themselves, capable of affording emancipation, have sometimes appeared with very slight catarrhal symptoms. He says it has been called, and especially by the German writers, spurious measles; but as it occurs more frequently when the genuine measles are epidemic, it is less properly a spurious than an imperfect or immatured rubeola. His prejudice for this being merely a variety of measles, and not itself an independent exanthem, prevented a right judgment being formed on the series of cases observed by Dr. Maton, already before him, which he considered to be varieties of scarlet fever, though distinguished from it by a long incubation period,—a character he notices to belong to certain cases of measles reported by Bucholz when the "efflorescence evinced a like procrastination, and appeared as late as the twenty-first day." *

To Dr. William George Maton, F.R.S., Physician-extraordinary to Queen Charlotte and a Fellow of our College of Physicians, is due the merit of discriminating the exanthem in question both from measles and from scarlet fever. In a contribution to the *Medical Transactions* of the College, vol. v. p. 149, Lond., 1815, Dr. Maton says, that having several times seen cases called either scarlatina or measles, in which the symptoms of illness were trivial and the external characters insufficient to decide their nature, he determined to keep a scrutinising eye on all similar cases. Then follow a series of eight cases. The first is a girl of thirteen, who on August 18th, 1813, had this rash; her face suffused with innumerable points, but she did not feel ill. A sister with her complained slightly, and had some fulness of the small cervical glands; next day she had the rash. In the room with these two sisters were four others of the family, aged from ten to seventeen years; two of these had the rash on September 4th and 5th, the other two on the 7th of September. Two other relatives, the eldest brother, aged twenty-four, and his infant son, a year and a half old, were taken on September 24th and 30th. Dr. Maton remarks: "There is only one other exanthem that I know to which these cases can be considered referable, that is, roseola; but tumours do not occur in roseola, nor is it infectious. The period intervening between the application of the infectious influence and the commencement of the disease was considerably longer than has been noticed in scarlatina. Hence it seems requisite to form a new designation, which, however, I do not venture to propose at present, being satisfied with calling the attention of my colleagues to the subject." It is needless to summarise all the notices of the disease from Dr. Babington to the present time (Trousseau, *Edin. Med. Jour.*) It has been long enough under general notice to deserve a name, and may well receive from the present International Congress a distinctive appellation, which Dr. Maton so wisely chose to defer. He has summed up all the characteristics of the disease in these few cases—the contagious property, the long incubation, the enlargement of the small cervical glands. All that subsequent observation has done beyond confirming these characters is to show that the disease is self-protective, and in fact possesses all those qualities of an independent and specific disease so well summed up by Sir Thomas Watson in the case of varicella, when he says, "This may be a very trivial disorder, but it is stamped with the characters peculiar to the group. It is contagious; it runs a definite course; it occurs but once in the same person." Of two allied diseases, the slighter form is more slowly recognised and distinguished. After the separation of measles, smallpox and varicella were long associated; but no one now doubts, however modified, their absolute distinction. Since the clear definition of scarlet fever, measles is found to have a near ally, as near as chicken pox is to smallpox.

* The Study of Medicine, Lond., 1825, vol. iii. p. 16.

Their gradual differentiation is brought about in the same way. The fever is observed to be less in the one than in the other; the severer disease affords no immunity from the slighter ailment, nor is that in any way protective against the other, while each is self-protective from recurrence. The difficulty of establishing these points may seem to be out of proportion to the practical good to follow; but definite ideas as to the course of the milder ailments, and of the effects produced by them, have important bearings, both as to the care of those attacked as well as on the safety of others, and all that can be collected concerning them is deserving of careful record.

Rubella, then, may be defined as a specific eruption, the rash appearing on the first day of the illness, beginning on the face in rose-red spots, extending next day to the body and limbs, subsiding with the fever on the third day, and rarely either preceded by catarrh or followed by desquamation; the former never for three days before, the latter never after three weeks. Propagated by contagion, it occurs in epidemics of limited extent with sporadic offshoots. It has a long period of incubation, mostly a fortnight, the extreme being twenty-one days; hence a difficulty in tracing the source of personal infection. This is increased by the slight and transient nature of the infection allowing patients to mix freely with others. Cases have been noted where the incubation may possibly have been as short as five or seven days.

One attack is preventive of a recurrence, but is not protective against either measles or scarlet fever, nor do attacks of either of these diseases in any way modify the liability to this one: it is as distinct from them as is chicken-pox from smallpox. During epidemics of measles or of scarlet fever, mild and irregular cases of both are not unfrequently mistaken for this exanthem; well-marked outbreaks of it are often attributed to measles, while slight attacks of scarlet fever are miscalled rötheln, or a hybrid disease imagined which has no existence. Young infants are said to escape; adults not unfrequently suffer, as many persons escape attacks in childhood; sex makes no difference. The disease is contagious even before the rash is thrown out, and it continues to be so for some days, or it may be for two or three weeks afterwards. Second attacks are rarer than for scarlet fever, but the rule against them may be less absolute than for measles. It is seldom fatal. When a mortality of attacks is reported as high as 3 per cent., measles is probably the epidemic present.

Slight fulness of head, heaviness, pain, or giddiness is felt, with a little aching of back and limbs, or tenderness of throat for a few hours on the evening before the rash appears. The cervical lymphatic glands are always perceptibly enlarged, and sometimes have been so for a few days before the rash appears. Mostly the rash is first seen with surprise, as the feeling of illness has already passed, or may have escaped notice. There is redness of the fauces or uvula; less mottled than in measles, not so intense as in scarlet fever; the tonsils are rather full and smooth; there is no ulceration. The eyes are suffused, but there is little or no coryza; the lids are somewhat swollen and irritable; the face is flushed, and the cheeks red or full even before the appearance of the spots; these are bright red, raised, rounded, with clear skin between them, but they soon coalesce; not grouped as in measles, the spots are more prominent than in scarlet fever, and there is not the finely diffused redness of the neck and chest as in that disease. Moreover, the rash is already fading from the face while extending to the limbs, so that it is generally subsiding by the third day. Sometimes an odour as of measles attends the rash. Some itching or a very fleeting yellowish tinge, but no discoloured mottling of the skin is left, and no desquamation, except of the branny kind, which occurs early, and is never delayed to the third week. However little illness is felt at the beginning, a continuous rise of temperature commences with or just before the rash; it may reach 102° to 103°, or

be only two degrees above the normal. With rest in bed this may fall one degree by the end of the second day, but is evenly maintained as the eruption proceeds, or subsides with it on the third day. During the following week it is readily disturbed, either raised by exertion or depressed by fatigue or chill. At this time recrudescence of the rash has been observed. Slight coryza may come on after the rash has faded, the eyelids become sticky, the nostrils stuffed, the throat sore, or some cough begins. Exposure or want of care at this time may determine serious disturbances of health, generally with pulmonary complications. The urine is often high coloured in the early part of the illness; the chlorides are increased, but there is no albuminuria, nor has this ever been known to follow. In some few cases transient complaint of the throat or of fatigue has been made a week before the rash, or epistaxis has occurred; fulness of the small cervical glands is often felt, but no constant intermediate symptoms are found, and any feeling of sickness is without fever.

The sudden onset of this form of rubeola without previous sneezing or cough distinguishes it from measles, to which it is much more nearly allied, as well by general characters as by the kind of rash, than to scarlet fever; but the spots are more evenly distributed, often at wider intervals, and not in groups. There is no gradual rise of temperature before the rash nor the sudden fall afterwards, both characteristic of measles. The small lymphatic glands are palpably enlarged in this ailment down the sides of the neck, and perhaps behind the ears, but not specially at the angle of the jaw, as in scarlet fever. The rash on the second day may look like scarlet fever, or the red flush of scarlet fever, at first sparsely distributed or with prominent red papillæ, may lead to mistake; but the sudden onset is much more marked in scarlet fever, when, should the rash appear as early, it will be more intense on the third day, especially on the neck and chest. Moreover, the fever persists till the fifth day in scarlet fever, even when not greatly elevated; there is also the state of the pulse and tongue or the prominence of throat symptoms. Sometimes it is not till the second or third week that the kind of desquamation, and possibly signs of renal irritation or the occurrence of other cases, complete the diagnosis. The length of interval between succeeding cases is also a distinction. Roseola is not contagious; it occurs in red points or spots not raised above the healthy skin between; there are no throat symptoms, no enlarged lymphatic glands, nor fever. Erythema affects parts of the skin only. Attention restricted to the character of its eruptions often leads to error.

Recovery is so much the rule, that were it not for the mischief any febrile disease may excite in weakly children, the risk of pulmonary disease from close confinement during the rash, or from premature exposure afterwards, all cases of rubella might be expected to do well. In all cases look to the throat and examine the chest. Two months may elapse before health is quite restored. It is not always the trivial disease so often met with. In any large number of cases some will develop unusually high temperatures, just as is seen in varicella; and in others, slow convalescence or marked deterioration of health is dated from this accident. This is due to the individual state or conditions, and not to change in the nature of the disease; it is seen with equal severity in those who have, as in those who have not, had measles previously. Where persons are said to have had measles twice, one of the attacks has been of this kind. A more serious mistake is often made in passing slight cases of scarlet fever under this category by the name, perhaps, of a rose rash or roseola, instead of rubeola or rötheln. Such terms as roseola or even rosalia, by which scarlet fever has been called, should be expunged from the nomenclature of the contagious exanthemata. Whether there be a distinct variety of scarlet fever concealed under the disease as now known is not under consideration. Scarlet

fever and measles, scarlatina and rubeola, belong to different types of disease; the one of short incubation, sudden ingress, and slow decline, with special tendency to throat and kidney mischief; the other of long incubation, gradual ingress, and sudden decline, with tendency to bronchial rather than to renal irritation. It is to this latter, the order rubeola, that the disease under consideration clearly and undoubtedly belongs. I would suggest that, as representing the allied but distinct form of rubeola, the diminutive of this word, "rubella," as already proposed in the United States by the American Dermatological Association, should receive the sanction of this International Medical Congress for general use and adoption.

## DISCUSSION.

Dr. FERGUS, Marlborough: The distinction between measles and scarlatina and rötheln has been very much confused by giving the name rubeola to rötheln. Rubeola was the old name of measles. The College of Physicians gave the name *morbilli* to measles and left rubeola out, so that no certainty of distinction can be had. Rötheln is very distinct from scarlatina, and has occurred immediately after an attack of the latter disease. It is most difficult to describe the difference in books, but when the diseases are seen in company the distinction is easy and pronounced. Albuminuria and desquamation rarely occur in rötheln. It would help to avoid confusion if the word rubeola were given to measles by the College. Morbilli is not a fit name for a disease like measles, and will never be understood by the general public.

Dr. JOHN GLAISTER, Glasgow: The conclusion that rötheln is a distinct exanthem from measles or scarlatina has been forced upon me by the fact that I have just lately experienced an epidemic of this disease, in which I saw at least twenty cases. So far as my observations have gone, the incubation period is not a fixed one, varying from four or five days up to a longer period. The characters of the rash are these:—It appears on the third day of the illness, at first on the face and chest, and then the rest of the body. It sometimes distributes itself in crescentic patches mixed with scarlatinal-looking patches; and it begins to disappear on the fourth day. In one case the rash became purpuric. The temperature at the beginning of the eruptive period is about 102° or 103°, gradually declining till the fourth day, when it comes back to normal. I have not seen any desquamation except branny scales. The catarrhal symptoms were in some of the cases very severe; conjunctivitis considerable, with great intolerance of light; sneezing generally present at the beginning of the eruption and before its appearance, but soon passing off. Fauces and tonsils were in most cases injected, sometimes much so; the latter were sometimes so much swollen as to cause difficulty in swallowing; there is occasionally sufficient laryngeal injection to cause difficulty in breathing. What has forced the distinct entity of rötheln upon my mind is, that children have taken it whom I had previously attended with ordinary measles and scarlatina; and when once it made its appearance, generally more members in the family were affected. The treatment is simple and ordinary.

Dr. MARTIN OXLEY, Liverpool: It is a very difficult thing to assign a place to German measles, and I have been very much struck by the fact that experienced practitioners have great difficulty in coming to a conclusion as to the nature of a case accompanied by an eruption of the skin. The mistake seems to me to be in naming a disease from the appearance of the rash. I think we

should wait until we can be certain. This so-called German measles always follows, in my experience, an epidemic of scarlet fever or measles. If it follows the former, it is like scarlet fever; if the latter, it has the appearance of measles. In Liverpool some years ago at a public institution the medical officer was in this difficulty. He had 120 boys packed in an orphan asylum in the centre of the town. He had, besides, two other buildings, one filled with girls, another full of infants. These buildings were in a ring fence. His difficulty was that a few boys had a rash like that seen in scarlet fever. They had slight fever, temperature 100·5°; but they were not ill—they were able to eat. They had little headache; the rash was out over the chest and legs; the disease was spreading, and it was necessary to decide on its nature. He waited, and found that there was no strawberry tongue on the second or third day. He therefore decided to treat the disease as German measles, *i.e.*, keeping in bed while the rash lasted, giving no medicine, and raising no alarm. There was no peeling of the feet or hands, and no child out of the eighty or ninety who had the complaint died or had albuminuria. Speakers here as well as writers elsewhere vary much in their description. On one hand they describe it as like measles, on the other as resembling scarlet fever. I think we must have more evidence of there being a distinct disease before we treat what is known as German measles as a throat complaint.

Dr. J. A. Wood, Liverpool: The rash in German measles is always characteristic, and is generally sufficient to base a diagnosis on. It not only comes out in an irregular and sudden-like way in elevated patches, but the colour alone is, I think, pathognomonic. The peculiar tint of colour is difficult to convey exactly, but it is not livid like plain measles, nor has it the injected efflorescence of scarlet fever. I do not wish to say anything of many other symptoms which may be like, but are not identical with, the symptoms of other complaints, which is a distinction with a difference. It is strange, viewing the whole case, that any confusion should ever arise.

Dr. D'Espine, Geneva: made some observations on a tolerably widespread epidemic of true rötheln which last winter followed an epidemic of measles at Geneva, and which towards its close was accompanied by cases of true scarlatina. He had no doubt of the reality of this disease as a distinct entity, though in some cases the diagnosis was difficult.

Dr. A. Jacobi, New York: The difficulty in making a diagnosis must be great in many instances, for the description of cases and epidemics varies largely even with the best authors. The difficulty is greatest with those in large cities, when and where, as a rule, there are complications with other epidemics, mainly measles (generally preceding) or scarlatina (often following). During the prevalence of any infectious disease, everybody is, to a certain extent, under its influence. Thus during epidemics of diphtheria, scarlatina, erysipelas, cholera, everybody is liable to be taken on slight provocation, or, at all events, to exhibit some of the symptoms. Thus pharyngitis and cervical adenitis are very common amongst people who are considered well. Thus I explain the fact that symptoms not belonging at all to rubeola are mentioned amongst its symptoms. In large institutions, distant from large cities, distant from epidemics of measles and scarlatina, rubeola has been observed, as a distinct disease, to be very contagious, very little febrile, no or very few cases attended with throat symptoms or adenitis, the eruption resembling measles more than scarlatina; convalescence absolute, safe, and easy, and desquamation (farinaceous) very trifling.

J. P. G. Houstoun, Savannah, Ga.: referred to cases of German measles as observed in Savannah and in parts of Florida during the winter of 1880–81 and spring of 1881. The cases were very numerous, very mild, and almost universal, even in the more remote country places, and where there were no cases of measles or scarlatina. It occurred irrespective of previous attacks of measles or scarlet fever. Physicians of large practice in these localities do not hesitate to consider the disease distinct from rubeola and scarlet fever.

Mr. W. Stewart, Barnsley: The force of the discussion of this question seems to hinge principally upon the question of diagnosis. The previous speakers seem to lay the greatest, and, indeed, the only stress, upon the character of the eruption as being the mark by which we are to recognise it as a separate disease. But are we to erect into the importance of a separate disease an affection which only shows its different character by a modification of the measly rash? In the tables distributed by Dr. Shuttleworth, I think all the cases might come under the head of either measles or scarlet fever; and in the two cases No. 10 and No. 26, the symptoms are more those of scarlet fever, especially the fact of their being followed by desquamation. I have recently had a case of a like nature, which for a little time puzzled me with regard to its nature, the premonitory symptoms in which were like those of measles, but where the character of the rash was too papular and discrete to say that it was measles straight offhand. On the second day after, hot applications being applied, the rash became confluent, and the after-history of the case clearly showed it was measles. I believe the affections are the same.

Dr. Shuttleworth, Lancaster: The series of cases I have presented were of a very mild type, contrasting with those of Dr. Cheadle, which were very severe, but might, notwithstanding, be of the same essential character. For my own part, I should be very sorry to undertake to diagnose a case of rötheln with certainty at the first onset, but I think I could generally do so on the *second* day of the rash. There is often a *brownish* hue in rötheln contrasting with the bright redness of scarlatina, and an absence of the crescentic arrangement of papules observed in measles. Moreover, the temperature in rötheln (in mild cases, at any rate) usually attains its maximum on the morning of the second day. This very rapidly subsides, and there is far less febrile disturbance than in measles or scarlatina. But it is only by observation of a *series* of cases, with *their sequelæ and histories*, that one becomes convinced of the distinctive character of rötheln. As medical officer to an institution containing 500 children, the differential diagnosis of rötheln is for me always a matter of much anxiety, and in any dubious case of rash, of course, precautions from the first are indicated. There are, no doubt, epidemics in which scarlatina and measles may prevail simultaneously, and each disease communicates its like; but this is not "German measles" in the sense in which that name and its equivalents were used in my paper; and I think such use of the term constitutes a danger to public health by inducing people to be careless during convalescence.

Dr. Cheadle, London: The papers by Dr. Lewis Smith and Dr. Shuttleworth confirm the fact that previous attacks of measles and scarlatina afford no protection against rötheln. In Dr. Lewis Smith's series of forty-eight cases, twenty-seven had had measles before, eight had had scarlatina. Out of Dr. Shuttleworth's thirty-one cases, nineteen had previously had measles, eight had had scarlatina. Dr. Shuttleworth appears to hold that, while the mild cases described were really a distinct exanthem, viz., rötheln, those of more severe form were something more distinct—hybrids between measles and scarlet fever. With regard

to that point I would remark, that the severe cases I have recorded bred neither scarlatina nor measles; they bred the disease ordinarily recognised as rötheln. The severe cases and the mild cases described in my paper were directly related, many of the latter springing from the former by immediate descent. With regard to the severity of the laryngeal symptoms, I would call attention to a point noted in the paper, but which I have omitted in the reading of it for want of time, viz., that in an epidemic prevailing at Folkestone simultaneously with that of 1879 in London, laryngeal symptoms were equally prominent and severe. I was informed by Mr. Tyson of that town that three cases of laryngeal obstruction had occurred, so extreme as to require tracheotomy, another in which the operation was threatened, and that other cases of the kind had been met with. The case mentioned by Dr. Oxley, in which a measles-like rash accompanied by severe throat symptoms and exudation on the fauces occurred in a patient who had had measles a short time previously, strongly confirms the conclusions put forward in my paper.

Dr. West, President: A general consent exists—for there are only two dissentients—as to the existence of a distinct disease, similar in some respects to, but not identical with, either measles or scarlatina. It is separated from the former by the incubation period, which is longer, and approaches more nearly to, though it usually exceeds, that of measles. It is further separated from scarlatina not only by a difference in the character of the rash, which, however, it is not always possible to appreciate within the first twenty-four hours of its appearance, but also by the non-occurrence either of desquamation or of albuminuria; while its stage of convalescence is not attended with any of the sequelæ which follow scarlatina or measles. A circumstance which marks the distinction of rötheln from either is the great frequency of its occurrence in those who have had both measles and scarlatina. Of Dr. Cheadle's cases, thirty-two in number, twenty-two had previously had measles, and ten of them under his own observation. Of Dr. Smith's forty-eight, nineteen had had measles, and in another epidemic eight had had both measles and scarlatina within six months; and though it is a well-known fact that both diseases may occur more than once, yet a frequency such as this is unexampled in the case of either disease. Its affinity seems to be rather with measles; and the disease resembles, if it is not identical with, Willan's *rubeola sine catarrho*. The rash often is closely similar in the two diseases; the general character of the symptoms approximates closely. As a rule, it is very mild in type, running no danger, and calling for no treatment, though the case related by Dr. Cheadle shows that, notwithstanding its distinctive peculiarities, it may present a very severe set of symptoms. An observation, too, of Dr. Jacobi's is to be borne in mind, that occurring, as it often does, either intercurrent between two epidemics, one of measles, one of scarlatina, or following or preceding one or other, its character may be so far modified as to approximate in some respects to one or the other. A remark this, to be borne in mind always, and well for our Epidemiological Societies to remember, as it was borne in mind by Sydenham, as his remarks on the epidemic conditions of different years testify. One great practical point is to take care in doubtful cases of scarlatina, because they may be mild, not thoughtlessly, and for the sake of giving present relief to the anxiety of parents, to say of a case, "It is German measles," and thus to be the unwitting cause of the spread of scarlatina in a family. Another is not to forget that its affinities are with measles, not with scarlatina, and that they resemble each other somewhat as varicella and variola—alike but not the same.

> "Facies non una, nec diversa tamen
> Qualis stolet esse sororum:"

not twin-sisters, indeed, but half-sisters at any rate.

## *Le Rachitis et la Syphilis héréditaire.*

Prof. PARROT, Paris.

Le rachitis reconnaît pour cause unique la syphilis héréditaire, et constitue l'altération la plus avancée parmi celles dont cette maladie frappe le système osseux.

Démontrer cette proposition, tel est le but que je me propose. Pour cela, je vais faire voir : qu'à partir des derniers mois de la vie intra-utérine, jusqu'à une époque qui n'atteint jamais le début de la seconde dentition, chez un certain nombre d'enfants, le squelette subit une altération polymorphe, qui évolue d'une manière lente et régulière, toujours la même, aux mêmes instants de la vie, et qui, sous sa forme dernière, est identique au rachitis. Or tous ceux qui en sont atteints sont la proie de la syphilis héréditaire.

Il faut donc avant tout que la syphilis ne puisse échapper ; qu'elle soit reconnue sous ses divers masques ; ce qui me paraît irréalisable à l'aide des moyens de diagnostic actuellement connus. Aussi pour établir la relation que je viens de formuler, dois-je faire connaître quelques signes, qui bien que très communs, n'ont pas encore attiré, du moins à ce point de vue, l'attention des cliniciens.

La syphilis héréditaire se révèle durant sa période d'activité, par des accidents actuels ; plus tard, quand elle est éteinte, on peut encore la reconnaître ; mais seulement aux empreintes qu'elle a laissées.

Les affections de la période active, bien connues, sont comme on le sait, à la surface : l'alopécie, les bulles, les taches, les plaques, les pustules, les ulcérations ; et profondément : les catarrhes, les abcès, les gommes ; et toute une série d'affections viscérales.

Je m'arrêterai quelques instants à une altération de la muqueuse linguale, encore peu connue, et qui présente ceci de particulier, qu'on l'observe avec les précédentes ; et beaucoup plus tard, alors qu'elles ont depuis longtemps disparu ; c'est-à-dire, depuis les premiers mois de la vie, jusqu'à la 7$^{me}$ année, et peut-être même au delà.

Cette affection, circinée et desquamative, débute par des tâches blanches circulaires, où l'épithélium est plus épais que dans le voisinage, se desquamant à leur centre tandisqu'à la périphérie elles envahissent de nouvelles régions, d'où résultent des croissants ouverts en avant, qui s'étendent et se succédent sur la langue, comme des ondes sur une surface liquide ; apparaissant et cessant d'exister, pour se montrer de nouveau, sans cause appréciable et durant des périodes indéterminées.

Propre à la syphilis héréditaire, dans les conditions que je viens de faire connaître, elle n'est pas sans présenter quelque analogie avec d'autres desquamations de la langue. D'ailleurs, ce n'est pas seulement à la surface de l'organe que le mal est apparent ; le microscope montre dans le corps papillaire des amas de corpuscules lymphoïdes, comme au niveau des altérations cutanées et viscérales.

J'aborde maintenant les points véritablement nouveaux de cette étude, c'est-à-dire, la description des empreintes laissées par la syphilis héréditaire, et qui la rendent aussi incontestable, que si elle était là présente en pleine activité. Comme les médailles commémoratives d'événements passés, dont l'effigie et le millésime s'effacent à la longue, mais qui, dans cet état fruste, sont encore visibles pour un œil exercé, elles peuvent servir à reconstituer l'histoire des anciens troubles morbides, et même à fixer leur date.

Parmi ces stigmates, les cicatrices cutanées tiennent la première place ; et jouent le principal rôle dans ce diagnostic posthume.

On peut les rencontrer sur les différentes régions du tégument externe ; mais comme les syphilides dont elles sont la trace, elles se groupent en des points que j'appellerai volontiers *singuliers*, et tirent leur principale valeur de ces localisations.

Sur les lèvres, elles se présentent comme des sillons blancs, à droite et à gauche du lobule, aux commissures, ou sur un plus grand nombre de points. Mais c'est à la partie postérieure et inférieure du tronc, au niveau du sacrum et du coccyx, et sur les membres inférieurs en arrière, notamment sur les fesses et les cuisses, qu'elles sont le plus nombreuses et le plus nettes. Circulaires et de diamètre variable, diversement groupées ; rouges, violacées ou d'un blanc nacré, déprimées ou non, plissées et parfois même un peu gauffrées, elles sont souvent entourées d'une zone de pigment. Celles qui succèdent aux gommes, ont un aspect particulier et tout-à-fait caractéristique. Limitées aux membres inférieures, toujours rares et éloignées les unes des autres, elles consistent en une dépression infundibuliforme, violacée surtout au début, au fond de la quelle, il y a une petite cicatrice linéaire. Longtemps les tissus mous restent indurés à leur niveau.

Sur les dents, les empreintes de la syphilis héréditaire sont beaucoup moins communes que les précédentes ; mais infiniment plus solides et tenaces ; peuvant garder leur physionomie originelle, non seulement durant la vie de l'individu, mais après sa mort, pendant de longs siècles. Nous en avons de nombreux exemples ; et l'on peut dire qu'après les documents fournis par les os, il n'en est pas de plus incontestables, pour démontrer la très haute antiquité de la syphilis.

Tout ce qui a trait à ces empreintes, se présente à nous avec une rigueur mathématique, tant au point de vue des relations qui lient celles des différentes dents, que, dans leur chronologie ; si bien que celle d'une dent étant connue, toutes les autres s'en suivent.

Je rangerai dans cinq catégories ces modifications diverses, et les désignant sous la dénomination commune *d'atrophie*, je les qualifierai de :

Cupuliforme.
Sulciforme.
Cuspidienne.
en Hache.
d'Hutchinson.

La première, de beaucoup la plus fréquente, atteint surtout les incisives de la deuxième dentition. Elle est constituée par de petites dépressions superficielles, rarement isolées, presque toujours disposées sur la couronne en lignes horizontales.

La seconde apparaît sous la forme de sillons parallèles, dans la même région que la précédente.

*L'atrophie cuspidienne* frappe toutes les dents, mais surtout les premières molaires et les canines permanentes. Par elle, la couronne se trouve divisées en deux parties inégales et très distinctes l'une de l'autre. La plus éloignée de la gencive, amoindrie dans toutes ses dimensions, irrégulière, avec des cuspides acérées, semble enchassée dans l'autre.

Ces trois modalités atrophiques sont fréquemment réunies sur une même dent.

Elles résultent de modifications pathologiques de l'ivoire et de l'émail. Ce dernier toujours aminci ; parfois, fait complétement défaut.

La carie est la conséquence fréquente de cette absence de l'émail.

Les deux dernières variétés de l'atrophie ne sont pas primitives comme les précédentes et ne datent pas, du moins avec leur forme typique, de la période

intra-alvéolaire ; elles ne se montrent que consécutivement à l'éruption des dents, et sont le résultat de la carie ou de l'usure en des régions prédisposées à les subir par une altération congénitale de l'émail.

Celle en hache n'existe que sur les incisives supérieures. Durant une certaine période, la partie la plus voisine de la gencive est seule érodée, et comme le tranchant est intact, on a l'apparence d'un fer de hache.

Dans l'atrophie d'Hutchinson, c'est au contraire la partie centrale du bord libre des incisives, qui a été rapidement usée à cause de l'amincissement qu'y avaient déterminé de nombreuses cupules, au moment des dépôt de l'émail. Il en résulte une encoche de profondeur variable ; triangulaire ou en croissant.

L'atrophie peut atteindre toutes les dents, à l'exception des secondes et troisièmes molaires, et des prémolaires permanentes.

Son existence chez des sujets atteints de syphilis héréditaire, son évolution qui coïncide avec la période d'activité de cette maladie, m'engage à la considérer, suivant la remarque, déjà faite par M. J. Hutchinson pour la variété qu'il a décrite, comme étant sous sa dépendance, et m'autorise à repousser, si non d'une manière absolue, du moins dans l'immense majorité des cas, toute autre origine. En effet, les pyrexies de l'enfance, dont on a parlé, n'apparaissent en général qu'après la seconde année, alors que la formation de la dent est achevée.

Pour ce qui est des convulsions éclamptiques, aux quelles M. Magitot et ses élèves, font jouer un rôle exclusif dans cette étiologie, je les repousse d'une manière générale ; parce que chez la plupart des sujets, l'altération s'est produite ou a débuté durant la vie intra-utérine, dont les accidents nécropathiques sont encore inconnus ; parce que la hauteur et la profondeur des lésions, si bien expliquées par la durée et l'intensité d'action de la syphilis héréditaire, sont inconciliables avec le temps, relativement très court, des accès éclamptiques. Enfin, parce que les deux dernières molaires ne sont jamais atteintes. Immunité qui s'explique aisément par l'étiologie que je soutiens ; l'activité de la syphilis étant éteinte dans la période où ses dents se développent ; tandis qu'elle est en contradiction avec l'opinion de M. Magitot, car les convulsions sont loin d'être rares à cette époque de la vie infantile.

Ainsi la syphilis héréditaire se révèle d'une part par des éruptions cutanées, une affection desquamative de la langue et des altérations viscérales ; de l'autre, par des cicatrices cutanées et une atrophie particulière du système dentaire. Grâce à ces signes, elle n'échappera que dans un nombre de cas restreint.

Je vais maintenant examiner l'affection du squelette, qui par une série d'étapes conduit au rachitis, et qui coïncidant d'une manière à peu près constante avec les marques que je viens de faire connaître, doit être mise sous la dépendance de la syphilis héréditaire.

Cette affection est essentiellement systématique, et, si je puis ainsi dire, chronologique. Elle se présente sous des formes déterminées et distinctes aux diverses périodes de la vie infantile ; mais chez les différents sujets, sa physionomie reste invariable pour une même époque.

Elle atteint, suivant un ordre constant, les pièces du squelette et pour chaque os, les mêmes régions. Ses variétés sont très nombreuses, mais elles peuvent être rangées sous trois types principaux, qui correspondent à trois époques successives de l'enfance ; ce sont, par ordre de date :

*Celui des ostéophytes durs.*
*Celui de l'atrophie gélatiniforme.*
*Celui du tissu spongoïde.*

L'altération du premier type se présente dans toute sa pureté chez les fœtus mort-nés, les avortons, et dans les premières semaines de la vie extra-utérine. Elle consiste essentiellement en des couches ossiformes nouvelles, véritables

ostéophytes qui se développent à la périphérie des os longs et plats ; et qui affectent une disposition tout-à-fait caractéristique sur la moitié inférieure de l'humérus notamment en arrière, et à la face interne du tibia. Ces couches nouvelles, un peu moins dures que l'os normal, s'en distinguent surtout par leur teinte et par la direction de leurs trabécules, qui sont perpendiculaires à l'axe diaphysaire. En même temps, dans beaucoup de cas, il existe au voisinage de l'épiphyte, sur une épaisseur variable, mais qui dépasse rarement un ou deux millimètres, une substance crayeuse, plus friable que le reste de l'os, que je qualifie de *chondro-calcaire*, parcequ'elle n'est autre chose que le tissu cartilagineux infiltré de sels calcaires.

Dans l'altération du second type, on trouve les deux modifications précédentes de la diaphyse avec cette légère restriction, que les ostéophytes ont une dureté moins grande ; mais, en outre, et c'est là ce qui la caractérise, certaines portions, en général très circonscrites, de l'os sont remplacées par un tissu mou, aqueux, transparent, de nuances diverses, souvent jaune maïs ou sucre d'orge, ayant l'apparence d'une gelée. C'est à elle que se rattachent les brisures juxta épiphysaires et la pseudo-paralysie syphilitique.

L'altération du troisième type est caractérisée par le tissu que M. Jules Guérin a qualifié de spongoïde ; élément nouveau et que l'on ne recontre en aucune autre circonstance. Il constitue les ostéophytes péridiaphysaires, et a pour siège de prédilection le voisinage de l'épiphyse, d'où il pénètre, sous forme de bourgeons, dans la couche chondroïde du cartilage, devenue exubérante. En même temps l'os tout entier a perdu sa dureté, et se laisse aisément couper à l'aide d'un scalpel, qui est du à sa décalcification et au développement anormal de l'élément médullaire. Ces modifications histologiques ont pour conséquences : les tuméfactions des extrémités des os, qui sont comme noueuses, la déviation des surfaces articulaires, la déformation des diaphyses et leur fracture.

Sous des influences qu'il serait actuellement malaisé de déterminer, le mal débute non d'une manière nécessaire par la première forme, mais par l'une quelconque d'entr'elles. Il s'y arrête, ou bien, au contraire, il parcourt tous les étages ultérieurs. Et quand il a atteint sa période ultime, quelle que soit, à ce moment, sa violence, quels qu'aient été les désordres accomplis jusque là, il se manifeste spontanément une tendance vers la guérison, qui peut ne laisser après elle aucune trace des troubles antérieurs, mais qui, d'autres fois, fixe irrémédiablement les déformations qui s'étaient produites.

Toutes les parties du squelette peuvent être atteintes comme les os longs, en même temps, et suivant des modalités identiques. Je ne m'occuperai que du crâne. Il subit l'atrophie gélatiniforme qui, de la région sous périostique, arrive parfois jusque sous la dure-mère et amène la perforation complète de sa paroi. Plus tard il se couvre d'ostéophytes, qui ont pour siège de prédilection les angles péribregmatiques du frontal et des pariétaux, d'où ils s'étendent souvent aux deux tiers antéro-latéraux de sa surface. Il en résulte habituellement une déformation typique, qui m'a fait qualifier de *natiformes* les crânes atteints de la sorte.

Durant la période spongoïde et du ramollissement osseux, tandis que les régions antérieures s'épaississent comme il vient d'être dit ; en arrière, les circonvolutions cérébrales usent la paroi et même la perforent. C'est le *crâniotabes* d'Elsaëesser.

Après cette étude morphologique, si l'on aborde celle des détails de structure on voit, que ces lésions sont d'importance très différente, que l'atrophie gélatiniforme, la décalcification et la médullisation, n'occupent que la seconde place, puis qu'elles consistent : la première en une fonte des éléments osseux auquels se substitue un réseau fibrillaire ; la seconde en la résorption des sels calcaires ; et la troisième en la prédominance des parties molles sur les dures ;

tous phénomènes qui peuvent se produire dans d'autres circonstances pathologiques ; au contraire, que les ostéophytes, qui marquent les différentes périodes et les relient entr'elles, constituent un fait propre caractéristique et d'ordre vraiment spécifique. L'on ne trouve plus, dans leur tissu, ainsi que dans l'os normal, des canaux de Havers, courant dans une direction parallèle au grand axe de la diaphyse avec leurs systèmes de lamelles concentriques et leurs ostéoplastes aux canalicules multiples. Ils sont remplacés par de larges espaces, disposés perpendiculairement à l'axe de l'os, occupés par un réseau, conjonctif, des vaisseaux et de rares médullocelles, et limités par les trabécules ossiformes, constellées de corpuscules irréguliers, semblables à ceux du tissu conjonctif, avec des angles, d'où partent des prolongements fibrillaires, qui s'anastomosent et forment ainsi dans cette substance fondamentale morbide, un réseau dont la densité, va croissant avec les progrès du mal et atteint son maximum dans le tissu spongoïde.

Dans ces production nouvelles, l'élément anatomique et fondamental est donc toujours le même, aux différentes périodes de l'évolution morbide. Ce qui varie et donne aux ostéophytes des différents types, une physionomie particulière, c'est le nombre de ces éléments, c'est la dureté de la substance fondamentale où ils sont enchassés, c'est la largeur des espaces qui séparent les trabécules ; toutes particularités de valeur minime, et qui laissent subsister, entre les produits morbides, la parenté que nous montre le microscope. Cette parenté d'ailleurs, se révèle chez certains malades, d'une manière saisissante, par la disposition stratigraphique, sur le même os, de couches de types différents, rarement intactes, il est vrai, et dans l'état où elles s'étaient originairement déposées, chacune ayant ses caractères propres, mais retenant quelque chose des précédentes.

Ces altérations osseuses, ne différent donc qu'en apparence. En réalité, elles font partie d'un même processus morbide, c'est-à-dire, que leur cause est la même ; leur physionomie diverse est une question d'âge, voilà tout. Si donc l'on peut découvrir l'origine de l'une d'elles, il faudra l'attribuer aux autres.

Eh bien ! j'affirme, pour l'avoir constaté plusieurs centaines de fois, que les deux premiers types se rencontrent exclusivement chez des sujets présentant, dans les viscéres ou sur la peau, quelque marque incontestable de la syphilis héréditaire. D'où il faut induire que le troisième et dernier type, est lui aussi sous la dépendance de cette maladie. Mais ce n'est pas tout ; chez un nombre considérable de sujets, enfants, adolescents ou adultes, actuellement atteints de l'affection chrondro-spongoïde, ou présentant les déformations qui en sont la conséquence ; on trouve soit des manifestations actuelles de la syphilis héréditaire, soit les stigmates que j'ai fait connaître.

Si maintenant l'on se reporte aux descriptions classiques du rachitis l'on voit que cette affection des os de l'enfance ne différe en rien de la syphilis osseuse arrivée à sa troisième période. Dans l'un et l'autre cas, c'est le tissu spongoïde qui sert de caractéristique ; car, sans lui, il peut y avoir de l'ostéomalacie, mais il n'y a pas de rachitis.

Donc le rachitis est engendré par la syphilis héréditaire.

Est-il suprenant de voir un mal aussi particulier, aussi typique, reconnaître cette cause unique et puissante au lieu de celles que jusqu'ici on lui a attribuées ? Non certes ! surtout quand on jette un coup d'œil sur cette étiologie.

A la tête des causes prédisposantes, on met l'influence des générateurs. Si leurs enfants sont rachitiques, c'est qu'ils l'étaient eux-mêmes ; ou bien tuberculeux, scrofuleux, épuisés trop jeunes, ou trop vieux, ivrognes, sanguins ou bilieux ; et d'après Van Swieten mous, oisifs, adonnés aux plaisirs de la table, aimant les plats gras et sucrés, mangeant peu de pain, buvant des vins trop doux, se servant d'eau chaude, épuisés par des maladies chroniques ou par l'amour.

Puis viennent les affections mêmes de l'enfance : la toux, la diarrhée, toutes les fièvres. On fait jouer un rôle considérable aux climats froids et humides, aux habitations basses, imparfaitement aérées et éclairées.

Les causes occasionnelles ne sont pas moins nombreuses. On a surtout accusé l'insuffisance et la mauvaise qualité des aliments. Guersent s'en prenait au lait et aux farineux. M. Jules Guérin, après avoir incriminé l'allaitement trop prolongé, voit au contraire aujourd'hui l'origine du mal, dans une alimentation trop animalisée. Trousseau a soutenu cette manière de voir. L'insuffisance des sels calcaires a joué un rôle non moins important que l'alimentation dans cette étiologie ; et M. Seemann pense que, chez les rachitiques, il existe un trouble digestif essentiellement constitué par la production insuffisante d'acide chlorhydrique, d'où l'absorption insuffisante de sels de chaux par le tube digestif. Pour Broca et M. Gamba, le rachitis n'est pas une maladie spéciale, mais bien la conséquence de toutes les causes qui troublent la nutrition pendant la période de croissance du jeune âge. Comme on le voit, tout est invoqué mêlé, confondu, dans cette étiologie si compréhensive, si disparate, si contradictoire.

Et en vérité rien n'est moins fait pour convaincre de l'efficacité de l'une de ces causes, que leur liste aussi longue que banale.

On comprend à la rigeur, que la plupart d'entr'elles, par leur action débilitante, aient rendu moins active la nutrition des divers tissus, et notamment celle du squelette ; mais de là à faire du tissu spongoïde et des ostéophytes, il y a loin. D'ailleurs ne sait-on pas qu'il y a des rachitiques gros et gras ; et par contre que beaucoup d'enfants malingres, chétifs et en proie à des diathèses comsomptives, ne sont jamais atteints de rachitis.

Il y a donc un vide considérable dans l'étiologie classique du rachitis. Ce vide je crois l'avoir comblé en faisant intervenir la syphilis héréditaire ; et en lui attribuant d'une manière exclusive la genèse de cette altération des os.

Il y a déjà longtemps que cela avait été dit ; tout ne l'a-t-il pas été ; mais à ma connaissance, cela n'avait pas été démontré à l'aide de preuves empruntées à la clinique et à l'anatomie pathologique, comme celles que je viens de présenter au Congrès.

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## *De la Syphilis comme Cause de Rachitisme et de Malformation dentaire.*

Dr. Bouchut, Paris.

Par suite de l'état cachectique qui l'accompagne si habituellement, la syphilis infantile, héréditaire, peut être une cause de rachitisme et de malformation dentaire. C'est l'opinion d'un grand nombre de médecins. Mais le rachitisme reconnaît bien d'autres causes. Leur nature mérite d'être indiquée avec soin ; et, puisque la question est mise à l'ordre du jour du Congrès International de Londres, je vais dire l'opinion à laquelle je me suis arrêtée.

Je parlerai—

1° *Des malformations dentaires cachectiques* attribuées à la syphilis.

2° *Du rachitisme* considéré comme la manifestation constante d'une diathèse syphilitique.

1° Les malformations dentaires d'un certain ordre sont depuis les travaux d'Hutchinson assez souvent attribuées à la syphilis. Mais s'il a donné la description de ces altérations, il n'a peut-être pas suffisamment établi le diagnostic différentiel entre cette malformation et les autres, de sorte que les

lecteurs ont pu supposer que sa description avait un caractère général s'appliquant à toutes les malformations de la seconde dentition. Des observations analogues ont été publiées par quelques autres médecins. On a depuis lors considéré presque tous les sujets dont les incisives permanentes étaient piquées, rougies ou échancrées comme atteints de syphilis héréditaire.

C'est là une affirmation de séméiologie et de diagnostic qui est trop absolue, et sur laquelle il importe de faire des réserves.

Magitot a déjà fait remarquer que les dents incisives permanentes, offrant l'altération décrite par Hutchinson, n'appartenaient pas à des sujets syphilitiques. W. Nicati en a dit autant, et, dans un travail trés-intéressant, a montré que certaines malformations des incisives permanentes étaient dues au rachitisme plutôt qu'à la syphilis et qu'il fallait établir un diagnostic différentiel entre les unes et les autres. Cela est à mes yeux un pas vers la vérité, mais ce n'est pas encore la vérité tout entière.

J'ai observé très-souvent les altérations et les malformations des dents de première et de seconde dentition chez les enfants. Mes conclusions différent totalement de celles d'Hutchinson et se rapprochent beaucoup au contraire de celles de W. Nicati. Je vais m'expliquer à ce sujet, lorsque j'aurai dit quelques mots des altérations des premières dents, dites *dents de lait.*

Ces dents s'altèrent avant ou après leur sortie de la gencive.

Avant leur sortie, elles peuvent s'altérer dans leur germe ou dans leur évolution, si l'enfant a des convulsions, ce qu'a signalé Magitot, s'il a eu du rachitisme congénital, enfin s'il a eu de *l'ostéoporose ou rachitisme acquis* par suite d'une dyspepsie prolongée, d'une entérite grave ou d'une autre maladie. Dans ces différents cas les premières dents sortent tard, sortent mal, sortent irrégulièrement, sont jaunes, altérées dans leur émail et tombent quelquefois prématurement.

Après leur sortie des gencives les dents de lait s'altérent, si les enfants sont gravement malades, s'ils ont une entérite prolongée avec acescence buccale. Elles noircissent, se corrodent, au niveau du collet, se creusent, et se cassent au bord de la gencive.

Si les enfants ont une stomatite ulcéreuse qui ronge la gencive et la détruit sur son bord libre dans une plus ou moins grande hauteur, les dents s'allongent et on en voit la racine dans une assez grande étendue.

Mais les malformations les plus curieuses sont celles de la seconde dentition. Elles portent principalement sur les incisives permanentes. Elles résultent d'une maladie de la première enfance qui a modifié la nutrition des germes dentaires.

Quelle est cette maladie ?

Cette maladie est-elle diathésique et donne-t-elle une forme particulière à la malformation des dents ?

D'après ce que j'ai vu, il n'y a pas de maladie spéciale diathésique qui produise la malformation spécifique des incisives permanentes. Toute maladie grave et prolongée de la première enfance produit ce résultat. La syphilis pas plus que le rachitisme ou l'entérite chronique n'a d'action particulière. Oui, la syphilis héréditaire, oui, le rachitisme déterminent la malformation des incisives permanentes, mais ce n'est pas comme syphilis ou comme rachitisme ; c'est comme état cachectique. J'ai donné pour preuve les mêmes malformations observées par moi à la suite de l'entérite chronique et des suppurations prolongées du premier âge. Ces altérations sont des *malformations marastiques ou cachectiques,* mais pas autre chose.

C'est donc comme état cachectique modifiant la nutrition des germes dentaires que la syphilis héréditaire et le rachitisme déterminent l'altération des dents permanentes, et non comme maladies diathésiques ; à cet égard elles se confondent avec toutes les autres maladies cachectiques du premier âge.

Il se passe dans les germes dentaires ce qui se passe dans les ongles, dans les cheveux et dans les os. La nutrition de ces éléments s'altére dans toutes les maladies. Les ongles, après s'être amincis un moment, s'épaississent lorsque l'enfant va mieux et présentent ces sillons que Beau a fait connaître dans la dyspepsie. Les cheveux sont secs, pâlissent, deviennent langoureux, étiques, et, comme on le dit, les enfants ont mauvais poil. Les os se gonflent au niveau des épiphyses, se raréfient et offrent de *l'ostéoporose.* Ils sont plus mous, se courbent facilement, ne peuvent plus momentanément porter les enfants, qui veulent rester au lit après avoir commencé déjà de marcher. C'est le rachitisme.

Toutes ces altérations analogues sont de même nature et dépendent de *l'état marastique, cachectique, cacochymique et cacothrepsique,* comme on dit à présent.

Les germes dentaires de la seconde dentition subissent la loi de généralité et ils deviennent cachectiques avec le reste de l'économie, quelle que soit la cause du trouble survenu dans la nutrition générale des sujets. Ils souffrent quand tout souffre, et les dents qui en sortent sont malades, altérées dans leur émail et dans leur ivoire, quand la première enfance a été très longtemps et très gravement maladive.

D'après ce que j'ai observé en dehors de la syphilis et du rachitisme proprement dit, on trouve les malformations dentaires signalées par Hutchinson et par Nicati comme appartenant à ces deux maladies. Ainsi pour ne citer que quelques faits je raconterai l'observation suivante relative à un jeune garçon dont je connais la famille et que j'ai suivi depuis dix ans. La malformation de ses dents incisives ressemble à la malformation décrite comme syphilitique par Hutchinson, et cependant elle n'est que cachectique.

X., fils de M. X., rue des jeûneurs, a failli mourir au moment de sa naissance, il y a 10 ans, d'une hémorrhagie nasale pour laquelle j'ai été appelé en consultation. Il est resté exsangu, et le perchlorure de fer employé comme hémostatique lui a laissé un coryza chronique qui dure encore, et qui gêne la respiration. Cet enfant a été malade toute sa première enfance, et sans rachitisme ou syphilis ; il est pâle, très maigre et a fort mauvaise mine malgré un assez bon état de santé. Les dents sont blanches ; il a quatre incisives écartées, et les deux moyennes échancrées au bord tranchant sans altération de l'émail. Il en est de même à la machoire inférieure. Il a ses premières molaires, mais bien qu'il ait 10 ans, il n'a pas de canines. Sur une fille de 12 ans, nommée Kertzel, au 24 de Ste. Catherine, sortie le 7 février, guérie d'une scarlatine, qui n'a pas eu de syphilis ni de rachitisme, et qui a été maladive dans le premier âge, on trouve les deux incisives médianes supérieures, intactes dans les cinq sixièmes supérieurs, mais près du bord libre un sillon transversal profond, anfractueux qui amincit tout à coup la dent et forme comme un pas d'escalier. Les autres dents sont saines.

Dans un autre cas, encore à l'hôpital en ce moment, il s'agit d'une jeune fille, L., de 11 ans, couchée au No. 27 de Ste. Catherine, entrée le 30 Decembre 1879, pour un vertige épileptique. Elle est petite, maigre, peu développée pour son âge, n'a jamais eu de signe de syphilis héréditaire après la naissance ni postérieurement. Elle a toujours été maladive, et dans la première enfance a eu de fréquentes diarrhées. Ses incisives supérieures présentent des érosions pointillées, c'est-à-dire, des piqûres noirâtres au niveau desquelles l'émail est a peu près détruit, en même temps que l'ivoire est altéré. Ces ponctuations sont au nombre de quatre sur chaque incisive, et elles sont placées sur une ligne horizontale près du bord tranchant qui est fortement ébréché par de fines dentelures. Au dessus de cette ligne de piqûres de l'émail se trouve un relief transversal formant comme une marche d'escalier.

Ces lésions ressemblent à celles que W. Nicati attribue au rachitisme, et cependant l'enfant n'est pas rachitique, n'a pas les côtes déformées ni la tête

volumineuse. Elle ne l'a pas été. Elle n'a été que maladive et cachectique dans la première enfance.

Sur une autre fille (V.) de 14 ans, non formée, couchée au No. 5 de Ste. Catherine et entrée le 30 janvier 1879, pour des crises nerveuses hystériformes, il y a aussi une malformation considérable des dents. Cette fille a été maladive dans toute la première enfance, a eu des convulsions pendant longtemps jusqu'à 10 ans et puis sa santé est devenue plus robuste. Aujourd'hui ses quatre incisives sont étroites en haut et plus larges en bas, échancrées, concaves et mousses à leur bord libre. L'émail est au dessus rugueux, piqué de points noirâtres et offre un sillon transversal déprimé. Les incisives inférieures sont également échancrées et concaves au bord libre qui est mousse. L'émail est altéré, et, au dessous du bord, offre un sillon très-déprimé, noirâtre. Les canines coniques et petites présentent également une altération de l'émail près de la pointe.

L'autre enfant, qui est un garçon, présenté à l'hôpital (30 janvier 1879), a 19 mois. Celui-là offre une malformation des dents de lait qui sont au nombre de huit. La première est sortie à 7 mois, âge réglementaire. Cet enfant ne marche pas, ses membres inférieures sont grêles, il a les symphises chondro-costales tuméfiées de façon à offrir le chapelet rachitique. Il n'est pas sujet à la diarrhée mais il tousse habituellement ; il a la poitrine *grasse*, et remplie de râles muqueux. Sa mère n'est pas syphilitique, on ne sait rien du père, et lui-même n'a pas offert de trace de syphilis héréditaire. Chez lui les deux dents incisives médianes supérieures sont écartées, jaunâtres, courtes, largement échancrées et mousses à leur bord libre. Les incisives supérieures latérales sont collées aux autres, coniques et jaunâtres. Les incisives médianes inférieures sont également échancrées ; mais ici elles se touchent et les incisives latérales sont plus courtes, coniques et jaunâtres. L'émail n'est pas piqué mais il est un peu altéré dans sa couleur.

Ici, la malformation a les caractères de ce que l'on a dit être la syphilis, mais elle est de nature cachectique par rachitisme.

Une autre petite fille de 4 ans, au No. 31 de ma salle Ste. Catherine, hémiplégique, à la suite de convulsions répétées, survenues à l'âge de 16 mois, peut cependant marcher. Elle a quinze dents, très mal formées et mal placées. Elle n'a ni rachitisme, ni syphilis ; elle est cachectique. A la mâchoire supérieure, elle a deux incisives droites obliquement placées ; la médiane assez large et jaune, et celle qui est à côté conique, petite et serrée contre la première. Les deux incisives de gauche sont également obliques, et forment avec celles du coté opposé un V ouvert par en bas. Elles sont également de mauvaise nature, mais elles ne sont ni échancrées, ni piquées, ni dentelées. Les dents incisives inférieures sont également mal rangées, jaunes et très-petites.

Ici, pas de syphilis ni de rachitisme, mais bien un état maladif, prolongé, suite de convulsions et de paralysie. C'est un de ces cas que l'on pourrait citer à l'appui de l'opinion de Magitot, qui a dit que des troubles trophiques de la nutrition des germes dentaires pouvaient résulter de l'état convulsif.

Dans un autre cas, que je pourrais citer, et qui est relatif à l'un de mes élèves, interne autrefois à l'Hôpital Ste. Eugénie, et aujourd'hui confrère parisien, les dents incisives supérieures sont piquées, pointillées et transversalement sillonnées par une dépression de l'ivoire. Il n'y a eu chez lui ni syphilis héréditaire, ni rachitisme, mais seulement un état cachectique prolongé dû à une première enfance maladive.

A l'hôpital des enfants, dans mon service, et à la consultation publique, ces faits de malformation dentaire sont très-nombreux. Ils sont plus rares dans la première dentition que dans la seconde et c'est principalement vers 10 à 15 ans que je les ai observés. J'en ai recueilli 114 exemples.

Tout étant bien considéré, après une analyse minutieuse des causes et des phénomènes de ces 114 exemples de malformations, il m'a semblé que si on les observait après la syphilis héréditaire c'était moins comme état syphilitique que comme conséquence d'un état cachectique ou marastique. En effet, sur ces 114 cas, je n'en ai trouvé que huit qui fussent syphilitiques. J'en dirai autant des malformations exclusivement attribuées au rachitisme.

D'ailleurs la recherche de ces causes est extrémement difficile et délicate. Sauf quelques cas où les parents avouent leur syphilis et celle de leurs enfants, et ceux dans lesquels le médecin a suivi l'enfant pendant plusieurs années, depuis sa naissance, on en est réduit aux conjectures et aux hypothèses les moins acceptables.

Que de fois n'ai-je pas vu considérer comme dents syphilitiques des dents appartenant à des enfants sur le passé des quels on n'avait aucun renseignement précis ; à des enfants non syphilitiques mais dont le père avait eu autrefois la syphilis ; ou, enfin, à des enfants dont on ne connaissait pas les antécédents, mais qu'on jugeait pouvoir être syphilitiques, parce que la plupart des pères peuvent avoir eu la syphilis. Il en est de la recherche des influences syphilitiques sur les dents, comme de la recherche de *l'arthritis* dans la production de certaines affections cutanées ou viscérales dites arthritiques. Avec une forte idée préconçue on trouve tout ce qu'on désire.

Ce n'est pas ainsi que doit progresser la science. Il faut en médecine bannir toutes les hypothèses, et n'y introduire que des faits certains et bien observés. Tout ce qui est vague doit être mis de côté, ou du moins ne doit être accepté qu'avec réserve.

Ainsi en est-il des dents réputées syphilitiques et rachitiques. Cependant ce qu'on a dit des dernières est bien plus acceptable que ce qui a été dit des premières. Ces altérations sont un effet de l'état cachectique et pas autre chose. Cachexie syphilitique, cachexie rachitique, cachexie scrofuleuse, cachexie intestinale ou pulmonaire, toutes déterminent *l'ostéoporose*, ralentissent le mouvement de nutrition du tissu osseux et ainsi engendrent les malformations dentaires et les déformations du squelette en général.

2° *Ramollissement des os par état cachectique.*—Tout ce que je viens de dire de ces malformations dentaires s'applique au ramollissement des os longs, au gonflement de leurs extrémités, à leur déformation, et à tout ce que les médecins appellent *rachitisme*. En effet, chez les jeunes enfants une maladie aigue prolongée, une fièvre éruptive, suivie de complications sérieuses, une entérite chronique, arrêtant l'évolution du tissu osseux, ramollissent les os dont les extrémités se gonflent, et dont la diaphyse se raréfie, enfin, amènent les difformités du squelette avec ou sans fracture consécutive. On produit ces déformations chez les oiseaux en supprimant tout phosphate de chaux de leur nourriture ; et chez de jeunes chiens d'une même portée on en fait autant par l'alimentation prématurée et par la claustration dans un endroit obscur. La maladie et l'état cachectique sont les causes de cette altération du tissu osseux.

Sans doute la syphilis héréditaire congénitale, ou développée peu après la naissance, peut amener l'ostéoporose ou rachitisme, mais au même titre que tout autre état de souffrance grave, que toute maladie chronique de l'intestin, de l'estomac, des poumons et des articulations, survenue dans les deux premières années de la vie. C'est l'état cachectique, quelle qu'en soit la cause, qu'il dépende d'une entérite chronique, d'une suppuration prolongée, de la scrofule, de la syphilis, qui en est la cause, en troublant la régularité du mouvement de la nutrition osseuse. La spécificité de la cause, ou, si l'on veut, la diathèse, n'y est pour rien ; c'est l'affaiblissement des jeunes sujets qui est le point de départ de toutes ces altérations.

En résumé, les malformations dentaires, l'ostéomalacie, le rachitisme, peuvent

être parfois le résultat de la syphilis infantile, mais, dans le plus grand nombre des cas, ces lésions n'ont rien de syphilitique, et elles dépendent de l'état cachectique ou marastique des jeunes enfants.

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## *Die Syphilis als eine Ursache der Rachitis.*

Dr. M. Kassowitz, Vienna.

Ich habe bereits in meiner im Jahre 1876 erschienenen Monographie über die Vererbung der Syphilis (S. 135) in Kürze diese Frage erörtert. Seitdem habe ich aber gerade die Rachitis und die krankhaften Veränderungen des Knochensystems bei der hereditären Syphilis, sowie überhaupt die normale und pathologische Knochenbildung einem sehr eingehenden Studium unterzogen, und ich bin eben im Begriffe, die Resultate dieser Unternehmungen der Oeffentlichkeit zu übergeben.*

Da aber die Section für Kinderkrankheiten des internationalen medizinischen Congresses diese Frage aufgeworfen hat, so stehe ich nicht an, auch jetzt schon meinen Standpunkt in derselben in möglichster Kürze zu präcisiren.

Nach meinen Untersuchungen liegt *das Wesen des rachitirten Processes* in einem chronischen entzündlichen Vorgange, welcher an den Appositionsstellen der wachsenden Foetalen und kindlichen Knochen seinen Ausgang nimmt, und sich hauptsächlich in einer krankhaft gesteigerten Neubildung von Blutgefässen und in einer bedeutenden Hyperämie sämmtlicher Gefässe in jenen Teilen des Knochensystems äussert, in welchen aber die Auflagerung neuer Knochenteile erfolgt, also hauptsächlich in der Knorpelwucherungsschichte der Chondoepiphysen, im Perichondrium und Periost, und endlich in der Nahtsubstanz der Schädelknochen.

Diese übermässige Vascularisation und Hyperämie des knochenbildenden Gewebe hat nun notwendiger Weise eine krankhafte Modification der Knochenbildung in ihrem Gefolge. Es haben nämlich meine Untersuchungen schon für die *normale Ossification* ergeben, dass die Erscheinungen der Knochenbildung und Verkalkung, sowie auch die Knocheneinschmelzung und die Resorption der Kalksalze in hohem Grade abhängig sind von den Blutgefässen des Knochens und der knochenbildenden Gewebe, und ich will diese Abhängigkeitsverhältnisse für unsere Zwecke in folgenden Punkten zusammenfassen :—

1.—Die Bildung von Knochengewebe und die Ablagerung von Kalksalzen in das letztere findet niemals in der unmittelbarsten Nachbarschaft der Gefässwände statt, weil die von dem Blutstrome ausgesendete Saftströmung in einem gewissen Umkreise um das Blutgefäss die Ablagerung der Kalksalze verhindert. Eine fortschreitende Ossification ist nur möglich bei nachlassender Saftströmung, wie sie entweder durch allmälige Obliteration der Blutgefässe oder durch die zunehmende Entfernung derselben von dem ossificirenden Gewebe bedingt ist.

2.—Eine experimentell hervorgerufene Verstärkung der Blut- und Saftströmung in einem bestimmten Teile des wachsenden Knochensystems verhindert in deutlich nachweisbarem Maasse die Ablagerung der Kalksalze in den neugebildeten Knochenteilen.

3.—Fertiges und bereits verkalktes Knochengewebe verliert seine Kalksalze

* Ein Teil dieser Arbeit ist bereits unter dem Titel : "*Die normale Ossification und die Erkrankungen des Knochensystems bei Rachitis und hereditärer Syphilis. I. Teil. Normale Ossification,*" bei Braumüller 1881 erschienen. Den zweiten Teil, welcher die Rachitis behandeln wird, bin ich eben im Begriffe zu bearbeiten.

und wird wieder in weiches Markgewebe umgewandelt, wenn es in die lebhaftere Saftströmung eines neugebildeten Blutgefässes geräth, oder wenn ihm ein bereits vorhandenes Blutgefäss nähergerückt wird, oder wenn ein solches aus irgend einem Grunde eine Steigerung seiner Blutfülle und der von ihm ausgehenden Saftströmung erfährt.

Wenn nun die knochenbildenden Gewebe und in weiterer Folge der ganze Knochen von überaus zahlreichen und blutüberfüllten Gefässen durchzogen sind, so wird nicht nur das neugebildete Knochengewebe eine abnorme Beschaffenheit aufweisen und speciell die Ablagerung von Kalksalzen in diesem neugebildeten Gewebe verzögert oder gänzlich verhindert werden, sondern es werden auch vielfache Einschmelzungen des bereits verkalkten und erhärteten Gewebes erfolgen, an die Stelle dieses harten Knochengewebes wird weiches Bildungsgewebe und später wieder unverkalktes und daher wenig resistentes neues Knochengewebe treten, und der ganze Knochen wird nach und nach in der bekannten Weise seiner Starrheit verlustig werden. Auch die für die Rachitis so charakteristische, übermässige Knorpelwucherung lässt sich ganz einfach auf die abnorm vermehrte Säftezufuhr zu dem wachsenden Knorpel zurückführen.

Es liegt auf der Hand, dass diese Auffassung des rachitischen Processes, wenn sie auf richtiger Basis beruht — was ich in meiner ausführlichen Arbeit beweisen zu können glaube — viel besser die gesammten Erscheinungen der Rachitis zu erklären im Stande ist, als die bisherigen Theorien, welche fast ausnahmslos eine mangelhafte Zufuhr oder eine mangelhafte Aufnahme von Kalksalzen in den Organismus zum Ausgangspunkt nehmen. Denn abgesehen davon, dass eine solche mangelhafte Zufuhr nur für eine sehr geringe Anzahl von Fällen wirklich angenommen werden kann, könnte eine solche höchstens die mangelhafte Verkalkung der neugebildeten Knochenteile erklären, dagegen würden die Gründe für alle übrigen so auffälligen Veränderungen im gesammten Knochensystem, für die vermehrte und abnorm beschaffene Knorpelwucherung, für die so bedeutend gesteigerten Einschmelzungsprocesse im Knorpel und Knochen, für die krankhaft *gesteigerte* Auflagerung von periostalem Knochengewebe u. s. w. vollkommen im Dunkeln bleiben.

Wenn nun wirklich ein entzündlicher Process die Grundlage sämmtlicher rachitischer Erscheinungen im Knochensysteme ist, so drängt sich uns sodann die Frage auf: Wodurch wird jener Entzündungsprocess zunächst in den knochenbildenden Geweben und dann im gesammten Knochensystem hervorgerufen? Warum entwickelt sich derselbe gerade nur in den letzten Foetalmonaten und in den ersten Lebensjahren? Und schliesslich, warum ist es gerade das Knochensystem, welches in dieser Lebensperiode in so enormer Häufigkeit einem solchen Entzündungsprocesse anheimfällt?

Knüpfen wir gleich an die letzte Frage an, so müssen wir vor Allem in Erinnerung bringen, dass *das Knochengewebe sich in einem wichtigen Punkte fundamental von allen anderen Geweben des thierischen Körpers unterscheidet*, und gewissermassen ein Unicum unter den Geweben bildet. Alle übrigen Gewebe wachsen nämlich *expansio*, und gleichzeitig in allen ihren Teilen, sie vergrössern ihr Volum dadurch, dass ihre sämmtlichen Gewebebestandteile wachsen und sich vermehren, dass also zwischen den alten immer wieder neue Gewebeteile sich bilden. Eine solche Art des Wachstums ist aber für den starren und unausdehnbaren Knochen durch die physikalische Unmöglichkeit ausgeschlossen, und es ist daher hier eine Volumsvermehrung überhaupt nur möglich durch *Auflagerung neuer Knochenteile auf der Oberfläche des bereits erhärteten Knochens*. Man denke nur, welche ganz enorme Steigerung das Wachstum an jenen Appositionsteilen des Knochens im Vergleiche mit irgend einem Bruchstücke eines in allen seinen Teilen gleichmässig expansio wachsenden Gewebes von gleicher Ausdehnung erleiden muss, namentlich wenn man die Apposition

an einem intensiv wachsenden Knochenende eines langen Extremitätenknochens oder einer Rippe in's Auge fasst. Was bei dem expansiven Wachstum in der ganzen Ausdehnung des Organes geschieht, muss hier in der räumlich so beschränkten Wucherungsschichte des Knorpels geleistet werden.

Daraus resultirt aber auch notwendiger Weise *eine gänzlich abweichende Verteilung in dem Zuflusse der Ernährungssäfte* für die wachsenden Teile des Knochens einerseits und für die übrigen Organe andrerseits. Während also z. B. die foetale oder kindliche Leber in allen ihren Teilen nahezu gleichmässig wächst, und demnach die Ernährungssäfte, welche das Material für das Wachstum liefern, auf dem Wege der Blutgefässe zu allen Teilen des Organs ganz gleichmässig zugeführt werden; muss dieses Ernährungsmaterial, soweit es zum Wachstum verwendet werden soll, fast ausschliesslich zu den Knorpelwucherungsschichten und in etwas geringerem Maasse zu dem Periost hingeleitet werden, während dem ganzen übrigen voluminösen Skeletteil nur jene verhältnissmässig wenig bedeutende Menge des Ernährungsmaterials zukommt, welches zu dem Lebensunterhalte des, eines weiteren Wachstums unfähigen erhärteten Teile des Knochens hinreicht.

Setzen wir nun den Fall, dass in der Säftemasse des foetalen oder kindlichen Organismus irgend ein abnormer Bestandteil circulire, welcher im Stande wäre, *einen krankhaften Reiz auf die Gefässwände* und auf die übrigen von der Saftströmung passirten Gewebe auszuüben, so wird es wol begreiflich sein, dass ein solcher krankhafter Reiz sich an jenen Punkten in ganz unvergleichlich gesteigertem Maasse geltend machen muss, wo eben aus physiologischen Gründen eine so ausserordentlich gesteigerte Menge des Säftematerials fortwährend die Gefässe und die Gewebe passirt; dass also, mit anderen Worten, ein jeder krankhafte Reiz, welcher den Gesammtorganismus des Foetus in den letzten Foetalmonaten und den des Kindes in den ersten Lebensjahren trifft, gerade an den Knochen, und gerade an jenen Knochenenden, welche eine besonders lebhafte Apposition aufweisen, sich ganz besonders geltend machen, und Anlass zu der Entstehung und Erhaltung jenes entzündlichen Zustandes der Gefässe und Gewebe geben muss, welchen wir an jenen Stellen bei der Rachitis *de facto* beobachten.

Wenn wir nun weiter fragen, wodurch jene abnormen Bestandteile und krankhaften Reize innerhalb der Blut- und Säftemasse der Rachitischen entstehen, so müssen wir zunächst die Umstände erwägen, unter denen die Rachitis überhaupt zu Stande kommt, und diese Umstände sind, soweit uns die klinische Beobachtung darüber belehren kann, die folgenden :—

1.—Die Rachitis ist vorwiegend eine Krankheit der Armut. Bei einer sehr sorgfältigen Untersuchung sämmtlicher Kinder *unter* 3 *Jahren*, welche mein Ambulatorium besuchten, ergab sich regelmässig und immer wieder ein Verhältniss von *mehr als* 90 *Rachitischen unter* 100 *Kindern* überhaupt.

2.—Die vorzüglichste Schädlichkeit, welche Rachitis hervorruft, liegt nach meinen Erfahrungen in den *schlechten Wohnungsverhältnissen*. Die Wohnräume der Arbeiter und ärmeren Handwerker, deren Kinder das Contingent für die öffentlichen Ambulatorien abgeben, sind fast immer eng, übervölkert, schlecht oder gar nicht ventilirt, und die Kinder verleben ihre ersten Jahre, insbesondere in der schlechteren Jahreszeit, fast ununterbrochen in einer verdorbenen und durch die menschlichen Ausdünstungen, die Effluvien der Küche, des Waschtroges, der Handwerkstube u. s. w. verunreinigten Athmosphäre.

3.—Die *schlechte Ernährung des Kindes* kommt bei meinem Materiale entschieden erst in zweiter Linie, wenn auch die grosse Bedeutung derselben für die Entstehung der Rachitis keineswegs unterschätzt werden soll. Die Mehrzahl der Kinder wird nämlich hier an der Mutterbrust ernährt, und dieselben zeigen oft trotz ihrer rachitischen Affection einen nicht gerade schlechten Ernäh-

rungszustand. Dagegen ist es zweifellos, dass die meisten Kinder bei nachlässiger künstlicher Ernährung, oder solche, die von kümmerlich genährten Müttern gesäugt werden, ganz besonders häufig den schweren Formen der Rachitis verfallen.

4.—*Eine jede den allgemeinen Ernährungszustand des Kindes in den ersten beiden Lebensjahren beeinträchtigende Erkrankung*, insbesondere eine jede chronische Affection der Respirations- und Verdauungsorgane begünstigt in hohem Grade die Entstehung der Rachitis, und bewirkt in einem noch viel höheren Grade die Steigerung einer bereits vorhandenen rachitischen Affektion.

5.—Die Rachitis beginnt viel häufiger, als man bisher angenommen hat, *bereits in den letzten Monaten der intrauterinen Entwicklung.* Die Totgeborenen und Frühverstorbenen der Gebärhäuser und der Findelanstalt ergaben mir bei der makroskopischen und insbesondere bei der histologischen Untersuchung der rasch wachsenden Knochenenden in einem sehr bedeutenden Percentverhältnisse bereits sehr deutlich nachweisbare rachitische Veränderungen. Ebenso konnte ich auch bei den lebenden Kindern ausserordentlich häufig schon in den ersten Wochen so greifbare Veränderungen im Knochensysteme nachweisen, dass man deren Entstehung unbedingt in die intrauterine Periode verlegen muss. In allen diesen Fällen wird man also notwendiger Weise zu der Annahme gedrängt, dass hier die krankhaften Reize aus der Säftemasse der Mutter in jene des Foetus übergegangen sind.

Es ist also nicht etwa eine einzige bestimmte Schädlichkeit, welche die Rachitis hervorruft, sondern offenbar ist *eine jede, durch welche Ursache immer hervorgerufene krankhafte Beschaffenheit der Säftemasse des Foetus und des Kindes in der Periode des energischesten Längenwachstums* im Stande, an jenen Stellen, wodurch die energisch gesteigerte Knochenapposition ein vermehrter Affluxus und demnach eine Potenzirung jener Schädlichkeiten erfolgt, jenen entzündlichen Vorgang herbeizuführen, welcher die rachitischen Veränderungen des Knorpel- und Knochengewebes und in weiterer Folge die rachitische Erweichung des Skelettes zur Folge hat.

Nun begreift es sich auch ganz leicht, dass eine *Heilung des Rachitis* nicht möglich ist durch eine künstliche Zufuhr von Kalksalzen, weil diese, in noch so grosser Menge zugeführt, sich niemals auf jenem hyperämischen und entzündlich afficirten Boden ablagern können ; wol aber durch die Entfernung und Bekämpfung jener Schädlichkeiten, welche die entferntere Ursache des entzündlichen Reizungszustandes an den Appositionsstellen sind ; ferner durch alle jene hygienischen Maassregeln und Heilbehelfe, welche die Lebensverhältnisse und den allgemeinen Ernährungs- und Gesundheitszustand des Kindes verbessern ; und endlich auf dem Wege der Naturselbstheilung, wenn nämlich nach Ablauf der ersten Lebensjahre die Energie des Längewachstums bedeutend herabsinkt, und dadurch auch die Verteilung der Ernährungssäfte in den einzelnen Skeletteilen eine gleichmässigere und wenigstens annähernd jenem Verhältnisse ähnlich wird, welches allen übrigen Organen des menschlichen Körpers *während der ganzen Wachstumsperiode* zugeschrieben werden muss.

Wenn diese meine Theorie der Rachitis die richtige ist, dann ergiebt sich *das Verhältniss der Syphilis hereditaria zu der Rachitis* ganz von selbst. Hier tritt uns eben eine von jenen supponirten schädlichen Materien, welche in der Blut- und Säftemasse des Foetus und des rachitischen Kindes circuliren sollen, in einer concreten Form entgegen. Wie immer man sich auch das Syphilitische Virus vorstellen mag, so kann es doch kaum zweifelhaft sein, dass dasselbe in der Blut- und Säftemasse verteilt ist, und dass es im Stande ist, in den verbindensten Organen und Geweben durch seinen Reiz Entzündungsprocesse hervorzurufen. Dass dieses Gift auch in dem Knochensysteme Entzündungs- und

Zerstörungsprocesse hervorruft, und dass dieselben beim Foetus und beim Neugeborenen speciell an jenen Knochenenden hervortreten, welche ein lebhaftes Längenwachstum aufweisen, ist durch die Untersuchungen der letzten Jahre hinlänglich bekannt geworden, und man wird sich nach dem Vorhergegangenen darüber um so weniger wundern, als eben notwendiger Weise auch in diesem Falle eine Sammlung des Giftes an jenen Stellen des lebhaften Appositionswachstums stattfinden muss.

Aber auch abgesehen von diesen intensiven Processen, welche zu einer weitgehenden Zerstörung des Knorpel- und Knochengewebes und zur vollständigen Ablösung der Chondroepiphysen von den Diaphysen führen können, vermisst man bei dem hereditär syphilitischen Foetus und Kindern der ersten Lebensjahre *niemals* die deutlichsten Zeichen einer entzündlichen Reizung, also die vermehrte Vascularisation und Blutüberfüllung der knochenbildenden Gewebe mit allen ihren Consequenzen, wie sie eben der Rachitis zukommen, und auch die augenfälligen Erscheinungen der Rachitis, die Auftreibung der Knochenenden, die Craniotabes, die charakteristische Veränderung der Schädelform, die Anomalien der Zahnbildung u. s. w. vermisst man nur sehr selten bei den hereditär Syphilisirten, ja man findet dieselben auch sehr häufig dann, wenn diese Kinder unter den günstigsten Verhältnissen gehalten werden. In diesen Fällen ist es also höchst wahrscheinlich das syphilitische Gift selber, welches den entzündlichen Process an den Knochenappositionsstellen hervorruft und unterhält.

Darin scheinen mir indessen einige Autoren der neueren Zeit zu weit gegangen zu sein, dass sie, wie Parrot in Frankreich und Lees und Barlow in England, der Syphilis die Hauptrolle bei der Entstehung der Craniotabes zuschreiben wollen. Nach meinen Beobachtungen ist die Craniotabes eines der häufigsten Symptome der allgemeinen Rachitis im ersten Lebensjahre. Ich habe aufs Geratewol die Notizen über die letzten 100 *Kinder unter einem Jahre*, welche in den letzten Wochen in mein Ambulatorium gebracht worden sind, in Hinsicht auf die Rachitis revidirt, und habe gefunden, dass von diesen 100 Kindern 84 zweifellos rachitisch waren, und nur 16 sich als ganz frei, oder noch nicht mit zweifellosen Erscheinungen der Rachitis behaftet erwiesen. Von diesen 84 Rachitischen waren 47 mit Craniotabes behaftet. Von diesen 100 Kindern waren aber nur 3 hereditär syphilitisch. Diese waren allerdings sämmtlich schon rachitisch und 2 zeigten die Erscheinungen der rachitischen Schädelerweichung. Alle übrigen 45 Kinder mit Craniotabes waren nicht hereditär syphilitisch.

Wenn ich also auch damit nicht übereinstimmen kann, dass der ererbten Syphilis ein numerisch so bedeutender Anteil an der Entstehung der Rachitis oder eines speciellen Symptomes derselben, nämlich der Craniotabes, zugestanden werden soll, so stimmt doch sowol meine Theorie der Rachitis, als auch die tatsächliche Beobachtung darin überein, *dass auch das syphilitische Gift im Stande ist, den rachitischen Process in den Knochen hervorzurufen und zu unterhalten.*

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## *Beiträge zur Pathologie der Rachitis.*

Dr. F. H. Rehn, Frankfort-on-the-Maine.

1.—Vergleichende Messungen des Schädelumfangs gesunder und rachitischer Kinder des ersten und der ersten Hälfte des zweiten Lebensjahrs ergaben *im Durchschnitt* grössere Werthe bei letzteren, besonders vom 8ten Lebensmonat ab.

2.—Die in diesem Lebensabschnitt noch vorhandene—durch pathologische Verhältnisse oft gesteigerte Dünnheit des Schädeldachs, lässt im gegebenen

Fall den Schluss auf ein grösseres Hirn-Volumen rachitischer Kinder zu. (Messungen aus späterer Zeit sind nicht maassgebend.)

3.—Der chronische Ventrikel-Hydrops, in seiner wesentlich extrauterinen Entwicklung, stellt eine sehr seltene Complication *angeborenen* Rachitis dar. Die Diverse ist prognostisch wichtig, in der der Vortragende einen hochgradigen Fall der Art nach nochmaliger Punction bei entsprechender diätetischer und medicamentischer Behandlung heilen sah.

4.—In einer Anzahl von Fällen hochgradiger Rachitis constatirte Vortragender eine geringe Vermehrung der weissen Blutkörperchen;—unabhängig von einer Milz-Vergrösserung, Vermehrung bis zum vierten Jahre der normalen Zahlung, (mit dem Malassiz-Hagen'schen Apparat, modificirt von Thoma-Zirpi).

5.—Den bei Rachitis vorkommenden Krampf-Formen ist, nach der Beobachtung des Vortragenden, auch der *Spasmus nutans* in seinen prognostisch, günstigen Formen—und zwar nicht immer mit dem Mittelglied erschwerter Dentition—zuzurechnen.

6.—Es kommen bei hochgradiger Rachitis, abgesehen von der Malacie der Schädelknochen, ausgeprägte Malacien der grossen Extremitäten-Knochen, speciell des Vorder-Arms und Vorderschenkel-Knochen vor, welche die Möglichkeit nahetragen, dass unter besonders ungünstigen Ernährungs-Bedingungen eine mehr oder weniger allgemeine Erweichung des Skeletts bei Rachitis eintreten können.

7.—In einem dieser Fälle wurde eine erhebliche Ausscheidung von Erdphosphaten—in überwiegender Menge phosphorsaurer Kalk—im Harn bei dreimaligen Proben nachgewiesen. Dieser Nachweis ist von Wichtigkeit, insofern er die Untersuchungs-Resultate neuerer Autoren (Neubauer, Seemann u. A.), welche keine Verwesung in Erdphosphate nachweisen konnten und die älterer Untersucher, welche erhebliche Mengen fanden (Marchand, Lehmann) zu ermitteln geeignet ist.

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## *Zur Pathologie der Rachitis.*

Dr. Adolf Baginsky, Berlin.

1.—Die klinische Erfahrung, dass Rachitis congenital vorkommt, dass die Mehrzahl mit congenitaler Syphilis geborener Kinder bei bester Pflege rachitisch wird, dass die Rachitis bei Kindern entsteht nach schweren fieberhaften Krankheiten, nach schweren chronischen Dyspepsien, bei schlechter Ernährung, mangelhafter Zufuhr frischer Luft, die weitere Erfahrung, dass Rachitis mit erheblichen Alterationen des Nervensystems verläuft (so Laryngismus Stridulus, Hydrocephalus, Hirnhypertrophie), alles dies schliesst schon klinisch die Möglichkeit aus, die Krankheit auf einen einfachen Defect der Kalksalze zu reduciren.

2.—Die Annahme, dass die Rachitis auf einem blossen Defect der Kalksalze beruhe, wird auch dadurch ausgeschlossen, dass das Verhältniss des Kalkgehalts des Knochens zu der organischen (fettfreien) Substanz desselben so erheblich verändert ist, cir. 160·100 gegenüber 563·100 in normalen Knochen, dass wenn man voraussetzt, man habe einem Kinde den Kalk ein ganzes Jahr hindurch gänzlich entzogen, dieses Missverhältniss nicht resultiren würde, sondern nach annähernder Berechnung auf 513·100 käme.

3.—In einem gewissen Gegensatze zu diesen klinischen Erfahrungen stehen die experimentellen Studien einiger Autoren (obenan Roloff, Berlin) welche den Nachweis geführt haben, dass man durch Entziehung des Kalks im Futter, Rachitis bei Thieren erzeugen könne.

(*a*) Reine Entziehung des Kalks im Futter erzeugt allerdings eine erhebliche Veränderung im Knochen.

(*b*) Dieselbe stellt sich makroskopisch und mikroskopisch dem Auge nahezu so dar, wie eine leichte rachitische Veränderung des Knochens.

(*c*) Der erzeugte Grad der Knochenveränderung ist aber, wie die anatomische Untersuchung, ganz besonders aber auch die chemische Analyse ergiebt, ein milder.

4.—Wenn man nun neben der Entziehung des Kalks, Milchsäure dem Futter zusetzt, so sind die erzeugten Veränderungen wesentlich beträchtlicher. Das ist beweisbar:

(*a*) Sowohl durch die makroskopische und mikroskopische Untersuchung des Knochens.

(*b*) Ganz besonders durch die chemische Untersuchung.

5.—Die beträchtliche Beeinflussung des Knochens durch die Milchsäure, neben der Kalkentziehung beweist, dass in der Kalkentziehung das Massgebende der Einwirkung nicht liegen könne.

6.—Vielmehr ist bei den Factoren, der Kalkentziehung und der Milchsäurefütterung, das Gemeinschaftliche, dass sie eine Alteration der Gesammternährung bewirken; das eine Mal geschieht dies durch Entziehung eines integrirenden Bestandtheils des Organismus, das andre Mal durch Einführung einer die Verdauung störende Substanz.

7.—Die Läsion der Knochen tritt nur deshalb in den Vordergrund, weil die Störung der Gesammternährung in eine Zeit fällt, wo das Knochenwachsthum der jungen Thiere besonders lebhaft ist.

8.—Man kann weiterhin den Nachweis suchen, dass neutrale Peptonlösungen Kalk zu lösen im Stande sind.

9.—Diese Eigenschaft der Peptone zeigt den Weg, wie bei Kindern, deren Blut durch fehlerhafte Ernährung alterirt ist, Kalksalze aus dem im Wachsthum und in der Ossification begriffenen Knochen gelöst und weggeführt werden.

10.—Die Ausscheidung der so weggeführten Kalksalze geschieht, wie man durch Untersuchung der Stuhlgänge rachitischer Kinder erweisen kann, durch den Darm.

11.—Demnach ist also die Rachitis eine Dyscrasie, hervorgegangen aus einer Alteration der Gesammternährung, welche wiederum durch die mannigfachsten, auf den jungen Organismus einwirkende Schädlichkeiten, bedingt wird.

12.—Damit stimmen die seit längst her gegenüber der Rachitis errungenen therapeutischen Erfolge durch solche Mittel, welche die gesammte Ernährung zu heben geeignet sind, insbesondre gute hygienische Verhältnisse, normale Nahrung, der Gebrauch von Bädern und tonisirenden Arzneien.

## DISCUSSION.

Dr. Jules Guérin, Paris: If the doctrine which purports to attribute rickets to congenital syphilis had not been put forward gravely by a man whose name and whose work lay claim to consideration, it would have been out of place to dwell on it at all. This doctrine is indeed not only an empty hypothesis, but manifestly false, and is in clear contradiction to all the facts of observation and experience. A few words will be enough to demonstrate this statement. As far as *observation* goes, the doctrine of syphilitic rickets is based alone on a material change, arbitrarily and quite exceptionally detached from the malady in its entirety, by the suppression of all the other symptoms and lesions which mark its course. Rickets, as we see it in its development under our obser-

vation, begins by a general disturbance of the economy—an alteration of the digestive functions. On this there follows a perversion of nutrition. The osseous system, to which, before my researches, the whole malady was exclusively referred, begins to undergo change only in a second stage of the disease. It suffers at first an arrest and then a perversion of its process of organisation. To keep to the most well-marked characteristics of this disturbance, I must call attention to the fact of the concentric layers of the long bones being the seat of a nearly colourless fluid effusion which separates them from one another and renders them isolated. This interlamellar effusion of liquid undergoes a progressive change of consistence and appearance. It becomes gelatiniform, and soon acquires the consistence of a reticulated tissue of a pink colour, to which I have given the name of "tissu spongoïde." At a more advanced period, if the malady tends to get well, this spongoid tissue becomes the seat of calcareous deposits, the islets and lamellæ of which can be seen by the naked eye. Finally, this newly formed bony tissue acquires all the consistence of normal bone. During this succession of changes of the newly formed tissue the old osseous tissue itself presents a series of modifications no less interesting and significant. Arrested at first in its development, it loses its consistence, its lamellæ waste and are partly absorbed. That which remains unites with the newly formed tissue and becomes inseparable from it. When the disease does not tend towards cure, all the old bony tissue disappears, and gives way to a mass of spongoid tissue, reddish in colour, areolar and elastic, which yields to the weakest muscular contractions, whence result the angular bendings and fractures of rickets. In a lesser degree of the disease, some of the lamellæ of the old bone still remain, but they undergo changes in form, owing to their defective resistance, the vertical action of the weight of the body, and the slow but repeated actions of the muscular contractions. In this way come about the more regular curvatures of the legs and thighs, the deformities of the pelvis and of the spinal column. What place and what meaning among this assemblage of facts, which are strictly successive and harmonious, and are united in one and the same morbid process, can be claimed by certain temporary changes of bone, accidental in character (the nature of which I refrain, indeed, from considering here), and which even the author of the theory I am combating recognises and allows as being often wanting? They can only be *epiphenomena*, the minute description of which testifies to the patience of the observer, but comment on which is only calculated to throw into confusion what is certainly and clearly demonstrated. Thus much for the testimony of observation. Now for that of direct experiment. Having been led by the observation of more than two thousand cases of rickets in children, who came before me at the Hôpital des Enfants, to ascertain that all of them, belonging to the working classes, had been prematurely fed on substances fit only for adults, either alone, or in combination with either a natural or artificial milk diet, I drew the conclusion that this malady might be the product of this premature alimentation. Since that time, at several intervals, I have submittted dogs of three or four weeks old to a course of premature feeding—bread, meat (and especially raw meat), and no milk. Not only have these animals exhibited in succession the phenomena of rickets as it develops in children—large belly, swelling of the epiphyses, curvature of the shafts of the bones, and difficulty in walking—but I have proved, by an interruption of the prescribed diet, a kind of suspension of the morbid process, and then the return and aggravation of the process when recourse was again had to the experimental regimen. Two of these dogs, killed at the most advanced period of the malady, showed all the alterations of the osseous tissue that are seen in rachitic children, and, in particular, the separation of the concentric layers of the long bones, and the production and deposit in their interstices of the characteristic spongoid

tissue. I have showed several specimens from these animals at the Academy of Medicine. I have had drawings made of them at the most advanced period of their deformity, and I have now in my collection, for all who wish to see them, two of the skeletons of these artificially rachitic dogs. To these unyielding facts, to those decisive experiments, M. Parrot has brought in opposition certain experiments which have not succeeded, and denials or erroneous interpretations. Among other things, our colleague says he has produced osteomalacia by premature feeding, but not the "spongoid" tissue. This result depends on the animals having been killed at too early a stage of the disease, when the bony tissue had not yet begun to be re-formed. As to osteomalacia, it is never found in dogs submitted to premature feeding any more than in children, for the good reason that osteomalacia, an essentially different disease from rickets, and belonging to a more advanced age, is quite apart from rickets in its causation. Finally, I have shown that in putting forward "spongoid" tissue as a specific mark of syphilitic rickets, M. Parrot has supported himself entirely on a merely transitory stage of the malady, on an isolated fact, throwing on one side all the other phases and phenomena which constitute rickets in its entirety.

Dr. Byers, Belfast: My object in taking part in the present discussion is to show how clinical experience gained at a provincial children's hospital points in reference to the *quæstio vexata* of the relationship of syphilis to rickets. At the out-patient department of the Hospital for Sick Children in Belfast, with which I am connected, cases of rickets are of frequent occurrence, and when I say cases of rickets, I must be understood to include in that category not merely those exaggerated forms in which almost every bone in the child's body is affected, and which more frequently come under the care of the surgeon than the physician, but also those milder varieties, too often overlooked, in which the morbid process is only just beginning, or has been arrested at an early stage of its development. On the other hand, cases of congenital syphilis are much less frequently met with. I have observed carefully a large number of these cases of rickets, keeping specially in view the question of syphilitic causation, and the result has been, that so far as these observations have gone, they are opposed to the view that syphilis *per se*, as has been insisted on very strongly by some, is the cause of rickets. The evidence afforded by these cases as opposed to the syphilitic causation of rickets is twofold:—(1.) In families in which there were rickety children I could get no evidence, in the great majority of cases, of the occurrence of syphilis in them, in other members of the same family, or in their parents. (2.) In the case of families in which some of the children presented unmistakable evidence of congenital syphilis, or in which I knew that the parents had been the subjects of this disease, I was unable to find in them, or in their brothers or sisters, those signs which we in England are accustomed to regard as diagnostic of rickets, with the exception of craniotabes, about which I shall have to say a few words. With reference to the occurrence of this symptom craniotabes, which some believe to be of such paramount importance as an early diagnostic sign of rickets, my observations would confirm the conclusions of Dr. Barlow and Dr. Lees, who have done such good work in elucidating the clinical value of this symptom—viz., that it is much more frequently present in the skulls of those who are the subjects of congenital syphilis than in the rickety. Indeed, I would be inclined to say—at least such is the result of my own observation—that this sign is by no means of such frequent occurrence in rickets as one would infer from a perusal of the current literature on the diseases of children. I have not as yet met with examples of children who at one time showed signs of hereditary syphilis afterwards becoming rickety; and even if cases of this kind did occur, they would

by no means prove that the syphilis was the sole cause of the rickets, unless we could exclude altogether every other existing cause, such as improper food, &c.; for I hold that it is just as illogical to argue that in such cases the syphilis and rickets stand in the relationship of cause and effect, as it would be to say that in the case of a child who at present is the subject of rickets, and who had previously suffered from measles, or any of the other exanthemata, that the measles was the sole cause of the rickets, unless in all such cases we were absolutely sure that there was no other element, such as unsuitable diet, bad hygienic condition, &c., which might have acted as a cause. I remember how Mr. Jonathan Hutchinson in his clinical lectures at the London Hospital used to impress on us that syphilis is the great simulator of other diseases, illustrating this specially by reference to skin diseases; and he has shown that in congenital syphilis a state of general periostitis is very common, in which the joint ends are swollen and tender and the child is in pain all over. Such a condition would be very liable to be mistaken for rickets, with which, however, it has no real connection. I am compelled, then, to agree with those who hold that syphilis is not the cause of rickets. My own observations lead me to conclude that errors in food, either in quantity or in quality, are the principal cause of rickets.

Professor STEPHENSON, Aberdeen: A question so important and general, cannot be decided by the changes in the structure of bone, nor yet on the general pathology alone. Physiological experiment is valuable, but not in itself sufficient. With these must be combined the clinical history. The researches must agree, not contradict each other. I have made the clinical history a subject of special study for twenty years in Edinburgh and in Aberdeen, where I have seen much of rickets. The result of my observations emphatically contradicts the statement of M. Parrot, that "hereditary syphilis is the constant cause of rickets." The clinical course of the two diseases, as a speaker has already observed, is wholly different, and in some respects opposite. M. Parrot has observed this, and tried to get out of the difficulty by saying, "The syphilis is spent. It no longer exists, but has substituted for itself a new affection. This is an incontestable example of transformation of disease." The pathological facts demonstrated by M. Parrot are extremely valuable, but his deductions therefrom are wholly erroneous. Syphilis and rickets are quite distinct conditions. They are like the red and yellow rays in a beam of light; they may combine to form an orange band, but to regard the one as caused by the other is as false as to say that the yellow light is transformed into the red. The key-note to the pathology is the recognition that rickets is dependent upon a diathesis, or, to avoid the differences of opinion attached to that term, I would say a *constitutional habit*. Bad hygienic conditions are not in themselves sufficient; they are common exciting causes of disease, in one child producing rickets, in a second tuberculosis, in a third scrofulosis. The determining element is the constitutional habit, but this opens up the general pathology, which is not before the meeting.

Dr. GIBERT, Havre: Although I hold but a modest position as a man of science, it is my duty to state the results which a long clinical experience has given me. I am a convert of M. Parrot's, but a convert in spite of myself; it by facts that I have been converted. There are two important points to touch upon. (1.) Can chronic enteritis produce the bony changes seen in rickets? I affirm that it cannot. Situated as I am at Havre, in a country where enteritis makes considerable ravages every year, I am able to say that bad or unsuitable feeding produces those disorders which have been described by Chossart, and

by M. Parrot in his excellent work on "Athrepsie." (2.) Rickets treated by antisyphilitic remedies gets well much sooner than by any other method of treatment. This assertion rests on a large number of cures. M. Guérin has made use of this argument in his appeal to the saying, "Naturam morborum ostendit curatio." In conclusion, M. Gibert asked that at the next Congress anatomical specimens should be shown, demonstrating that rickets is produced by bad feeding alone.

Professor RANKE, Munich: I would express the opinion that large centres like Paris, where syphilis frequently occurs, may not be suitable places for the study of the etiology of rickets. I have myself a large field of observation at Munich, 4000 to 5000 children a year, and I see amongst that number a great many cases of rickets, and only comparatively few cases, perhaps twenty a year, of hereditary syphilis,—the latter disease being rare among the Munich population. A great many cases of rickets are also continually brought to my clinique from the country, children of well-to-do peasant proprietors, amongst whom syphilis may be said to be scarcely known. I have, on this field of observation, come to the conclusion that there is simply etiologically *no* connection between syphilis and rickets. Of course, with this statement, I do not mean to deny that hereditary syphilis may not, in some rare cases, show a certain resemblance to rickets, as Dr. Wegner has shown some years ago, and as some of the specimens exhibited by Professor Parrot seem to point out. But the greater part of Professor Parrot's specimens appear to me to demonstrate merely a co-existence of syphilis and rickets in the same subject. This co-existence of the two diseases may be frequent enough in very large cities, where syphilis abounds, but it remains, nevertheless, a mere coincidence. In conclusion, I would say that M. Gibert's method of coming to a conclusion regarding the etiology of rickets by way of therapeutical observation seems to me not a scientific one. If M. Gibert has frequently seen a cure of rickets after the administration of a few spoonfuls of his syrup, I can only say that he has seen a good number of cases of rickets getting well without any medicinal treatment at all.

Dr. SANSOM, London: The result of my observation at the North-Eastern Hospital for Children is opposed to the idea that syphilis is a constant cause of rickets. It is quite true that in a certain proportion of cases these are associated, but it appears much more probable that the true relation is, that syphilis is a cause of a general dyscrasia upon which rickets is engrafted. I consider that rickets is the attendant of a dyscrasia which is dependent on a great variety of morbid causes. I have found a prime phenomenon of rickets to be pyrexia; subdue the pyrexia (as you can by cold ablution), and treat the cases hygienically and medically with judgment, and the rickets becomes cured; but antisyphilitic treatment is by no means a *sine quâ non*. Whilst differing in my conclusion, I have to express my cordial thanks to M. Parrot for his admirable researches on bone-disease in syphilis.

Dr. ROBERT LEE, London: From an analysis of about 10,000 (10,833) cases of diseases of children which have come under my care during the last three years, I find 364 of rickets and 60 of syphilis. If I added to these the cases during the previous six years, we should have a total of about 1000 of rickets and 180 of syphilis. In addition to this I have the histories of 20 cases of family syphilis; in these only two cases are recorded of rickets. From this experience I am of opinion that when syphilis does affect the bones, which it does but rarely, then it produces the condition described by M. Parrot. These are syphilitic and special to syphilis, but these are not what we usually term rickets;

so that it seems to me as true to say that syphilis never produces rickets as to say it alone produces rickets. If we take the analysis of M. Parrot's views, we should agree with him in the first six paragraphs. As we go on we find him introducing a theory, and attributing to osteomalacia those conditions which we universally call rickets. We therefore have a difference of meaning in our terms, and thus our discussion must be unsatisfactory. In regard to the twenty cases of family syphilis, let me observe that the difficulty which some meet with in obtaining histories of cases has really no excuse, and that from my experience there is no difficulty in obtaining perfect histories if we only take the trouble to do so.

Dr. EDDISON, Leeds: I am glad to express my sense of the value of the discussion that was begun by the paper of M. Parrot. I object strongly to what I must describe as a vicious argument based on therapeutic results, and would point out that it is possible that the action of mercury may be most useful in cases of true rickets, in which there is no specific taint whatever. In all large towns in England there are ample opportunities of watching the course of cases over long periods of time, cases which certainly are improved by improving the food and the hygienic condition of the patients. Moreover, these cases do not present the various other symptoms of hereditary syphilis.

Dr. NORMAN MOORE, London: Every one who begins the study of rickets soon comes, I suppose, to some one hypothesis with respect to its cause. I must confess that I did so, and that my first conclusion was that rickets had but one cause, and that that was improper feeding during the period of suckling. Subsequent observation convinced me that there are cases in which one must seek some other etiology, and no doubt cases of syphilis are amongst these. It has seemed to me that there is one point which has been overlooked—that of the geographical distribution of the disease. It occurs everywhere; but when I read with great respect and admiration the observations of M. Parrot, and found him describe as common what is certainly rare, at any rate, in some regions of observation in this country, it seemed to me that the most probable ground of the difference is the fact that this disease, occurring at a period when the individual is so subject to the habits of the population in which he lives, is one of the most likely to be affected by local conditions and conditions of race. With regard to the question of syphilis, I think too little exactness is often used in the determination of the existence of syphilis in any given case. Applying the most exact tests which I could frame to the numerous cases of rickets which I have observed, I must say that I have been able to regard syphilis as a cause of rickets in but a small proportion of cases.

Dr. GOODHART, London: Without detaining the members of the Section more than two minutes, I can, and I think ought to, add something to the discussion with reference to craniotabes. And as a preliminary let me say, that while thinking, as every one must do, most highly of M. Parrot's work and preparations, I, in common with most English observers, have been, and am still, unable to accept the doctrine of the unity of rickets and syphilis. And when my friends Drs. Barlow and Lees published their observations upon craniotabes, a disease heretofore regarded as rachitic, but by them considered to be syphilitic, I was quite opposed to the belief in its syphilitic origin. However, the accuracy of all their work made me go into the question of craniotabes again more minutely; and of the cases that I have seen since then, some ten or twelve, they have all, with I think one doubtful exception, presented more or less evidence of congenital syphilis. Thus, so far, I am

coming round to the opinion that craniotabes is an affection associated with congenital syphilis; at the same time, I am sure, if such a statement is worth anything, that rickets to its most pronounced degree is often unassociated with any evidence whatever of syphilis, and occurs in families—of which each of us could, I feel sure, give examples—where our intimate knowledge of all their surroundings enables us to say with much assurance, that they are free from any taint of such a disease, spent or otherwise.

Dr. A. JACOBI, New York: Craniotabes is by no means a symptom of syphilis; the immense majority of cases have nothing to do with the latter. Mercury is used freely in cases of dyspepsia, as an antifermentative, as an antiphlogistic, and its alleged efficacy in rachitis does not prove the syphilitic nature of the latter. The counting of blood-cells after the use of mercury in small doses given a long time appears to show that their number is increased. Syphilis may be *one* of the causes of rachitis, but there are many others. Still Professor Parrot asserts that syphilis is the only one. I might just as well say that mothers' milk is the only cause of rachitis, because some babies get rachitical when fed exclusively on caseinous and fat milk.

M. PARROT, Paris: The arguments which have been brought against my views rest chiefly on two points, etiology and treatment. With regard to etiology, M. Gibert has already given a good answer to objections. It is well known that there is a large number of children weakened by chronic maladies, enteritis, pulmonary tuberculosis, bronchitis, Pott's disease, &c., who have no sign of rickets, while there are rickety children who are both large, fat, and of healthy appearance. These causes of debility produce osteomalacia—*i.e.*, the disappearance of a certain quantity of lime salts, the enlargement of the Haversian canals, and the substitution of soft tissue for the hard material. But they do not ever produce rachitis—*i.e.*, a lesion essentially marked by the existence of spongoid tissue; for without this spongoid tissue there is no rachitis. It has been said that rickets has been produced in animals (especially in dogs) by M. Jules Guérin; and as they do not get syphilis, the conclusion has been drawn that rickets is not always produced by hereditary syphilis. But M. Tripier of Lyons, who has made numerous experiments on the production of rickets after Guérin's method, has been unable to produce it. I too have tried to produce rickets according to this method on two litters of puppies. I have produced a slight degree of softening of the bones, but never rickets. With regard to treatment, it has been said that if rickets arose from syphilis we ought to be able to cure it by antisyphilitic treatment. I, for my part, say that we *can* cure the osseous lesions of hereditary syphilis in their two first stages—those, namely, which precede the rachitic period. But the syphilis has spent itself in producing rachitis, and is no longer syphilis. It is a new disorder, and I consider that here we have an example of the transformation of disease. Have we not similar instances in dermatology? I give iodine in rachitis, but I give it as we do in many other morbid states where syphilis is not concerned. I may add, however, that I have often doubtlessly referred its efficacy to the source from whence the rachitis springs.

M. BOUCHUT, Paris: Syphilis, considered from the point of view of a specific poison, goes for absolutely nothing in the causation of rickets. It only contributes to such a state by the cachexia which it involves. In this light it takes its place with scrofula, anæmia, and all chronic maladies which arrest the regular progress of growth and which produce a malnutrition of the body generally and the bony system. Among these cachexias, chronic enteritis is the

true cause. Whatever it is due to, prolonged diarrhœa arrests development and produces all the changes seen in rickets. To such an extent is this true, that one may see a child of a year old, unaffected by syphilis and able to walk alone, suddenly fall ill, and be prostrated for five or six weeks; and when it is convalescent, it is found that it can no longer hold itself up when the endeavour is made to induce it to walk. The bones are soft and painful, and it perhaps will be three months before the child can walk again. We see rickets produced under these conditions, and under these conditions we can bring it into existence. Again, rickets can be produced at will in dogs. If half of a litter of puppies be shut up, and stuffed with food, causing diarrhœa, while the other half are given their freedom and suitable nourishment, the former become rickety, the latter undergo normal development. Where do we see here a place for the syphilitic element? Syphilis, then, as such, is no factor in the production of rickets, and I see nothing in such an affirmation but an hypothesis badly supported.

Dr. H. Rehn, Frankfort: I must maintain the non-identity of rickets and hereditary syphilis. It is most remarkable that a dispute should have taken place as to the existence of rickets, on the soil where the definition of the disease by Glisson originated. With regard to the most creditable researches of Parrot, I must refer to Wegner's investigations on hereditary syphilis in young children, published some years ago. In Parrot's description I miss the termination in necrotic caries and detachment of the epiphysis. According to Wegner, it is only in the first stage (*i.e.*, the stage of proliferation in the intermediary cartilage) that a similarity exists between the syphilitic and rickety processes at the epiphysis; later the two processes are widely separated. Clinically they differ, in my opinion, in the marked local signs of inflammation accompanied by fever which are confined to syphilis, in which disease also characteristic lesions of skin and mucous membrane are rarely absent. Moreover, the earlier period of life is not the proper time to decide this question. Those cases are especially confirmatory in which the well-known signs of rickets present themselves at the end of the first or second year, at the time of weaning, from sudden alteration in nutrition, improper food, or after a gastro-intestinal disorder following a grave acute disease. With regard to the accurate decision of these points, let me point out the favourable position of the family doctor. Professor Parrot's opinion regarding the cessation of syphilis at the moment it produces rickets, his supposition of a *transformation*, and his statement regarding the non-contagious nature of this rickets, is the making use of unintelligible and inadmissible notions. Professor Parrot's additional three propositions regarding the relation of osteomalacia to rickets are also highly confusing, since they place us on entirely new ground. I would ask whether, if (as Professor Parrot states) the causes which have been considered those of rickets can induce osteomalacia only, the conclusion does not logically follow that the usual definition of rickets resolves itself into syphilis and osteomalacia? With regard to the third proposition of Professor Parrot, "Osteomalacia may be produced artificially, rickets never," I must refer to the recent experiments of Dr. E. Voit of Munich, with microscopical examinations by Professor Buhl.* My views lead me to the conclusion that rickets and syphilis are processes of slight similarity only with regard to their localisation in the skeleton, but in their etiological, clinical, and therapeutical aspects differing totally.

Dr. West, the President: You will, I trust, not think that I arrogate a right to myself in consequence of the honour you have done me in appointing me

* *Zeitschrift für Biologie*, 1880.

your President, if I ask permission not so much to give a *résumé* of what has been said by previous speakers, as to express my own opinion with reference to some of the questions which have come before us, and which have been discussed with so much ability and in a tone which eminently becomes a scientific congress. It has struck me that some light might be thrown on the question of the relation of syphilis to rickets, and something might be gained, by an appeal to history; for if syphilis were its invariable cause, rickets ought not to have been known before the siege of Naples. Now, although we owe the first formal description of the disease to our countryman Glisson, I think one finds traces of a disease affecting the young and producing deformities like those of rickets long anterior to that memorable event. Another point which seems to me worth notice is the important one raised by Dr. Norman Moore, and concerning which I had hoped he would have given us further information—namely, *the influence of climate and geographical distribution*. My impression is that rickets in Paris presents a less severe character in general than that which it commonly presents in London. A Brazilian physician whom I met in Paris told me that while syphilis is extremely common in the Brazils, rickets is an unknown disease; and it would throw much light on the question if it were ascertained that in the islands of the Pacific, the inhabitants of which are known to have been decimated by syphilis, rickets was rare or unknown. With reference to the indications of past syphilis on which M. Parrot relies, some of them, as the condition of the tongue, which he pointed out to me as syphilitic, did not to me appear conclusive, as I think I have seen this in isolated members of a family which was otherwise healthy, in connection with symptoms of indigestion, and wholly independent of any symptom of syphilis past or present. With reference to the one special condition, craniotabes, I do feel a certain hesitation as to its possible connection with bygone syphilis, but my feeling is one of hesitation and nothing more. As to the general manifestations of rickets, they do not in general show themselves with distinctness before the second year, often even later, and in the great majority of instances are ushered in by a general febrile disturbance, and by other symptoms indicative not of a special affection of the osseous system alone, but of a general constitutional disease. Moreover, there is one peculiarity already referred to by M. Guérin which is characteristic of all the deformities produced by rickets—namely, that they are attended by an arrest of development. This, however, is not the case with children who may yet be most markedly syphilitic, and who have grown up to the age of six or seven years, or older, with evident tertiary syphilitic symptoms untreated, or certainly uncured. The arrest of development, too, does not affect the whole skeleton alike; the vertebral column, though twisted and deformed, is not shorter than natural were it but stretched out, while the pelvis is dwarfed, and the long bones present a length far below what is natural. Besides, the possibility of producing rickets in animals by feeding them on unsuitable and consequently unnutritious food, and their recovery when properly dieted, points in the same direction; and the experiments of M. Guérin and M. Wegner, and the later ones of Rollo of Berlin and of Voit of Munich, all lead to the same conclusion. For all these reasons, I can come to no other conclusion with reference to the opinions of our distinguished colleague M. Parrot than that, in the words of the Scotch verdict, they are *not proven*. Once more I apologise for having spoken; but I thought that having grown grey in the study of children's diseases, you would expect me to have an opinion on this important question, and to express it.

Dr. Rehn, Frankfort-on-the-Maine: Demonstrated the skeleton of a child of probably 1½ or 2 years of age, the only hitherto known case of osteomalacia in

childhood. Microscopically the absence of the common rickety changes at the epiphysial line is striking. Professor von Recklinghausen of Strassburgh made the microscopical examination, and considered it to be real osteomalacia. The skeleton was demonstrated for the first time at the Congress in the Section of Diseases of Children, and the case is described and discussed by Dr. Rehn in the chapter "Rachitis" of Professor Gerhardt's "Encyclopædia of Children's Diseases."

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## *Conditions Governing the Occurrence of Paralysis and Albuminuria in Diphtheria.*

Dr. West, the President, opened the subject by suggesting the following questions:—What governs the *albuminuria?* Does it bear any invariable proportion to the severity of a case? It always exists to a high degree in malignant fever, and appears slightly in all where the difficulty of respiration becomes extreme; but otherwise—What rule is there about it? Does it appear in some epidemics more than in others? Does it, and if so, in what circumstances, lay the foundation of abiding albuminuria, as that of scarlatina does? Is there a connection between it and subsequent occurrence or degree of paralysis?

*As to paralysis*, my own impression is that it is more frequent in France. It differs decidedly in different epidemics. It occurs sometimes with formidable risks in early stages of not apparently very severe diphtheria. When it comes on as sequela, it usually, but not invariably, begins at and extends from the seat of original deposit. But why does it become generalised? Does it leave post-mortem appearances in the nervous system? Why does it come on at a longish interval of time? What is its pathology?

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## *Paralysis and Albuminuria in Diphtheria.*

Dr. A. Jacobi, New York.

### I.—*Paralysis.*

Local paralysis of the soft palate, and sometimes the muscles of deglutition and epiglottis, often attends the local deposit on these parts, with œdematous swelling of the same.

The diphtheritic paralysis, properly so called, is an affection of apparent convalescence. The majority of cases occur after mild attacks, sometimes after those with but little fever and a slow general course. It does not usually occur in those complicated with albuminuria and nephritis. There is no symptom indicating its occurrence in the future.

### II.—*Albuminuria.*

Albuminuria is often found in diphtheria, but is mostly no grave symptom. It may be the result of high temperature or of suffocation, as in other diseases. It often appears in the first few days of the disease, not always with high fever, and appears to be the result of rapid elimination of the poison. Acute diffuse nephritis makes its appearance at an earlier period than it does in scarlatina.

## *Albuminuria in Diphtheria.*

Dr. JOHN ABERCROMBIE, London.

I have recently had under observation ninety-one cases of diphtheria, in all of which the urine was examined.

Albuminuria was found in twenty-four. Of the ten patients in whom the urine became albuminous after they came under observation: in one, albumen appeared within twenty-four hours of the onset; in three, not until the tenth day of disease; in the others, it appeared between the fifth and eighth days. Judging from the cases where albuminuria was present when the patient first came under observation, it is evident that it is usually developed early in the disease, and its appearance so late as the tenth day must be looked upon as rather exceptional. Albuminuria does not, as a rule, last for long; in one case it only lasted two days, and in no case more than a fortnight. In no case was any anasarca observed, nor was the urine ever smoky, though a few blood corpuscles with epithelial and blood casts could be seen under the microscope.

I have never seen any symptoms of uræmia. Examination of the kidneys after death revealed the ordinary changes of acute parenchymatous nephritis. In one case that I examined with my friend Dr. Samuel West, we found the capillary vessels in the cortex crowded with minute shining bodies, which we take to be micrococci. I may add that the autopsy in this case was made seven hours after death, and that the kidney was only examined after hardening in chromic acid and spirit. It was the most malignant case I have ever seen. Of the twenty-four patients in whom albuminuria was discovered, no less than fourteen died, whilst of the sixty-seven in whom there was no albuminuria eight died; so that it is evident that the danger to life is much greater when there is albuminuria than when there is not, but it is also evident that freedom from albuminuria is no guarantee that the patient will recover.

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## *On Diphtheritic Paralysis in Children.*

Dr. JOHN ABERCROMBIE, London.

The unusual opportunities for studying diphtheritic paralysis that I have had at the Hospital for Sick Children, Great Ormond Street, will, I hope, justify me in bringing the results of my experience before the members of this Section.

I have to express my thanks to the members of the medical staff of the Hospital for their kind permission to make use of the cases.

The total number of cases included in this paper is eighteen, of which number nine proved fatal. Nine patients were boys. Half of them were between the ages of two and four years; with the exception of one boy who was nine years and three months old, the others were under seven years of age.

Only one patient was under observation during the diphtheria; it is necessary, therefore, to analyse the histories of the other patients, as they all came under observation after the development of the paralytic symptoms. Four patients had been treated at home by a medical man for diphtheria. In two cases the father had been under treatment at home for diphtheria. In four cases there was a history of sore throat, nasal discharge, and lumps in the neck. In three cases a history of sore throat, lumps in the neck, and fever with insanitary

conditions in house or neighbourhood. As regards the remaining four patients, in two there was a history of sore throat and swelling in the neck, in one of languor and sore throat, and in one of lumps in neck only. Most of the patients had not had any treatment for the primary illness, and several of them had not even kept their beds.

The *onset* of the disease is always gradual and insidious, and it is difficult, therefore, to fix with any degree of accuracy the earliest date at which the paralytic symptoms were noted. Excluding cases where thickness of speech was said to have persisted from the commencement of illness, I find that in two cases the paralytic phenomena appeared in two weeks from the onset of illness, and that in four cases they were not observed until five weeks from the onset; the other cases occurred between these limits.

In fourteen of the cases the *earliest symptom* was either nasal voice, return of fluids through the nose, or some difficulty in swallowing; in the remaining four, weakness in the legs was the first thing noticed.

In the majority of cases, the order of symptoms is much as follows:—Thick and nasal speech, cough when drinking, return of fluids through nose, weakness in legs, back, and arms. Occasionally, however, this order is almost exactly reversed. Thus in one of the very few cases in which I had the opportunity of watching the symptoms develop from the commencement, there was first weakness in the legs, then weakness of the arms, cough, alteration of voice, impairment of vision, difficulty in drinking, and finally inability to sit up or hold head up.

The only constant symptom was paralysis of the soft palate, which was present in all the cases. In twelve the soft palate was ascertained to be anæsthetic; in some of the other cases the point could not be determined.

As regards the relative frequency of other symptoms:—

*Irregularity of the heart's action* was noted in six patients. In one it was so marked that the case was at first thought to be one of organic heart disease. It is an early symptom, and often subsides after the patient has been kept in bed for a few days.

*Albuminuria* was found in four patients, one of whom recovered. In several of the fatal cases the urine could not be obtained; its absence was noted in seven cases, two of which proved fatal. It is not very easy to see why it should occur in this disease; possibly the patients in whom it was noted had already had nephritis with their diphtheria, and it may be due to a relapse of this condition, or the nephritis may never have cleared up. In one case hyaline granular and epithelial casts were found microscopically, but I regret that I have omitted to examine under the microscope the kidneys from any of the fatal cases. It must always be a symptom of grave import.

Incontinence of urine or fæces, as a rule, only occurs in the last stages of the disease.

The *pupils* are always *dilated and sluggish*; in two cases there was marked inequality. I have never been able to recognise any changes in the fundus oculi with the ophthalmoscope.

*Convergent strabismus* was present in seven cases; no other form of squint was seen. It has been pointed out to me that partial paralysis of accommodation in a hypermetropic person would produce this squint, and in one patient with squint it was ascertained that he was hypermetropic. It is not very easy in children to determine whether there is paralysis of accommodation or not, but in five patients this symptom was ascertained to be present, and three of these also had convergent strabismus.

*Facial paralysis* was noted in three cases; it was always slight and transitory.

*Paralysis of the muscles of the chest-wall* was present in ten cases; in one of the patients, who had had for some days paralysis of his intercostals, the diaphragm was paralysed for twenty-four hours, the patient breathing by the aid of his neck muscles alone. This patient was the only one of the ten who survived, and in my experience this symptom is the most formidable one that can arise.

The *knee phenomenon*, or patellar tendon reflex, as it has been called in this country, was found to be absent in nine cases; it was not sought for in the others. It is often, however, so difficult to obtain this knee jerk in healthy children, that we must be careful not to set too high a value on its absence.

The *cutaneous reflexes* were noticed to be natural in three cases—slight in one, absent in two.

*Cutaneous sensibility* was but little if at all affected in any of the cases.

The response of the leg muscles to *faradism* was found to be good in four cases; in one case where the constant current was employed the excitability was diminished.

*Duration of fatal cases.*—Two patients died about the ninth day after the onset of the paralytic symptoms; one between four and five weeks from their first appearance; in the other cases death occurred between these limits. In all the cases death resulted from pulmonary catarrh, in seven cases supervening upon paralysis of the diaphragm, in the remaining two upon paralysis of the intercostal muscles.

*Duration of the non-fatal cases.*—In the majority of cases the symptoms lasted from five to six weeks; in one patient they had disappeared at the end of three weeks, in another they persisted for fifteen weeks; this was by far the most severe of the cases that recovered.

As regards *post-mortem appearances*, in all the cases there was marked pulmonary congestion, and in two the heart was empty. There were no lesions of the nervous centres discoverable with the naked eye. In seven cases I have examined the medulla oblongata and spinal cord methodically after hardening in Müller's fluid; the sections were stained with hæmatoxylin.

The only pathological changes I have been able to detect are in the grey matter of the anterior cornua, and they consist in a swollen condition of the large motor cells. The margins of these are very ill defined, and the processes have in most instances entirely disappeared. The contents of the cell have a granular aspect and the nuclei have disappeared, or, where still visible, are highly granular. These changes only occur in very limited areas, and I have not found them constantly in any one region of the cord, but more commonly in the upper and middle dorsal regions than elsewhere. Where one cell of a group is affected, all the cells of that group show some change. In some places the cells appeared shrunken rather than swollen. Perhaps this is a later stage of the same process. In some specimens the neuroglia corpuscles were perhaps unduly numerous, and the central canal was sometimes found blocked with a homogeneous substance; but I am not prepared to lay any stress upon these points. No lesions of the white matter of the cord were recognised. The blood-vessels were in most cases filled with blood, but no extravasations were seen. The examination of the medulla oblongata yielded negative results with one single exception, where some of the outermost cells of the vagus nucleus were found to be rounded and more or less completely deprived of their processes.

Of the other two fatal cases, I examined the cord, in conjunction with Dr. Cheadle, in one; but our examination was not so exhaustive as it might have been, and we did not find any morbid condition. The other patient died during my absence from town, and I had no opportunity of examining the nerve centres microscopically. I have not examined the nerve roots or muscles in any of the cases.

The occurrence of hemiplegia in connection with diphtheria is so rare that I would briefly mention the following case :—A delicate boy, seven years old, came under treatment on the third day of a severe attack of diphtheria, the urine being albuminous when he was first seen. At the end of a fortnight all membrane had disappeared and the urine was free from albumen. On the sixteenth day he was convulsed, after which he complained of right frontal headache, and there was left facial paralysis. On the following morning he was again convulsed, after which there was complete left hemiplegia. Ultimately naso-pharyngeal paralysis appeared, and he died from paralysis of his intercostal muscles on the twenty-eighth day from commencement of illness. The right middle cerebral artery was found occluded at its bifurcation by a firm, pale, adherent thrombus.

I would briefly call attention to an account of an examination of three fatal cases by M. Déjérine in which he describes changes similar to those which I have found, but more advanced. He has further investigated the state of the anterior and posterior roots, and found the latter invariably healthy, from which he infers that there cannot be an ascending neuritis in this disease; but I am not aware of any facts which tend to prove that inflammation cannot travel up a motor nerve. A much stronger (indeed in my opinion conclusive) argument against the theory of ascending neuritis is to be found in the cases (which are not so very uncommon), where the paralysis does not commence in the palate, but in some remote part, such as the legs, for instance.

It has been said that paralysis may follow a simple angina, but when we recollect how common it is to find well-marked diphtheria in one child of a family, while its brothers or sisters have only a slight tonsillitis evidently due to the same poisoning, I think it is clear that in many cases an apparently simple angina may in reality be due to diphtheria. Every one will admit that a child may have scarlatina without the rash being noticed. Why may not a child then have diphtheria without the throat condition attracting notice?

As regards prognosis, my experience leads me to look upon this affection as one of the most formidable ones a child can have. As to whether paralysis is a common sequel of diphtheria, I do not find myself in a position to give a positive opinion. From the extreme rarity of its occurrence in children who come under observation for diphtheria, one would think not. But if it is so very rare, what an extraordinary number of cases must go unrecognised! I think the probable explanation must be that the good feeding and care taken of the children in hospital prevents it. Of course one must bear in mind that about 28 per cent. of the children die from their diphtheria before sufficient time has elapsed for the paralytic symptoms to appear.

With regard to treatment I have but little to say. Belladonna in one case, given in large and oft-repeated doses (half a drachm of the tincture every two hours), certainly seemed to act beneficially. At any rate, the child recovered after having had paralysis both of diaphragm and intercostal muscles. But in other cases it has failed, nor has the hypodermic injection of atropine proved of much use. Strychnia, iron, iodide of potassium, and arsenic have all been tried, but I doubt if they exercise much influence. The patients should be kept in bed, and especially protected from draughts; they should be given a liberal diet and stimulants.

I would submit that the foregoing observations justify me in drawing the following conclusions :—

1.—That diphtheria leaves behind it a special tendency to subacute myelitis, which has certain definite clinical characters, and that this tendency is at least as great after a mild attack as after a severe one.

2.—That the prognosis should be guarded.

3.—That the same precautions should be exercised in the after-treatment of diphtheria, with a view to prevent paralysis; as are observed to prevent the occurrence of nephritis after scarlatina.

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## *On Paralysis after Diphtheria.*

Dr. William Squire, London.

Diphtheria is not the only acute specific or febrile disease followed by paralysis of the kind so well known as its sequela; but it offers the type of this kind of paralysis, and that with such frequency as to make us look for it, in some degree, as an almost constant consequence of diphtheria. We find it also as a rare exception after rheumatic and enteric fever, erysipelas, uncomplicated scarlet fever, quinsy, and croup.

With all these diseases diphtheria has this much in common: sudden onset of high temperature, the increased waste of general fever, tissue change at the seat of local mischief, high vascular tension, and renal congestion, often with albuminuria, and always with impaired elimination.

To the general considerations I have to offer, time has added great results from the advance of nerve pathology, and so brought into clearness much that was most obscure. Accumulated clinical observations have also largely co-operated towards a fuller understanding of diphtheritic paralysis.

We shall best begin by taking some of the simpler instances of disease followed by paralysis. After deep-seated inflammation of any part, an alteration of sensation and motion will persist long after apparent recovery; nor is this simply the physical result of some exudation still left; altered sensation in one finger has persisted so long after erysipelas of the hand and arm, as to make a limited injury to nerve or nerve-trunk almost certain.

In acute rheumatism with any cardiac inflammation, remarkable slowness of pulse is very likely to follow, with ready after disturbance. The ends of the vagus at the seat of inflammation first suffer, and then the ganglionic innervation. After or during inflamed throat from any cause, there is dysphagia from effusion in the muscular structure, from imperfect muscular consensus, and fluids will return by the nares; the sensibility of the parts invaded by exudation is lessened, and the proper reflex is not readily excited. Sometimes, as in the case of erysipelas given, the nerve filament of the part is injured; and, in diphtheria at least, nutrition changes may reach the nerve origin and extend there, causing further mischief in the centres themselves. Diphtheritic paralysis may from its very commencement depend upon centric changes, presenting so far an analogy with infantile or essential paralysis, but in diphtheria the symptoms do not come on early, and will completely disappear. The centric disturbance in diphtheria is more often functional from temporarily impaired nutrition than from active irremediable organic degeneration, and is attended by loss of reflex and not by exaggeration of it, or by rigidity. An exact counterpart of this form of paralysis, as seen in diphtheria, has lately occurred to me in a young man twenty-five years of age—after rheumatic fever complicated by albuminuria. The part played in this case by renal congestion and consequently diminished elimination in delaying recovery and determining the paralytic symptoms is incontestable. Three weeks after the attack some numbness of fingers and then of the feet occurred; this quickly passed off, and then loss of power in varying muscles was noticed; the hands and abdominal muscles soon recovered, but the legs were altogether powerless; patella-reflex was lost; the extensors of

legs and feet, though acting tardily to the interrupted current, remained almost useless for three months. As in most such cases of diphtheritic paralysis, recovery was complete. Paraplegia after or during enteric fever beginning with more acute spinal symptoms, has disappeared less completely in longer time, and not without recurrence of some symptoms in later years. In rheumatic fever this more acute paraplegia of spinal origin is shown to be not very rare by a proneness to sacral bed sores in certain cases, with increased pain on turning the patient in bed, and a persistence of fever.

There are three ways in which these paralyses occur :—

1.—By the general effect of waste matters in the blood, where fever has been suddenly high, and the means of elimination insufficient.

2.—By the effect of local inflammation on the innervation of the part, with or without changes in the nutrition of the nerve-tissues.

3.—By nutrition changes in the nerve-centres as a consequence of altered blood supply during or after the illness. This may be one of the consequences of the first state, but it may also result from embolism, or from a local change in the blood-vessels, or from changes in the corpuscular elements in the blood, and not from any poison in the blood. Some of these changes may be attributable to special matters in the blood, as in the case of embolism from detached fibrin, but this generally leads to hemiplegia, and though frequent after rheumatic fever and not unknown in scarlet fever, is rare in diphtheria, and is not here under consideration. Where a special poison is imagined, the facts are less certain. It is the question of special poison I wish here to speak of. It is not the special poison of syphilis that paralyses; any irritant, or the accidental bleeding of a vessel, may determine the proliferation of cells in a syphilitic subject that will encroach upon the nerve structure and destroy it, if not removable by treatment. Why imagine paralysis the result of a special ferment in rheumatism, or of a special poison in diphtheria, when in both cases the waste products of those fevers remaining in the blood or tissues suffice to set up the changes on which those paralyses depend? No one imagines a special poison for infantile paralysis. There is an observation of Dr. Buzzard of excess of uric acid accompanying this kind of seizure, of some value as an illustration here.

The mere relaxation of small vessels during fever with excited heart leads to their dilatation; afterwards, a slower current may cause congestion, and either favour effusion, or prevent the removal of waste products which remain in the tissue to excite irritation; or, the blood itself may contain such waste, and so exclude, contract, or dilate the arterioles. This view harmonises well with what is seen in diphtheric paralysis; but there is another possibility to be contrasted with this, and also with the special poison theory, and that is the influence of a special germ or organism directly concerned in producing this as well as other phenomena of the disease. Given the dilated glomeruli in the febrile stages of diphtheria and scarlet fever, albuminuria with intertubular congestion and clouded epithelium with desquamation might be expected without the intervention of special microzymes to permeate the tissues and cells, block the vessels, and disintegrate the structure; though such particles are seen in the epithelium cell or in the nerve fibril, they may mark the degeneration without being the cause of it. To those who look for either germs or a special poison in all the manifestations of diphtheria, certain paralyses are as pathognomonic of the disease as fibrinous exudation; on the other hand, paralysis *per se* is no more pathognomonic of diphtheria than fibrinous exudation is. Diphtheritic paralysis is of two kinds: the one local, appearing early, directly produced, and of a gravity *proportioned* to the intensity of the local or general disease present; the other general, or secondary, coming on later, not before the second or third week, and *not in proportion* to the severity either

of the local lesion or of the general disease. Sometimes, as in the paralysis of the ciliary muscles, the loss of power of accommodation, and consequent impairment of sight, this accident, like the desquamation of scarlet fever, is the only sign by which we know that a patient has had diphtheria. This second form is much the same whatever the original seat of mischief.

The leading characteristics of post-diptheritic paralysis are: first, the passing and shifting character of these paralyses; they are generally functional, and of good prognosis, pointing to a temporary disturbance of nutrition, such as a passing congestion of nerve centres, or a limited vasomotor disturbance. Second, the time of their occurrence; this is at the end of two or three weeks, or even as late as six weeks, when the balance of healthy function throughout the body is being restored. It is to be noted that when this period is retarded, and the patient very weak, these functional paralyses are more likely to be prolonged, and may be associated with impairment of nerve structure, when co-ordination, or patella-reflex has been long in abeyance, and the reaction of certain muscles to faradisation almost lost. In many cases of diphtheria, where paralysis has come on late in the course of the disease, albuminuria has existed to a greater or less extent at some part of the illness; this may either persist at the time of the paralysis, or have already disappeared. Diphtheria may also be followed by paralysis when no albuminuria has existed at any time; some of these cases have occurred early, associated with a certain intensity of the local process. Terminal filaments of a nerve may be destroyed by ulceration, or their structure and that of a mixed nerve fibre invaded by the degenerative changes set up by inflammation; but short of this, the function of the nerve may be in abeyance as a consequence of over-stimulation, and here also the nerve centre is concerned. Hence the frequency with which mere return of fluids by the nose is followed by altered voice and impaired deglutition rather than by the more serious affection of the vagus, and loss of power in the muscles of the neck, or in those of respiration. In the earlier paralyses mixed nerve-trunks are implicated, and not the nerve centre to the same degree, for there are alterations of sensation, and the muscles readily respond to the stimulus of the interrupted current; in the severer cases this response is weakened; the centre involved is in the medulla or near the root of the phrenic; death may occur before any marked degeneration of the grey matter is established. Actual disintegration of both grey and white nerve structure and of the nerve axis has been shown by Sir W. Gull, Professor Charcot, and more recently and completely by M. Déjérine. Whether in these cases the degeneration has been propagated upwards by the destroyed nerve, or the nerve has suffered from injury at its ganglionic centre, is not so clear to me; probably both conditions occur, the latter most frequently; the former being more likely in the early incidence of paralysis after severe local lesion. There is a form of early paralysis, with dangers of its own, not connected with direct organic nerve lesion. Signs of cardiac paralysis, as early as the first week, arise from degeneration and infiltration of tissue, implicating the ends of nerves and not their centres. During paralysis of the respiratory muscles congestion of lung may set in, and not clear off when movement of the chest is restored. The ganglionic centres, either of the heart or the great sympathetic, may be affected; the former is shown by fatal cardiac asthenia apart from thrombosis, the latter by a case of Dr. Heyl (*American Journal of Medical Science*, April, 1880), of unilateral exophthalmos and chemosis.

There are thus clinically three great classes of post-diphtheritic paralyses:—

1.—Those occurring soon after the appearance of the local manifestation of the disease, even when there has been no serious local lesion, and with or without albuminuria.

2.—Those occurring somewhat later with grave local and general symptoms, likely to be associated with degeneration or disintegration of nervous structure.

3.—Those occurring after the second or third week of the disease, often as late as the sixth week, where there has mostly been albuminuria in the course of the disease, but not always a severe, and sometimes even a trivial initial lesion. These cases are mostly readily restored by the use of tonics, as iron and especially strychnine, and by the use of the continuous galvanic current; if not so aided, nerve degeneration may result, generally a descending paralysis with wasting of muscles. Cases of this kind form the bulk of those to which our attention is called after the first dangers of the disease are over.

These three clinical and pathological classes, if not exactly thus counterparts of each other, show a parallelism sufficiently close to be reasonable in theory and useful in practice. Diphtheritic paralysis does not always depend upon one and the same cause.

## DISCUSSION.

Dr. G. B. Barron, Southport: My experience is that diphtheria is not so frequent in England as it used to be, and that there is now no place in this country where it may be considered to be an epidemic disease. Many cases are called diphtheria which are not true diphtheria at all, with small patches of leathery ulcer, and even with slight albuminuria, but with no subsequent paralysis. My opinion is decidedly opposed to the prevalent idea that croup and diphtheria are identical. No facts that I know of convince me of this, and the result of a prolonged observation is negative. I have not seen a case of true diphtheria for fifteen years, but many that partook of some features of it. It is also much less frequent in the vicinity of Manchester than formerly, nor are such cases when seen of so malignant a character. As regards treatment, my experience leads me to rely upon great precautions against movement, properly administered nourishment, cleanliness of the fauces by washing with a weak solution of nitrate of silver, but not the application of the solid caustic, internally iron and chlorate of potash, strychnine in the later stages. The treatment has been of a very varied kind, some men using one thing, some another. Indeed the treatment of this disease is the odium of medicine, and wants some more defined principles. Tracheotomy is rarely necessary. I saw a case bearing upon this question, where a cast of the whole length of the trachea with a short cast of the upper part of the bronchi in a perfectly cylindrical form was coughed up, in a woman forty years of age. It was well marked with the ring indentations of the trachea; microscopical examination failed to show ciliated epithelium. The case was of course fatal. It was a case of acute fibrinous tracheo-bronchitis.

Dr. C. A. Leale, New York: I wish that I could agree with the last speaker in regard to the diminution of diphtheria in New York City, for nearly every month in the year I see cases of the disease, and occasionally the entire number of children in a family are lost on account of its intense malignancy. I was recently called in consultation by Dr. Fruitnight where the last of three children was in the comatose condition of the intensely poisonous stage of the disease, and where, from the bad hygienic surroundings, recovery was almost impossible. In regard to the occurrence of albuminuria, I have found it present in a very large proportion of the worst cases; the albumen being often 50 per cent. Of these cases, some may expect to recover. In regard to casts, the degenerations of the kidneys may be watched by their increase, number, and character. Some authorities state that fatal results follow if a great abundance of casts are present. In regard to the occurrence of paralysis, I have seen it commence in the vocal chords most frequently. I have observed a number of cases of paralysis

following diphtheria, most of which have recovered. In regard to treatment, I have found strychnine and iron most useful, combined with other tonics to improve the general condition. I have observed large accumulations of serum in the different serous cavities in the following order of frequency—peritoneum, pleura, pericardium, and arachnoid. I have seen instances of each recover.

Dr. A. JACOBI, New York: In regard to Dr. Barron's remarks, and the frequency of fibrinous tracheo-bronchitis: The literature is small, the first large monograph on the subject being by Professor Biermer of Breslau. In the spring of 1879 I have seen three cases in children, probably half-a-dozen altogether. About as many more I have seen in the post-mortem examinations or in fresh specimens. In regard to diphtheritic paralysis: The main diagnostic symptom, though the soft palate or the muscles of accommodation commence to be affected first, is the intact condition of the sphincters, and the irregularity in the order in which the several localities are taken. If there were nine fatal cases in Dr. Abercrombie's eighteen, it is probably because his cases were carried to the hospital when in very bad condition. I have seen a few cases of paralysis of the respiratory muscles, and all of them died, I believe, when the affection was fully established. The average cases of diphtheritic paralysis are mild, will get well in from five to ten weeks or more, and require tonic stimulant and nervine treatment,—the faradaic and galvanic currents, iron, and strychnia (either internally or *subcutaneously*). The latter is contra-indicated, it appears, in the usual forms of acute or subacute myelitis, but it is *certainly* the very best nervine in diphtheritic paralysis. The case of hemiplegia will, I think, not be claimed as diphtheritic by Dr. Abercrombie. There were two attacks of convulsions, which are the frequent cause of partial or complete paralysis, or death.

Dr. S. SPRATLY, Birkenhead: said that out of an experience of between 120 and 130 cases of diphtheria, and thirteen deaths, he had only seen two cases of paralysis, one severe and one mild.

Dr. C. WEST, President: There is no doubt of the contagious nature of diphtheria, and the possibility of infection direct from one person to another; also that this stands in some connection with damp, locality, and season of the year. The essential nature of the contagion is unknown, but while everything turns our attention in the direction of Pasteur's discoveries, it must be remembered that the presence of bacterian or of any other microscopic growth in the membrane, does not prove that to be the cause. A question to be considered also is, how far does the fact that one attack does not preserve from another tell against the bacterian theory? In the case of charbon, in which the contagium is of a bacterian nature, inoculation preserves the subject from an attack.

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## *Observations on the Spread of Diphtheria.*

Dr. HUBERT AIRY, London.

§ 1.—The remarks which, with your permission, I venture to offer upon the circumstances under which, in the rural parts of England and Wales, diphtheria is usually found to take an epidemic character, are directed more especially to the question whether or not diphtheria is spread by the agency of the wind; and from the facts adduced, an attempt will be made to form some tentative inferences as to the nature of the diphtheritic contagium.

The subject is one for which I must claim no small degree of international importance, for nation has given to nation this fell disease, and nations have been made fellow-mourners by the ravages it has caused. Let it be an object of international zeal and rivalry to track the paths and arrest the progress of this common enemy of mankind!

§ 2.—Already, in specifying the "rural" parts of the country, I have reminded you of one circumstance which curiously distinguishes diphtheria from other infections, namely, its greater frequency among a population that is scattered in small villages and isolated homesteads than among the inhabitants of our crowded towns.

It is almost an axiom of epidemiology that the prevalence of infectious disease in a community bears some direct proportion to the density of the population in the district. This holds good, on the evidence of the Registrar-General's returns of mortality, with regard to epidemic, endemic, and contagious diseases generally. From these diseases the mortality in large towns is double that in the country. In the very first report of the Registrar-General, and much more elaborately in the fifth report, this contrast is exhibited by Dr. W. Farr.

Diphtheria, on the other hand, by the same evidence, affects our rural population more than our towns. For example, during the ten years 1861–1870, the average annual death-rate from diphtheria in London was 1·75 in every 10,000 of the population. In the south-eastern group of districts, in the same period, the rate was 2·00. In the eastern counties, which contain fewer large towns, the rate was 2·95.*

We have yet to learn the cause of this paradoxical inequality of incidence of diphtheria on town and country. Nothing is more certain than that the diphtheritic infection, like others, is favoured by personal intercourse, and inasmuch as personal intercourse is more frequent in towns than in the country, the natural consequence should be a greater prevalence of the disease in the former than in the latter. The actual reversal of this forecast proves that there are other factors at work in diphtheria besides personal communication—factors which operate to the disadvantage of rural as compared with urban communities.

§ 3.—The contrast is well set forth by Dr. Charles Kelly, Medical Officer of Health for the West Sussex Combined District, who draws a marked distinction between two classes of cases known by the same name:—

"1.—Those which seem to depend upon inhaling sewer gas, and where the patient suffers from a form of blood-poisoning. Such cases are generally confined to the house where the local defect in the drainage exists, and there is no extension of the disease. Other inmates may be attacked, because they are all exposed to the common cause. *To this class belong those cases which break out in towns.*

"2.—Those which seem to occur independently of any defective drains or water-supply, and which are most common in bleak and exposed situations. Such cases are met with in clean, well-built cottages, as well as in poorer tenements, and very often the disease breaks out with greater severity among the well-to-do than among those who are badly housed and clad. . . . *This form of disease, unlike the former class, appears in an epidemic form.*" †

§ 4.—It sometimes happens that diphtheria will spread from the country into the immediate neighbourhood of a town, and yet will seem unable to take root or extend itself epidemically in the town itself. A singular case of this kind occurred within my own experience. Diphtheria broke out among

* See "Supplement to the Thirty-fifth Report of the Registrar-General," pp. 4–11.

† Report for 1879, p. 90.

scattered homesteads within a mile or two of a small market-town (Builth) in Brecknockshire. The children affected were attending the village school. Other children from the immediate outskirts of the town attended the same school, and were mixed indiscriminately in school with those who came from the hill-side farms and cottages. Yet not one of the town children caught so much as a sore throat.*

I need hardly say that instances to the opposite effect are by no means infrequent, but in the long-run the general result tells in favour of the towns.

§ 5.—While the rural districts as a whole suffer more severely from diphtheria than the towns, there are found certain tracts of country which are almost wholly free from this disease, and others in which, with brief intermissions, it is almost constantly present, year after year. Among the former I may cite, from the Registrar-General's returns, the district of Fordingbridge in Hampshire, which, with a population of 6409, had only one death from diphtheria in the course of ten years, 1861–1870. Alresford district in the same period lost only two out of 7226. Andover district lost only seven out of 17,361. St. Alban's district, only four out of 20,003.

Compare with these such regions as the country around Chelmsford, where, out of a population of 33,269, diphtheria claimed 170 victims in the same ten years, a death-rate of 5·11 per annum in 10,000. Certain parts of Kent and Sussex, and many districts in Wales, are similarly noted for epidemic diphtheria.

The localities thus affected are for the most part described as cold wet clay lands; but there is evidently great variety in the soils on which diphtheria can prevail, for it is found in full force on the chalk downs of Kent, on the loamy sands and clays of the Sussex Weald, on the alluvium and boulder clay of Essex, on the marls of the new red sandstone, and on the slopes of the slate rocks of Wales.

§ 6.—Within these larger local preferences, it is noticed that certain villages or hamlets appear to be favourite haunts of diphtheria, which reappears from time to time, now in one and now in another. Certain houses in like manner are found to be revisited again and again by this disease. Whether these reappearances are due to a revival of the contagium derived from previous outbreaks in the same place, or to some favouring conditions which these places offer for the development of infection derived from some other quarter, must at present remain a doubtful point.

§ 7.—Much interest attaches to an observation which has often been made by writers on diphtheria who have had opportunities of studying its habits in the country. They have been struck by its frequent occurrence in high exposed situations, in a little village on a hill, or in an isolated cottage or farmstead on the crest of a bare ridge, where the wonder is that any disease at all should arise except such as might be caused by exposure to the violence of wind and weather.

I have already quoted a passage in which Dr. Charles Kelly incidentally alludes to the "bleak and exposed situations" in which rural diphtheria so commonly occurs.†

Among the valuable records of the great outbreak in 1859, published in the Second Report of the Medical Officer of the Privy Council, there is evidence to this effect. Dr. Sanderson there says: "In the district around Launceston the localities which have been most severely visited by diphtheria have certain topographical characters in common, which, although not striking at first sight,

* See also G. H. Fosbroke, S.Sc. Cert. Camb., in *Sanitary Record*, Feb. 15, 1881, p. 296.

† Report for 1879, p. 90.

are well worthy of attention. Their universality is suggestive to the minds of a real relation between diphtheria and the conditions they express. *All the villages invaded by diphtheria, with scarcely an exception, are in elevated situations. Many of them are on the ridges or slopes of the hill.*"

In Kent the same observer noticed that diphtheria prevailed with the greatest fatality in an elevated and exposed tract of country of considerable extent lying on either side of a line drawn from Dover Castle to Canterbury. Dr. Sanderson names certain villages situated in valleys in which diphtheria occurred, but of one of them he noticed that the cases occurred at a farmstead "on the crown of a neighbouring hill." At the same time "in the town of Sandwich, which is completely surrounded by marshes, only a few isolated cases of diphtheria occurred, and in the villages on the marsh . . . not a single case had been met with."

In the same report from which I have quoted these passages there is published a communication from Dr. (now Sir William) Gull on "Diphtheria in Metropolitan Districts." Dr. Gull says: "The conclusion I have formed from the cases I have seen is, that an exposed locality is as liable to diphtheria, and perhaps more liable, than more sheltered spots."

Dr. Slade King and Mr. Wynter Blyth in Devonshire have met with facts telling the same tale even more distinctly. Dr. Slade King found that nine-tenths of the cases (in an outbreak at Ilfracombe in 1873–74) were confined to the higher parts of the town, the lower escaping almost entirely. Mr. Wynter Blyth, studying the same epidemic, which extended into his district from Ilfracombe, says: "The fact of its preference for high, open, and airy situations was extremely marked; the places of selection being isolated houses on lofty hills, supplied with polluted water and surrounded with nuisances." *

§ 8.—In the outbreaks which have come under my own observation, the disease appeared to originate in a high situation in thirteen instances, in a low situation in seven, and midway in four.

The thirteen high sites were the following:—

1.—Killingworth village, on a well-marked ridge of sandstone in a generally level part of Northumberland a few miles north-east of Newcastle.
2.—Staple Cross, a hamlet on a conspicuous hill-top in the east of Sussex, overlooking the valley of the Rother.
3.—Felsted, a village on a hill-top near Dunmow, in Essex.
4.—Henley, a village on high ground a few miles north of Ipswich, in Suffolk.
5.—Chipping, near Clitheroe in Lancashire. At a farmhouse known as Abbot's Barn, on an eminence near the village.
6.—Llanllyfin, near Carnarvon. At a cottage high up on the hill-side.
7.—Manorbier in Pembrokeshire. At a lonely cottage on the crest of a ridge of old red sandstone known as the Ridgeway.
8.—Manorbier in Pembrokeshire. At the village of Jamestown, on a range of high clayey ground.
9.—Mathry, near St. David's in Pembrokeshire. At an isolated cottage on a bleak ridge.
10.—Woolfardisworthy, near Bideford in Devonshire. At an isolated farmhouse on a commanding hill.
11.—Forest Row, near East Grinstead in Sussex. At an isolated farmhouse high up on the north side of Ashdown Forest.
12.—West Hoathly, near East Grinstead, a village on the top of a commanding hill.

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* See "Blyth's Dictionary of Hygiène and Public Health," art. "Diphtheria." London, 1876.

13.—Church Gresley, near Burton-on-Trent, a village on the top of a commanding hill on the border of Leicestershire.

The eight low sites were as follows :—

1.—Blanchland in Northumberland, a village at the bottom of a deep thickly wooded valley.
2.—Killingworth Colliery, in Northumberland, in flat open country.
3.—Castle Gresley near Burton-on-Trent, at the foot of the Church Gresley hill above mentioned.
4.—Wrexham in Denbighshire. At a house in the lower part of the town.
5.—Glanaber, near Beddgelert in Carnarvonshire, a hamlet at the foot of a thickly wooded hill.
6.—Llandewi'rcwn, near Builth in Brecknockshire. At an isolated cottage in an upland valley, at the foot of high hills.
7.—Chipping in Lancashire. At a low-lying spot called the Bottoms, on the bank of a stream.
8.—Penkridge, near Stafford. At a cottage at foot of hill.

The three sites which could neither be called high nor low were :—

1.—Onibury, near Ludlow. At a cottage on the slope of a hill.
2.—Lurgashall in Sussex. On the higher and more exposed part of a wide clayey common.
3.—Raunds, near Thrapstone in Northamptonshire. On the slope of a valley side.

It will be noticed that this list contains instances drawn from many different parts of England and Wales, and so far may be supposed to be free from undue influence of any one set of topographical peculiarities.

On the whole, I think it must be admitted that diphtheria outbreaks in their commencement show a decided, though by no means exclusive, preference for elevated exposed situations.

§ 9.—Intimately related to the question of situation is the question of conveyance of infection by the wind ; and to this point I hope to return shortly. But first I wish to ask your attention to another preference which is manifested by diphtheria. I mean the preference that it shows for a certain season of the year as the time of commencement of its epidemic activity.

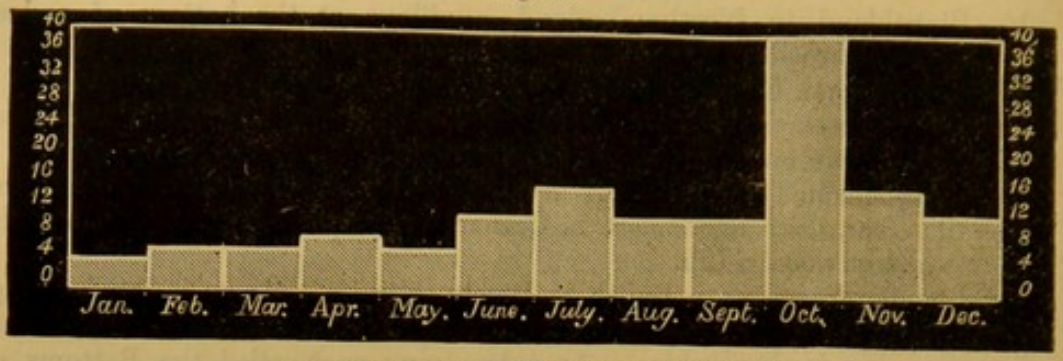
From various sources, but chiefly from the data gathered by Drs. Greenhow and Sanderson, and the medical inspectors of the Local Government Board, I have collected records of 147 outbreaks or isolated occurrences of diphtheria, in which it was possible to fix pretty closely the date of the first attack. The result is curious. The number of outbreaks occurring in

| | | | | | |
|---|---|---|---|---|---|
| January | is 4 | | July | is 16 | |
| February | ,, 6 | = 16 | August | ,, 11 | = 38 |
| March | ,, 6 | | September | ,, 11 | |
| April | ,, 8 | | October | ,, 40 | |
| May | ,, 6 | = 25 | November | ,, 16 | = 68 |
| June | ,, 11 | | December | ,, 12 | |
| Total in first six months, | 41 | | Total in last six months, | 106 | |

These numbers may best be exhibited by the aid of a diagram (fig. 1), in which the remarkable excess of outbreak frequency in October stands conspicuous.

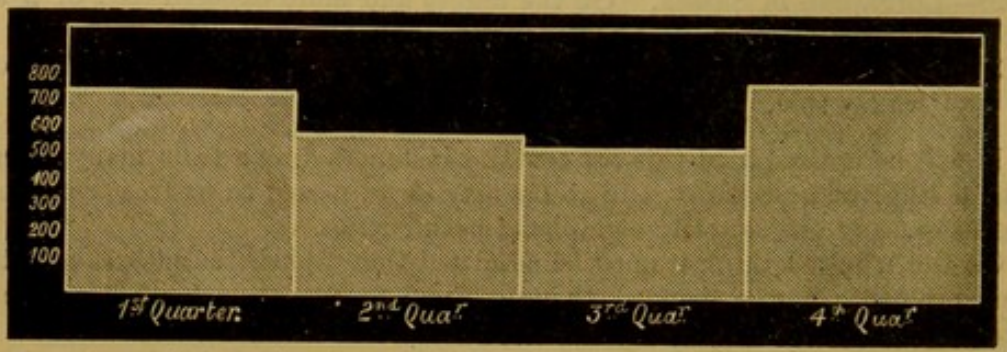
If this curve of outbreak frequency is compared with the curve of mortality from diphtheria, it will be seen that there is a remarkable difference between

Fig. 1.




Monthly Frequency of Diphtheria Outbreaks.

Fig. 2.




Quarterly Mortality from Diphtheria in England and Wales, 1870-79.

Fig. 3.


Monthly Rainfall at Greenwich, average of 65 years.

them. Fig. 2 shows the average diphtheria mortality in each quarter during the ten years 1870–79 for England and Wales, as obtained from the quarterly returns of the Registrar-General. This gives only the main variations of the mortality as between different quarters, and necessarily conceals all minor features. The two curves agree in having their maximum in the fourth quarter, but while in point of frequency the outbreaks sink to a minimum in the first quarter, and thence rise through the second and third to the fourth, the mortality on the other hand is scarcely less in the first quarter than in the fourth, declines further in the second, and does not reach its minimum till the third; so that, while the number of outbreaks that take origin in the first half of the year is less than two fifths of that in the second, the resulting mortality is just about the same in the two halves of the year.

This difference can be simply and naturally explained by the consideration that an epidemic of diphtheria breaking out in the autumn will probably increase in the next month or two, continue raging through the winter, and perhaps not subside till the height of the following summer.

§ 10.—The minor features of the curve of diphtheria mortality for London alone are exhibited in one of a valuable series of diagrams which illustrate a recent paper by Mr. Alexander Buchan on "The Weather and Health of London."* This curve, which is constructed from the weekly returns of deaths in London extending over a great number of years, is in some respects at variance with the curve (fig. 2) for the whole of England and Wales. The London curve shows a more rapid decline from the maximum in the fourth quarter, and apparently reaches its minimum not in the third quarter, but in the second; and the mortality in the second half of the year is evidently much greater than that in the first. I venture to suggest that this may be due to the purely urban character of the data on which this curve is based. We have already seen reason to believe that diphtheria in towns is less prone to take an epidemic form than in the country. The natural consequence would be (*cæteris paribus*) an earlier diminution in the towns of the mortality due to the autumnal outbreaks.

§ 11.—The sudden increase in the number of diphtheria outbreaks in October is, at first sight, highly suggestive of the presence of some fungoid organism depending on the season of the year for its development and reproductive activity.

It is true that after Dr. Burdon Sanderson's experiments with the fluid of a simple peritonitis,† we cannot regard as utterly inconceivable the idea that the human body out of its own elements might develop an infectious disease; and the advocates of the theory of spontaneous development of diphtheria in the human body might point with some degree of assurance to the fact that October is the wettest month of the twelve, and that there is a certain similarity between the curve of monthly rainfall and the curve of monthly frequency of diphtheria outbreaks (see figs. 1 and 3), both having their minimum in the first quarter of the year, both rising in the summer, both falling again before October, and both in October rising to a conspicuous maximum, from which both fall to the end of the year. The only marked disagreement between the two curves is in January; and we must remember that in that month a great deal of the "rainfall" is in the solid form, from which we should not be justified in expecting the same results as from the same element in the liquid form. But the data on which the curve of outbreak frequency has been formed are perhaps too scanty to admit of criticism of the more minute features of the curve.

§ 12.—In view of the general agreement between the two curves in question, it is difficult to avoid the impression that the one is in some way dependent upon the

* See *Nature*, June 16 and 23, 1881, pp. 143 and 173.

† "Infective Processes of Disease," by J. Burdon Sanderson, M.D., F.R.S.

other. It might be suggested that the general preference of diphtheria for a wet clayey district seems to harmonise with the idea that the disease in its origin owns some dependence upon rainfall. The spontaneists would probably urge that exposure to wet caused simple colds and sore throats; that these spontaneously developed, by rapid steps of the kind artificially reproduced by Dr. Sanderson, into a virulent infectious disease. The contagionist, if he regarded diphtheria as caused by a morbific organism capable of multiplication only in the human body, would say that probably the damp favours the vitality of the germs thrown off by a diphtheria patient, and so favours the transmission of the infection to others, and that it also probably acts upon the systems and tissues of those who are exposed to it in a way that makes them more receptive of the diphtheritic infection. He would also with great force urge analogy with those diseases of the zymotic class in which the presence of specific self-multiplying organisms (*bacilli*) in the blood and tissues has been proved, and would express strong confidence that before long the observations of Oertel and Hüter in this direction, in respect of diphtheria, would be confirmed by experimental researches of cultivation and successive inoculation, which alone could set the question at rest.

The spontaneist would perhaps question the specific nature of these bacilli, and speak of them as adventitious and not essential to the morbid process. And he would proceed to ask how, except on the supposition of spontaneous origin, could the fact be accounted for that the great majority of diphtheria outbreaks are perfectly inscrutable and untraceable in their commencement, while in several marked instances they have been preceded by a prevalence of apparently simple sore throat in the surrounding district, suggestive to the skilled observer of a "progressive development of infectiveness."*

§ 13.—At this point a third theory, which may be called the "exogenist," is advanced, which accounts for the apparently spontaneous character of diphtheria outbreaks by supposing that they are caused by an organism which has a life external to the human body as well as within it,—"now fructifying" (to use the expressive words of Mr. Simon †) "in soil or water of appropriate quality, and now the self-multiplying contagium of a bodily disease." The advocate of this theory would claim that it fully accounts for the untraceable origin of diphtheria epidemics: for their excessive frequency in October, and the general correspondence between their monthly frequency and the average monthly rainfall (and at the same time for particular exceptions to this general rule); for the preference this disease exhibits for certain soils and certain localities, and for its infrequency in towns as compared with rural districts; while at the same time the theory maintains solidarity with the advance of microzymic discovery. In this relation the researches of Klebs and Tommasi Crudeli‡ proving (as predicted by Mr. Simon in the paper to which I have already referred) that ague is caused by the inception of a specific microzyme (*Bacillus malariæ*) which has an external habitat in the soil and the air close to the surface of the ground in certain marshy tracts,—researches which have been recently supplemented by successful experiments in the transfer of ague by inoculation,—have especial importance.

§ 14.—But which of these three theories can account for the preference that diphtheria shows, in the commencement of an outbreak, for a dwelling on the top of a hill?

Can the spontaneist plead that children who live in exposed situations are

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* "Remarks on the Origin of Infection," by Dr. Thorne Thorne. *Transactions of the Epidemiological Society*, vol. iv. part ii. p. 244.

† "Essay on Contagion," *Brit. Med. Jour.*, Dec. 20, 1879, p. 974.

‡ *Archiv. für Experimentelle Pathologie*, July and Oct. 15, 1879 (referred to by Mr. Simon in his "Essay on Contagion").

more likely to suffer from the simple throat inflammation whence he supposes the infectious disease to be derived?

This plea is met at once by the objection that in the majority of such cases there has been no antecedent series of sore throats, but the very first attack is of undoubtedly specific nature.

Can the exogenist suppose that hill-tops and bleak ridges are the favourite native haunts of the diphtheria organism before it enters the human body? So unlikely an hypothesis cannot be listened to without a particle of evidence to support it.

What says the contagionist? Can he tell us of any mode by which the diphtheria contagium can with any probability be conveyed undestroyed across the country, and strike by preference the dwellings that are perched on heights that seem securest from any infectious disease?

He is not at fault for an answer, but boldly declares his belief that the contagium is carried by the wind. Mr. Wynter Blyth, now Health Officer for Marylebone, in his valuable paper on "The Prevention and Propagation of Diphtheria," in considering the question as to what is the most probable cause of the outbreak of the so-called spontaneous or sporadic cases, says, "I believe we have fairly strong evidence to accuse the atmosphere, and that such cases may be ascribed with some certainty to the wafting of the specific poison by currents of air."* Mr. Blyth mentions that this theory was held by Dr. C. Budd of North Tawton in Devonshire, from whom he quotes an instructive case, and he cites two cases from his own experience in support of this view.

Another observer, Dr. Edwyn Slade King, Health Officer for Ilfracombe, in his "Notes on the Spread of Diphtheria" (1880), says, "The only reasonable explanation (of the untraceable spread of diphtheria) seems to be that the atmosphere becomes contaminated by contagia from the bodies of the sick, and that currents of air carry the poison with them in their course."

The idea of infection being carried in the air is, I need hardly say, a very old and very popular one; and for very short distances—for example, within the four walls of a room—the idea is accepted and acted upon by every medical practitioner who warns a family to isolate their fever-stricken child. But for distances greater than this, it is perhaps a little too much the habit to neglect the question of aërial infection, probably because of the difficulty of putting it to the proof. Certainly the idea is not necessarily absurd, and I am not acquainted with any set of careful experiments or observations that conclusively disprove it. Yet it is a point of the very highest importance to prove or to disprove, and I trust I may be pardoned for dwelling upon it awhile.

§ 15.—What do we imagine to be the act whereby a healthy person, present in a room which contains a diphtheria patient, himself contracts the disease? Besides direct bodily contact and common use of drinking-vessels and the like, do we not suppose the infection to be caught by inhaling air which contains infectious particles derived from the body of the patient? Doubtless the risk of infection is greatest in the immediate neighbourhood of the sick, where the air must be most thickly charged with infective particles, but there seems no sufficient reason for supposing that one single particle inhaled by a susceptible person may not be enough to give rise to the disease.

Now no one will deny the possibility of the mechanical transportation by the wind, and the chance inhalation by some person at a distance, of a single particle from an infected house. The air is full of particles,—pollen grains, mould spores, soot, globules of smoke tar, and a multitude of nondescript fragments of matter, organic and inorganic (as any one may see by fitting a funnel on a wind-vane so as to direct the wind-stream against a spot of glycerine

* "Prevention and Propagation of Diphtheria," by A. Wynter Blyth, M.R.C.S., &c., *Sanitary Record*, May 15, 1880, p. 407.

on a glass slide, and then examining under the microscope), and at every breath we inhale a cloud of solid matters. Why should not one or two of these particles have come from a distant sick-room, where the windows have been thrown wide open for ventilation,—that is, for the committal of infection germs to the stream of the wind? A cloud of infection germs thus launched upon the air will not necessarily be dispersed, according to any fixed mathematical law, like the radiation of light from a centre, but a good deal of it may go in a body down the wind, somewhat as the smoke of an ocean-steamer may be seen hanging in a streak above the horizon long after the vessel herself has passed out of sight.

But it may be objected that the vitality of the contagium would be destroyed by such exposure in the open air. I very much doubt it. A poison which can retain its virulence for weeks and months exposed to the air in an empty room, would not so readily lose it in floating quietly down the wind for an hour, in which time it might easily traverse a distance of ten or twelve miles,—the average velocity of the wind at Greenwich being from 250 to 300 miles in twenty-four hours. The health officer of a small district, perhaps only a few parishes, knowing probably nothing of an outbreak in a neighbouring district ten miles to windward, if called suddenly to a severe case of diphtheria in a clean, healthy hamlet, might well feel tempted to regard it as a spontaneous development, and take no note of the direction of the wind.

§ 16.—From the foregoing considerations I think we must admit that there is no physical impossibility in aërial infection over a distance of several miles; and supposing such infection can really take place, I think we can see reason why it should take place preferentially in high, bleak situations. The air rushing over a ridge may be likened to water poured out over the edge of a deep basin; and just as in turbid water particles suspended at different levels will all have to pass within very narrow limits (measured vertically) over the basin-edge, so any matters suspended at different levels in the valley air will all be compressed within much narrower limits of level in crowding over a high ridge of land; and, therefore, if diphtheria germs are diffused through the atmosphere at different levels, or, still more, if they rather affect the strata above the lowest,* persons who live on a ridge are more likely to breathe air containing such germs than persons who dwell on lower ground.

§ 17.—The presence of diphtheria germs in the air may perhaps one day be demonstrated. They may be arrested to leeward of an infected house by some form of aspirator, or by suitable funnel-and-vane apparatus, and be submitted to microscopic examination, and to those processes of experimental cultivation and inoculation which have proved so instructive in some other zymotic diseases. But in the meantime, our task is to gather such evidence as we can find that has any bearing on the question.

Mr. Wynter Blyth, in his paper on the "Prevention and Propagation of Diphtheria," has suggested, as a means of testing the truth of this theory, that disease charts should be prepared, in which the direction of the winds and the localities of diphtheria, with the dates affixed to each, should be plotted out. Such a systematic record of observations over a wide area, for which the health officer of a large district has exceptional opportunities, would probably soon solve the question.

§ 18.—As a contribution towards this end, I have collected from reports of the medical inspectors of the Local Government Board a number of instances of diphtheria outbreaks—as many as I could find in which the date of the first attack was pretty accurately known—and have noted the wind facts for the fortnight preceding each outbreak, together with any information I could obtain of recent previous diphtheria in the neighbourhood. The wind facts are taken

* As suggested by Dr. Slade King in "Notes on the Spread of Diphtheria."

from the Registrar-General's weekly returns of births and deaths in London. They are true for Greenwich, and when the wind is steady they may be taken as true for the greater part of the country, but when the wind is shifting they cannot be trusted as strictly applicable to distant places. I should say, however, that in comparing the Greenwich records with the daily weather-charts of the Meteorological Office as published in the *Times*, I have found no such disagreement as would materially affect the question at issue.

The results arrived at appear at first sight highly significant. Of thirty outbreaks thus examined, twenty seem to satisfy the requirements of the wind theory—that is to say, within eight or nine days of the first attack the wind was blowing from a quarter in which diphtheria had recently prevailed, at an average distance of seven miles; in nine the agreement is doubtful as regards the probable source of infection, though the wind conditions appear favourable; in only one are the wind conditions apparently inadequate.

A list of these thirty outbreaks is given below in a tabular form :—

| Place of Outbreak. | Date of First Attack. | Probable Date of Infection. | Direction of Wind. | Previously Infected Place. | Distance in Miles. |
|---|---|---|---|---|---|
| | 1875 | | | | |
| 1. Coggeshall, Essex . . | Nov. 11 | Nov. 6 | W. and W.S.W. | Braintree or Black Notley | 6 |
| 2. South Ash, Kent . . | Dec. 27 | Dec. 20–24 | S.W., S.S.W. and W.S.W. | Sevenoaks subdistrict | 9 |
| 3. Stokesay, Shropshire . | Dec. 24 1876 | Dec. 19, 20 | S. and S.W. | Onibury | 3 |
| 4. Bromfield, Shropshire . | Feb. 3 | Jan. 29–31 | S.E., S., S.S.E. | Ludlow? | 3 |
| 5. Penkridge, Staffordshire | Feb. 14 | Feb. 6–8 | N., N.N.E. | Stafford | 6 |
| 6. Brailes, Warwickshire . | May 6 | April 30–May 3 | N.E., N.N.E. | Tysoe | 5 |
| 7. Padstow, Cornwall . | June 18 | June 14, 15 | S.S.W. | New Quay | 12 |
| 8. Radwinter, Essex . | June 22 1877 | June 15, 16 | S.E. | Coggeshall? | 18 |
| 9. Anlaby, Yorkshire . | April 24 | April 16–18 | E., E.N.E., N.E. | Hull | 3 |
| 10. Swanland, Yorkshire . | May 11 | May 4–6 | E., E.N.E., N.E. | Hull | 6 |
| 11. Denbigh . . . | Aug. 10 | Aug. 2–4 | W.N.W. | Llanfairtalhaiaru | 8 |
| 12. Wrexham, Denbighshire | Nov. 23 1878 | Nov. 18–20 | N.W., W.N.W. | Brymbo | 3½ |
| 13. Cranfield, Bedfordshire | June 26 | June 14, 15 | N.E., N.N.E. | Bedford? | 7 |
| 14. Bleadon, Somersetshire | Oct. 10 | Oct. 5–7 | S.S.E. | Wedmore | 8 |
| 15. Staple Cross, Sussex . | Nov. 4 1879 | Oct. 28–30 | W.N.W. | Rotherfield | 15 |
| 16. Swanscombe, Kent . | July 26 | July 21, 22 | N.W. | Barking | 12 |
| 17. Trottersclifle, Kent . | Sept. 15 | Sept. 8–12 | S.W. | Shoreham subdistrict? | 6 |
| 18. Felstead, Essex . . | Sept. 12 | Sept. 7–9 | S.W. | London? | 30 |
| 19. Henley, Suffolk . . | Nov. 21 | Nov. 12, 13 | N.W. | Needham | 4 |
| 20. Woolfardisworthy, Devonshire | Aug. 30 | Aug. 25–27 | S.W. | Kilkhampton subdistrict | 7 |
| 21. Llandewi'rcwm, Breconshire | Oct. 23 | Oct. 16–19 | N.W., S.W. | ? | ... |
| 22. Ridgeway, Pembrokeshire | Sept. 18 | Sept. 10 | W.N.W. | St. David's subdistrict? | 25 |
| 23. Jamestown, Pembrokeshire | Nov. 17 | Nov. 12, 13 | W.N.W. | St. David's subdistrict? | 25 |
| 24. Llanllyfni, Carnarvonshire | Sept. 26 | Sept. 22, 23 | S.S.W. | ? | ... |
| 25. Glanaber, Carnarvonshire | Oct. 31 | Oct. 26–29 | E.N.E. | Denbigh? | 30 |
| 26. Tremadoc, Carnarvonshire | Dec. 1 | Nov. 24–28 | N.E. | Garddllygaidydydd | 5 |
| 27. Raunds, Northamptonshire | Nov. 15 1880 | Nov. 11 | S.W. | Wellingborough | 7 |
| 28. Forest Row (1) Sussex . | April 1 | Mar. 25–27 | E.N.E. | Tunbridge Wells | 10 |
| 29. Forest Row (2) Sussex . | May 26 | May 17, 18 | N.E. | Forest Row (1) | 1 |
| 30. West Hoathly, Sussex . | Aug. 15 | Aug. 11–13 | E., N.E., E.N.E. | Forest Row | 4 |

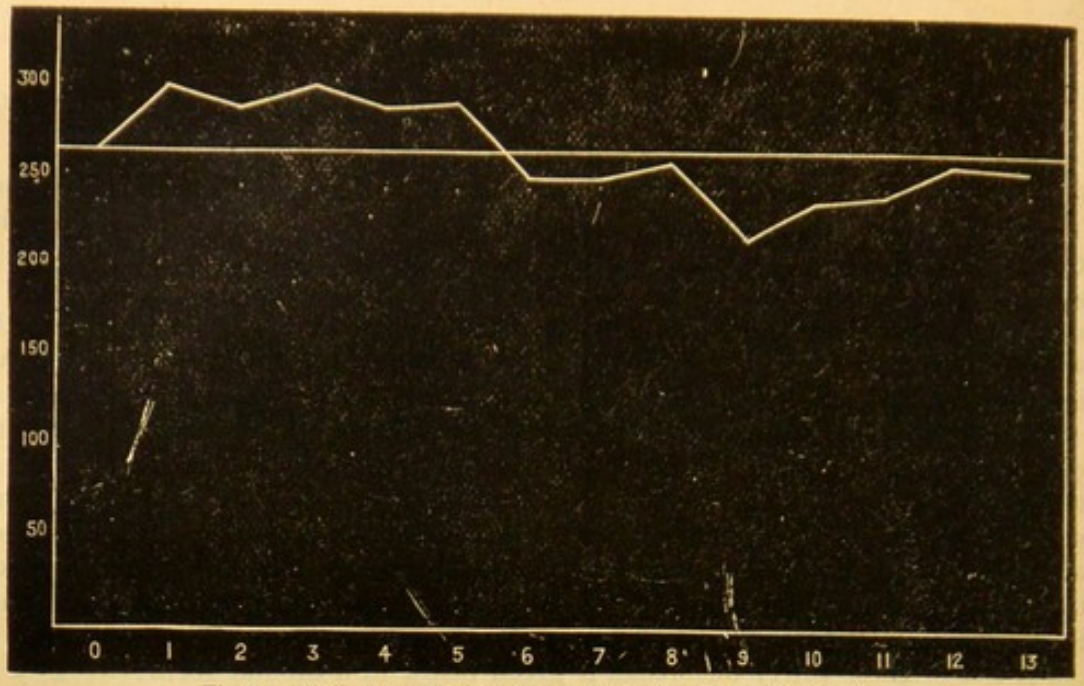


Fig. 4.—Wind-mileage for 13 days preceding 30 Diphtheria dates

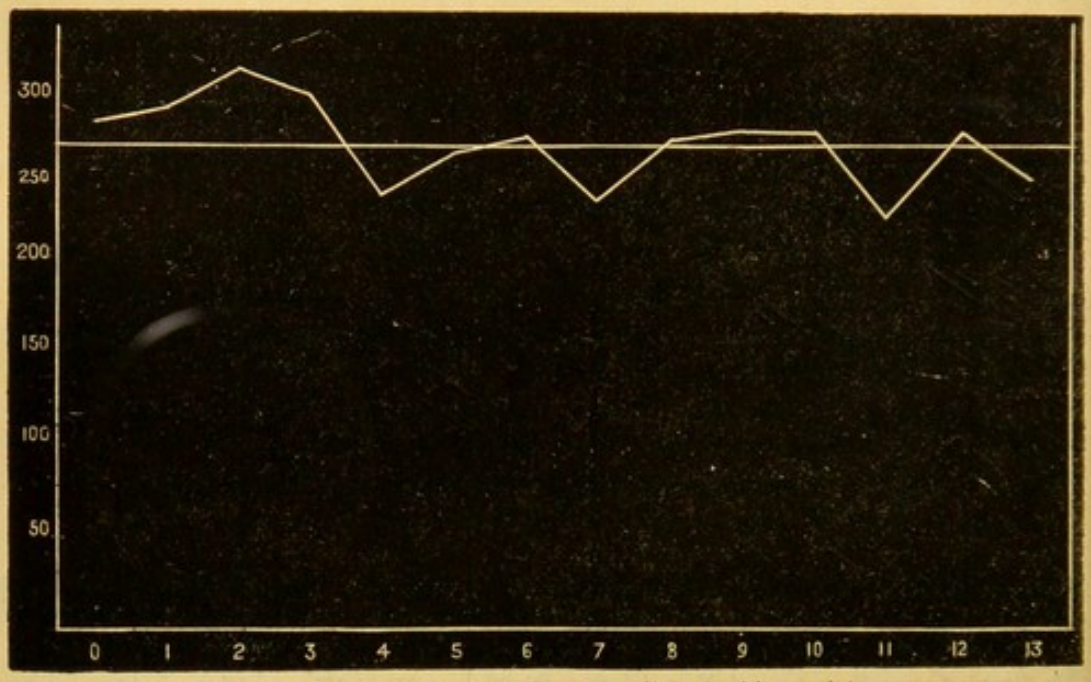


Fig. 5.—Wind-mileage for 13 days preceding 30 arbitrary dates.

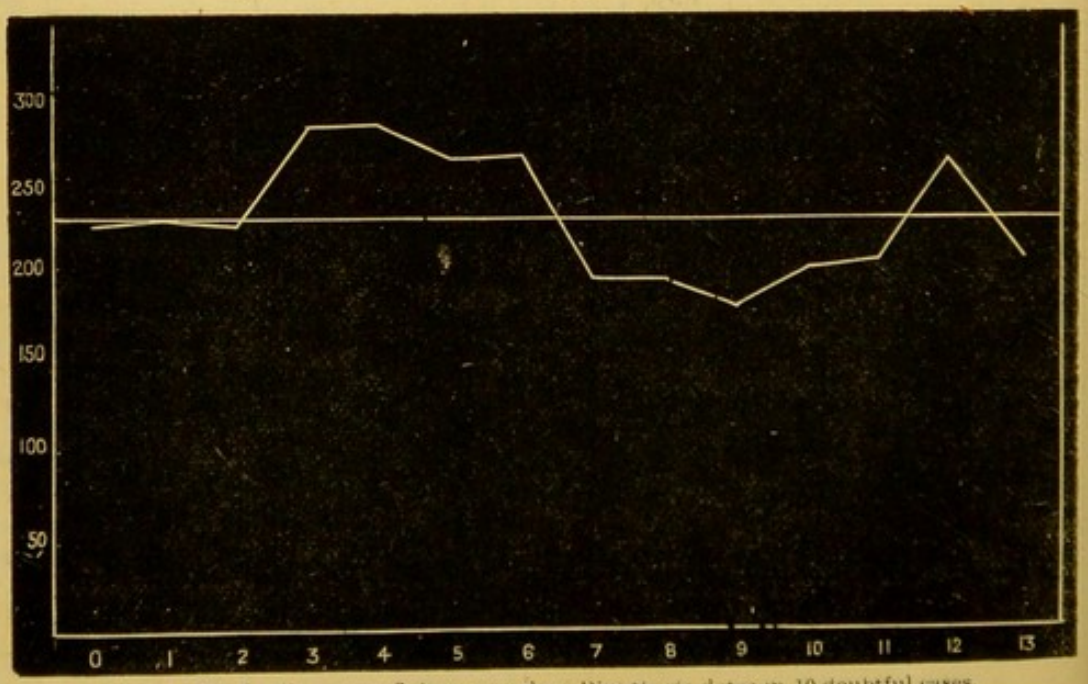


Fig. 6.—Wind-mileage for 13 days preceding Diphtheria dates in 10 doubtful cases.

§ 19.—In estimating the bearing of these facts there are several points to be noticed and several fallacies to be guarded against. At first one is struck by observing how often it happens (in twenty-nine cases out of thirty) that the date of an outbreak is preceded at an interval of a few days (averaging three to six) by a breeze of more than average daily velocity. The impression is confirmed if we take the mean for the whole thirty cases of the number of wind-miles in each day of the fortnight preceding an outbreak; for it then appears that, on an average, during the five days immediately preceding the date of outbreak, the wind has exceeded its average run, while during the eight days before then it was below the average. The curve is shown in fig. 4. Similar results are obtained by taking the mean daily pressure of the wind instead of the number of miles travelled.

The impression thus obtained is somewhat moderated, though not destroyed, if, in order to test its value, we apply a similar analysis to an equal number of dates taken at random. Thus, treating the last day of each month in the years 1878 and 1880, and the 15th of September, October, and November, just as we have treated the above thirty diphtheria dates, we find, in the first place, that in twenty-three out of the thirty arbitrary cases (as against twenty-nine out of the thirty diphtheria dates), there was a breeze of more than average mean daily velocity, preceding the arbitrary date at an interval averaging three to seven days; and taking the mean for the whole thirty, we obtain a curve of wind-mileage (fig. 5) presenting quite as much inequality as that belonging to the diphtheria dates in fig. 4. The inequalities, however, in this second curve (fig. 5) are of a capricious character and unsustained, whereas in the former I think we discern a structure with features large enough to bear a meaning. It seems not unreasonable to suppose that a series of comparatively quiet days followed by a series of comparatively windy ones might first favour the accumulation of the diphtheria poison at infected places, and then its dispersion in considerable quantity to a distance.

We must not expect (even assuming the theory to be true) to find every case exhibiting close agreement with the wind-curve here presented; for, not to mention the probably wide limits of diphtheria incubation, there is the chance of error in the date of first attack; the probability that some of the outbreaks were really due to unnoticed personal or mediate communication; and the possibility that, besides all modes of contagion, the disease may at certain seasons of the year arise through the agency of an external organism, though even then the wind may be the chief disseminating agent. In reference to this last point, the possibility of an exogenous origin in some cases, it may be noticed that of the ten doubtful cases in the list I have given, one occurred in February, two in June, four in September, two in October, and one in November, showing a preponderance of doubtful cases in the autumn.

The wind-curve for these ten cases (fig. 6) does not, however, materially differ from that for the whole thirty (fig. 4); and this agreement of the partial curve with the general curve gives additional value to the latter.

We may further test the representative value of the general curve (fig. 4) by dividing the thirty cases which give that general curve into three groups of ten each, and plotting out partial curves for these three groups. The curve for the first ten cases is shown in fig. 7, that for the second ten in fig. 8, and that for the third in fig. 9.

I think we can recognise in these three curves so much general agreement with one another in the main feature of an ascent about a week before the date of diphtheria outbreak as makes them fairly comparable with, and confirmatory of, the general curve for the whole thirty.

Of the ten doubtful cases above mentioned, two are included in the first

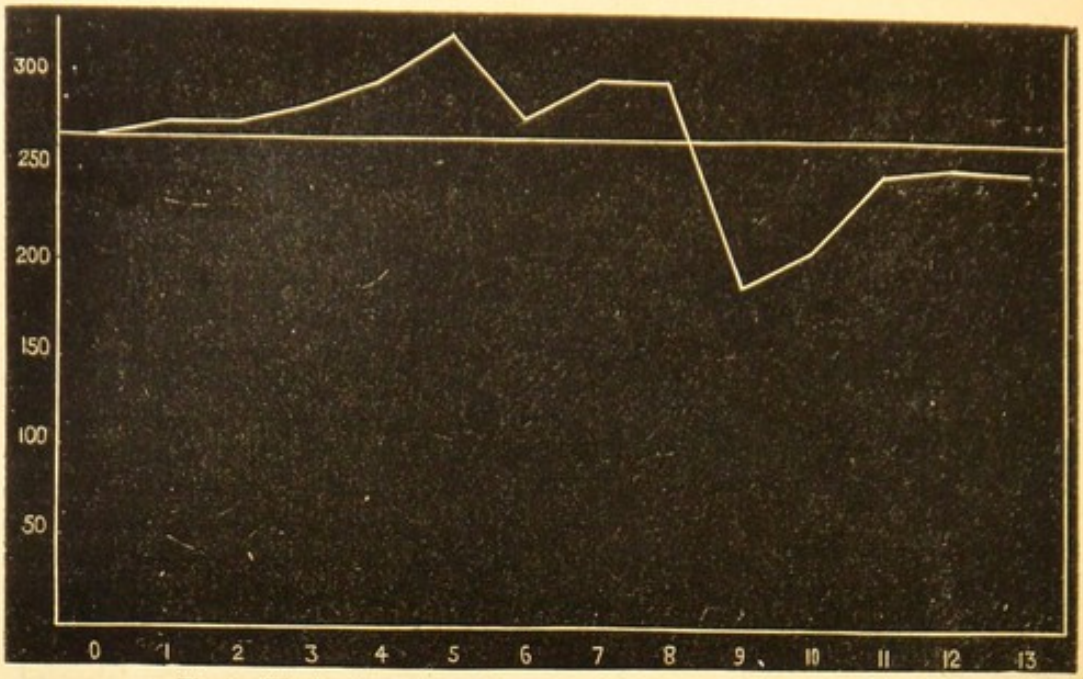


Fig. 7.—Wind-mileage for 13 days preceding *first ten* Diphtheria dates.

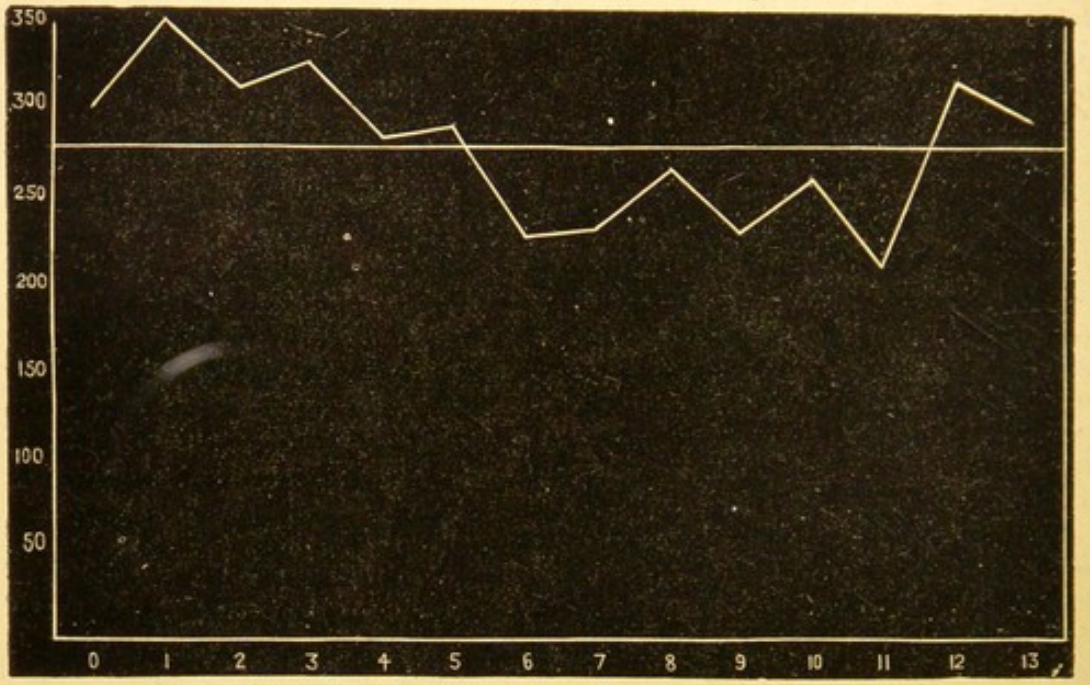


Fig. 8.—Wind-mileage for 13 days preceding *second ten* Diphtheria dates.

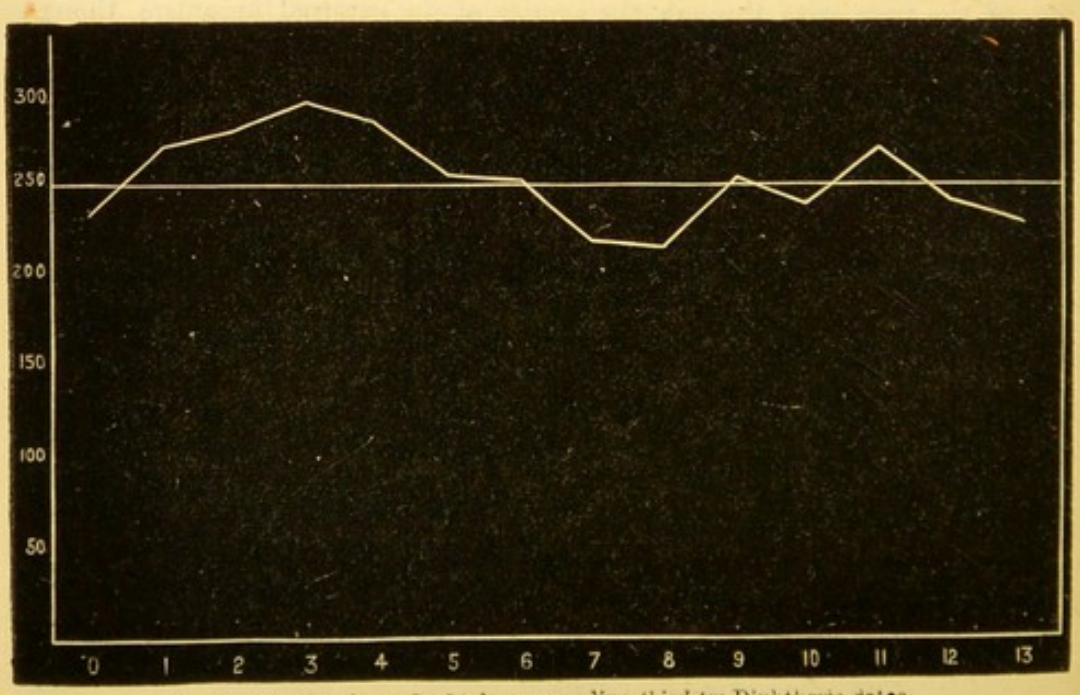


Fig. 9.—Wind-mileage for 13 days preceding *third ten* Diphtheria dates.

group of ten, three in the second, and five in the third. We have, in fact, four partial groups, each confirming the evidence given by the whole.

§ 20.—In applying the wind-theory to any given set of diphtheria dates, there is room for fallacy owing to the frequent variations of the wind within the space of a fortnight. We ought, therefore, by way of test, to apply arbitrary winds (supposed in the direction of previous diphtheria) to the dates of our thirty outbreaks, and see how far the arbitrary winds find place among the winds recorded in the fortnights preceding those dates. This I have tried to do by taking in succession the winds that held good for the several diphtheria dates and applying them to all the other dates in turn. The result is, that I find the arbitrary winds fairly represented among the actual wind-facts in 439 cases out of 900—nearly one-half. In order to correct the exaggeration caused by the greater frequency of south-west winds, I made trial in another way, by applying sixteen arbitrary winds (from the sixteen chief points of the compass) to fifteen arbitrary dates in 1880, with the result that these winds were found among the actual facts in only 78 cases out of 240. The same sixteen arbitrary winds applied to the thirty diphtheria dates found place in 213 cases out of 480.

Each of these trials leaves a decided advantage on the side of the theory, which counts twenty successes to ten doubtful cases.

In each of these trials, however, I have supposed, for each imaginary case, only one wind—that is, only one direction in which diphtheria infection could be conveyed by the air. But this is not quite fair, for among the thirty diphtheria localities there were some that were within reach of two or more possible sources of infection, and every additional focus in a new direction would increase the chance of finding among the wind-facts something that would serve the wind-theory.

We must therefore find some test that shall avoid this fallacy. This may be most simply done by applying to the thirty local conditions of outbreak a new set of thirty arbitrary dates with such wind-conditions as those new dates may entail. For dates let us take in succession (say) the twenty-first of each month in 1879 and 1880, with extra days, say the seventh, in the months September, October, and November in each of those years. The result is that all necessary conditions appear to be fulfilled in only ten instances; in five the probable source of infection is doubtful; in thirteen no probable source can be named; and in the remaining two the wind fails to reach the required standard of velocity. Thus the thirty real diphtheria dates satisfy the theory twice as often as the arbitrary dates; but if the theory were groundless, the results ought to be very much the same with either set of dates.

Thus far, then, we find the theory distinctly confirmed by the tests to which we have submitted it.

§ 21.—There are also points of topography to be noticed, which cannot be included in a test, but which contribute to impress the mind of the observer. In several cases in the list of thirty outbreaks given above, the natural features of the country are such as would favour the passage of the wind from the supposed source of infection to the point of subsequent outbreak. For example, in Case ., the valley of the Blackwater leads straight from Braintree to Coggeshall, and at the latter place it turns southward, so that a west wind at this point would have to force itself up the sloping ground on which the village lies. Similar conditions are present in Cases 5 (Penkridge in relation to Stafford), 15 (Staple Cross in relation to Rotherfield), 19 (Henley in relation to Needham), 27 (Raunds in relation to Wellingborough), 28 (Forest Row in relation to Tunbridge Wells), and 30 (West Hoathly in relation to Forest Row).

Many of the places in the list are in notably high situations, and therefore

liable in an especial degree to be touched by anything that is carried by the wind. Such are Nos. 2 (South Ash), 8 (Radwinter), 15 (Staple Cross), 16 (Swanscombe), 18 (Felstead), 19 (Henley), 20 (Woolfardisworthy), 22 (Ridgeway), 23 (Jameston), 28 (Forest Row, 1), 29 (Forest Row, 2), and 30 (West Hoathly).

Others, somewhat less exposed, are on hill-slopes; only seven out of the thirty are in low situations.

I am far from supposing that the instances and arguments I have here brought forward are sufficient to establish the theory of aërial diphtheria spread; but I think they are sufficient, in conjunction with earlier observations, to give the theory a right to be disproved, and, pending disproof, to be provisionally entertained.

§ 22.—Upon the practical importance of the question in respect of other infectious diseases as well as diphtheria I need not dwell; but I feel bound to return for a moment to the speculative considerations from which I have diverged, to ask what is the bearing of the "aërial infection" theory on the question of the nature of the diphtheritic contagium? If true, it seems to cut away much of the ground from under the "exogenist" theory, by explaining those very cases of apparently spontaneous origin of diphtheria which that theory was expressly framed to account for. It does not, however, explain "progressive development" of simple sore throat into diphtheria; nor does it explain the preference of diphtheria for certain regions, and for a certain season of the year. In short, it does not decide the question. Its bearing is practical rather than ætiological, and its aid might be invoked almost equally by all three of the supposed theorists whose divergent views we have had under consideration.

For my own part, if I may venture to express an opinion, where so many inquirers better qualified than myself are yet in doubt, I still incline, though perhaps with lessened confidence, to regard as most probable the hypothesis that diphtheria is caused by an organism naturally haunting air or water in certain regions, but also capable of living and multiplying in the human body.

I feel that I must apologise at once for the length of this paper, for its inconclusive character, and for the omission of many points of great interest and importance in the dealings of this most subtle and capricious disease.

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## *On the Nature and Propagation of Diphtheria.*

Dr. A. Jacobi, New York.

Diphtheria is pre-eminently a disease of childhood. Of 300 cases of laryngeal diphtheria in which I performed tracheotomy, but one was over 50, one over 30, two over 13 years old. Of 501 deaths by diphtheria in Vienna in 1868, but one was 62 years old. Of 10,000 cases of diphtheria in England, according to Thursfield (*Lancet*, August, 1878), 90 per 1000 were under 1 year, 450 per 1000 from 1 to 5 years, 260 from 6 to 10, 90 from 11 to 15, 50 from 16 to 25, 35 from 26 to 45, 25 were 45 or over.

Of 9354 reported deaths of diphtheria in the United States—the States of Massachusetts and New Jersey, and the territories of Wyoming, Arizona, and New Mexico, and the district of Columbia not being corrected, the reports being those of physicians, and not of State Boards—only 445 occurred under one year, 778 in the second, 1034 in the third, 1011 in the fourth, 991 in the fifth; thus, 4219 under five years (45 per cent.), 3057 between the sixth and tenth, that is,

as many as in the three years beginning with the third and ending with the fifth year. Between 10 and 15 there were 1275, about as many as in the first and second together, and but few more than in either the third, or the fourth, or the fifth year. There were between

| | | | | | |
|---|---|---|---|---|---|
| 15 | and | 20 | years | 329 | less than in the first year. |
| 21 | „ | 25 | „ | 112 | „ „ |
| 26 | „ | 30 | „ | 57 | „ „ |
| 31 | „ | 35 | „ | 28 | „ „ |
| 36 | „ | 40 | „ | 28 | „ „ |
| 41 | „ | 45 | „ | 18 | „ „ |
| 46 | „ | 50 | „ | 13 | „ „ |
| 51 | „ | 55 | „ | 10 | „ „ |
| 56 | „ | 60 | „ | 10 | „ „ |
| 61 | „ | 65 | „ | 4 | „ „ |
| 66 | „ | 70 | „ | 2 | „ „ |
| 71 | „ | 75 | „ | 2 | „ „ |
| 75 | „ | 80 | „ | 2 | „ „ |
| 81 | „ | 85 | „ | 2 | „ „ |
| 86 | „ | 90 | „ | 1 | „ „ |

The age from 91 and over furnishes no mortality from diphtheria.

The cause why children are so much more exposed than adults is mostly anatomical. The mucous membrane of the oral cavity in the child is more succulent, and often the seat of catarrh and inflammation, which dispose to, or are transformed into, a diphtheritic process. The nasal cavities are small, and often affected with catarrh, and but too often cleanliness is insufficiently attended to. With the exception of nutritional changes and atrophy, every change in the mucous membrane predisposes to diphtheria.

The tonsils are large and the pharynx narrow, so that poisons, either chemical or parasitic, will find an easy resting-place. Besides, the size of the child brings it into closer contact with the exhalations of the soil, or sewers, or gutters. This fact is of some account when we remember that the diffusion of gases is very great, and the distance of 50 or 80 centimetres more or less is of importance; and though a direct connection between sewer-gas and diphtheria has not been proven, the general health cannot but suffer from such exposure. Moreover, after the local affection has taken place, the size and number of the lymphatics, which can be more easily injected in the child than in the adult according to Sappey, and have lateral and intercommunicating stomata according to Schenck, aggravate the case by accelerating the appearance of general symptoms.

The number of cases and deaths in the first year is relatively small, as the above figures show. Of those who are taken under a year of age, most are either less than three or more than seven months old. The reason is this: from the end of the third to the seventh month there is a very copious secretion from the infant's mouth, with the result of washing away noxious importations.

*Predisposition.*—Diphtheria once endured, creates a predisposition. It may be that the mucous membrane undergoes, in the first attack, such changes of a catarrhal or merely anatomical (epithelial) character as to fix that predisposition. It may be that in the enlarged glands diphtheria poison is stowed away for occasional resuscitation, as we know it to be, for instance, in syphilis.

Even that form of diphtheria which is as little apt to give rise to constitutional symptoms as it is very liable to terminate fatally has been known to return. Guersant, Gill, Quincke have performed tracheotomy twice on the same patients.

Predisposition is not only individual, but clings to families. Certainly the

cause must be anatomical; for there is a family pharynx, a family tonsil, a family larynx, a family nasal contour, as there is a family foot or hand. I have seen cases of laryngeal diphtheria, membranous croup, destroying a number of children of the same family, not in the same year or house, but in the course of many years, and while living in different dwellings. In other families I have seen perfect, or almost perfect, immunity for more than twenty years.

Of more than 300 cases of laryngeal diphtheria on which I performed tracheotomy, and of the same number observed by me besides, the majority were males. Of the recoveries the majority occurred in females. Other forms of diphtheria exhibited in my experience but slight differences in regard to the sexes of the patients.

*Causes.*—Sudden changes in temperature are apt to give rise to diphtheria by producing catarrhal effusions. These changes occur in different months in different countries, or parts of countries. Thus the difference of opinion relating to the prevalence of diphtheria is easily explained, and no invariable season law, as proposed by Besnier, exists. Persons with a predisposition to catarrh and diphtheria will do well to avoid cold and dampness.

Exhalations of sewers, gutters, privies are frequently accused of being of themselves causes of diphtheria. If they be, their direct influence has not been so well proven as is the case in regard to dysentery and typhoid fever. They will swell the number of cases of diphtheria sufficiently by merely lowering the general standard of health of the individual or the community, thus facilitating the final appearance of diphtheria, which is produced by a specific poison. The cases reported as having occurred after drinking contaminated water or milk require farther study. In the latter cases particularly, the facility of carrying the disease in clothing, tools, and vessels, is an established fact.

The nature of the poison I do not wish to make the subject of an extensive discussion here. It is impossible to finish it, or to convince a sceptic, in the course of a discussion. The comparative value of the proofs for the chemical or parasitic nature of the poison can be studied only by referring to a number of facts and a vastness of literature. This I have tried to do in my treatise on diphtheria (New York, 1880). I cannot come to the conclusion that the victory has been won by the parasitists. The war-cry, "No bacteria, no diphtheria," is not any more justified than that fermentation or putrefaction depend on bacteria only. The presence of bacteria in the diphtheritic blood has not been proven. Bacteria are destroyed by alkaline solutions. There is no theoretical ground for assuming that preventing the bacteria contained in a diphtheritic patch from making their way through the underlying mucous membrane will *per se* prevent general diphtheritic infection of the system. On the contrary, the septic and putrid poison is claimed by A. Hiller as distinctly chemical. Of the same nature—viz., chemical—is in all probability the poison of those of the infectious and contagious diseases in which the presence of a characteristic parasite at a certain period of the disease is a recognised fact, as anthrax and relapsing fever. Its regular presence does not prove its being *the* cause.

*Entrance of the poison, local or general.*—It may be, and frequently is, quite local. Diphtheria may commence in a local wound, or on an exposed mucous membrane or denuded cutis, and from that origin infect the whole system. It may also, though not affecting the system of the patient who suffered locally, be thus communicated to others with all its severe consequences.

Besides, diphtheria is a frequent occurrence after operations, mainly in the pharynx. Within from sixteen to twenty hours the surface of an excised tonsil is covered with diphtheritic membrane, with or without fever, and with or without symptoms of general diphtheria. In common times and under favourable

circumstances this does not occur, but it so does whenever there is an epidemic of diphtheria; that means, when everybody is so predisposed that a slight additional cause is sufficient to produce the visible symptoms. Thus these operations ought to be postponed until after the disappearance of an epidemic. Thus in these latter cases the local cause supersedes the constitutional predisposition. In them, without the latter the former could not act; without the former the disease would not break out.

*The constitutional origin of diphtheria* is claimed by many for the following reasons:—It bears all the marks of a disease both infectious, contagious, and inoculable. It occurs in epidemics. If it occurs sporadically, the same must be said of measles or scarlatina, which nevertheless do not lose their standing in nosology. It differs from the latter inasmuch as it runs no typical course, and instead of destroying or diminishing, is apt to increase future predisposition.

It does not differ from them in this, that the epidemics are of varying intensity.

There are cases in which general symptoms precede, or appear to precede, the local ones, and in which the whole of the course is followed by general adynamic symptoms, and nervous prostration or paralysis.

Albuminuria is also of a more frequent and earlier occurrence than the height of the temperature would explain.

The constitutional origin, when assumable, must be explained by the direct unobstructed admission of the poison into the lungs, particularly in those cases in which nose, mouth, and pharynx are large and healthy and the tonsils small. The fifth part of the whole amount of blood is within the lungs, the exposed surface of their alveoli, of about two thousand square feet, being very permeable, for it consists only of basement membrane and fine capillary network, with a thin epithelial layer, which, moreover, is sometimes absent.

The constitutional symptoms depend on the amount of poison absorbed, the power of resistance of irritability on the part of the organism, and *very much* on the rapidity of individual metamorphosis and elimination. Protracted slow poisoning with diphtheria or typhoid poisons, accompanied with *rapid and steady elimination*, will result at most in gradual discomfort, malaise, or the ambulatory type of the disease, and not give rise to the outbreak of a serious illness.

Thus the entrance of diphtheria may be local or constitutional—that is, through the lungs or mixed; and accordingly the general symptoms, such as fever, lassitude, &c., may follow or precede the visible local symptoms.

Diphtheria is very contagious, the contagion may be transmitted by the patient, his dwelling, furniture, towel, his visitors, all his surroundings. The cracks in the floor, the carpets and curtains, may communicate the disease after months.

It may be transmitted, too, from some classes of animals in whom it has been observed—kittens, hens, and domestic fowls generally, calves, cows. Perhaps also through milk.

In dwellings it follows the current of air, mostly upwards.

Mild cases may give rise to serious ones, and *vice versa*.

The period of incubation lasts from two to fourteen days. Fresh wounds, however, will produce membranes within sixteen hours. On mucous membranes denuded of all their diphtheritic deposits I have seen thick membranes constructed in seven hours.

| State. | SEX AND AGE. | | | | | | | | | | | | | | | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | Total. | Under 1. | 1. | 2. | 3. | 4. | Total under 5. | 5 to 10. | 10 to 15. | 15 to 20. | 20 to 25. | 25 to 30. | 30 to 35. | 35 to 40. | 40 to 45. | 45 to 50. | 50 to 55. | 55 to 60. | 60 to 65. | 65 to 70. | 70 to 75. | 75 to 80. | 80 to 85. | 85 to 90. | 90 to 95. | 95 and over. | |
| Alabama | 23 | 2 | 5 | 7 | 4 | 2 | 20 | 1 | ... | 2 | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... |
| | 31 | 2 | 2 | 6 | 5 | 1 | 16 | 8 | 3 | 3 | ... | ... | ... | ... | 1 | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... |
| | 5 | ... | 1 | 2 | 1 | ... | 4 | ... | 1 | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... |
| | 3 | ... | ... | ... | ... | ... | ... | 3 | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... |
| Arkansas | 23 | 4 | 2 | 4 | 3 | 2 | 15 | 6 | 1 | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... |
| | 17 | 4 | 1 | ... | 3 | 3 | 11 | 4 | 1 | ... | 1 | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... |
| | 3 | ... | 1 | ... | ... | ... | 1 | 1 | ... | ... | ... | ... | ... | 1 | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... |
| | 5 | 2 | ... | 2 | ... | ... | 4 | ... | ... | ... | ... | 1 | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... |
| California | 23 | 1 | 1 | 2 | 3 | 5 | 12 | 4 | 3 | 2 | 1 | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... |
| | 36 | ... | 3 | 6 | 4 | 4 | 17 | 12 | 4 | 1 | 1 | ... | 1 | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | 1 |
| Colorado | 29 | 2 | 3 | 3 | 1 | 4 | 13 | 6 | 4 | ... | 3 | 3 | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... |
| | 35 | 2 | 3 | 3 | 3 | 7 | 18 | 14 | 3 | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... |
| | 37 | 3 | 3 | 3 | 4 | 6 | 19 | 12 | 5 | ... | ... | 1 | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... |
| Connecticut | 56 | 1 | 3 | 9 | 11 | 5 | 29 | 21 | 2 | ... | 1 | 1 | ... | ... | ... | ... | ... | 1 | ... | ... | 1 | ... | ... | ... | ... | ... | ... |
| | 2 | 2 | ... | ... | ... | ... | 2 | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... |
| Dakota | 18 | ... | ... | 2 | 2 | 1 | 5 | 11 | 2 | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... |
| | 23 | ... | 2 | 1 | 4 | 3 | 10 | 5 | 6 | 1 | ... | 1 | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... |
| Delaware | 8 | ... | 3 | ... | 1 | 4 | 8 | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... |
| | 8 | ... | ... | 2 | ... | ... | 2 | 4 | 1 | 1 | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... |
| | 1 | ... | ... | ... | ... | ... | ... | 1 | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... |
| Florida | 4 | ... | 1 | 2 | 1 | ... | 4 | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... |
| | 2 | 1 | ... | ... | ... | ... | 1 | 1 | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... |
| | 1 | 1 | ... | ... | ... | ... | 1 | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... |
| | 1 | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... |
| Georgia | 31 | 3 | 4 | 5 | ... | 3 | 15 | 10 | 1 | 1 | ... | 1 | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | 1 |
| | 28 | ... | 5 | 7 | 3 | 4 | 19 | 8 | 1 | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | 3 |
| | 7 | ... | 2 | ... | 1 | ... | 3 | 4 | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... |
| | 17 | 1 | 1 | 4 | 1 | ... | 7 | 8 | 1 | 1 | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... |
| Idaho | 1 | ... | ... | ... | ... | ... | ... | ... | 1 | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... |
| | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... |
| Illinois | 242 | 7 | 21 | 34 | 25 | 30 | 117 | 75 | 33 | 10 | ... | 3 | 1 | ... | ... | ... | 1 | ... | ... | ... | ... | ... | ... | ... | ... | ... | 2 |
| | 228 | 17 | 26 | 23 | 27 | 24 | 117 | 61 | 32 | 10 | 3 | 3 | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | 2 |
| | 3 | ... | ... | ... | ... | ... | ... | 3 | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... |
| Indiana | 174 | 11 | 12 | 22 | 16 | 31 | 92 | 48 | 20 | 6 | ... | 3 | ... | ... | 1 | 1 | ... | 1 | ... | 1 | ... | ... | ... | ... | ... | ... | 1 |
| | 259 | 19 | 25 | 37 | 42 | 22 | 145 | 65 | 27 | 9 | 3 | 1 | 1 | ... | ... | 1 | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | 7 |
| | 2 | ... | ... | ... | ... | ... | ... | 1 | ... | ... | ... | ... | 1 | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... |
| | 1 | ... | ... | ... | 1 | ... | 1 | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... |
| Iowa | 381 | 12 | 25 | 36 | 50 | 43 | 166 | 102 | 68 | 19 | 8 | 2 | 2 | 1 | 1 | 1 | ... | 1 | 1 | ... | ... | 2 | ... | ... | ... | ... | 7 |
| | 368 | 11 | 19 | 43 | 33 | 27 | 133 | 118 | 81 | 19 | 8 | 1 | 1 | 2 | ... | ... | 1 | ... | ... | ... | ... | ... | ... | ... | ... | ... | 4 |
| | 1 | ... | ... | ... | ... | ... | ... | ... | ... | 1 | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... |
| Kansas | 205 | 8 | 24 | 17 | 21 | 26 | 96 | 68 | 23 | 10 | ... | ... | ... | 1 | 1 | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | 6 |
| | 180 | 5 | 16 | 24 | 16 | 18 | 79 | 62 | 24 | 6 | 3 | ... | 1 | 1 | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | 4 |
| | 6 | 1 | ... | ... | 1 | 1 | 3 | 2 | 1 | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... |
| | 3 | ... | ... | ... | ... | ... | ... | 2 | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | 1 |
| Kentucky | 55 | 2 | 7 | 9 | 8 | 7 | 33 | 12 | 9 | 1 | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... |
| | 52 | 1 | 8 | 10 | 6 | 3 | 28 | 14 | 6 | ... | 1 | ... | 1 | 1 | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | 1 |
| | 6 | ... | 2 | 1 | 1 | ... | 4 | 2 | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... |
| | 5 | 2 | 1 | 1 | ... | 1 | 5 | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... |
| Louisiana | 5 | ... | 2 | ... | 1 | ... | 3 | 2 | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... |
| | 6 | ... | ... | ... | 1 | 1 | 2 | 3 | ... | ... | 1 | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... |
| | 7 | ... | ... | 2 | 1 | 2 | 5 | 2 | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... |
| Maine | 7 | 2 | 1 | ... | 1 | ... | 4 | ... | 1 | 1 | ... | ... | ... | ... | ... | ... | ... | 1 | ... | ... | ... | ... | ... | ... | ... | ... | ... |
| | 85 | 3 | 4 | 5 | 11 | 9 | 32 | 33 | 13 | 3 | ... | ... | 1 | ... | ... | 1 | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | 2 |
| | 96 | 1 | 6 | 12 | 12 | 8 | 39 | 28 | 16 | 7 | 2 | 2 | 1 | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | 1 | ... | ... | ... |
| Maryland | 38 | 2 | 1 | 6 | 7 | 8 | 24 | 10 | 3 | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | 1 |
| | 41 | ... | 7 | 6 | 2 | 6 | 21 | 14 | 6 | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... |
| | 2 | ... | ... | 1 | ... | 1 | 2 | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... |
| | 2 | ... | ... | 1 | 1 | ... | 2 | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... |
| Michigan | 246 | 11 | 21 | 29 | 23 | 32 | 116 | 86 | 35 | 3 | 1 | 1 | ... | 1 | ... | 1 | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | 2 |
| | 271 | 9 | 18 | 29 | 14 | 32 | 102 | 96 | 43 | 10 | 5 | 4 | 2 | ... | ... | 1 | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | 8 |
| | 2 | ... | ... | ... | ... | ... | ... | 1 | 1 | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... |
| Minnesota | 112 | 7 | 6 | 4 | 11 | 10 | 38 | 41 | 23 | 5 | 1 | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | 4 |
| | 130 | 3 | 5 | 6 | 9 | 11 | 34 | 53 | 23 | 12 | 5 | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | 3 |
| Carry forward | 3698 | 164 | 303 | 428 | 396 | 407 | 1699 | 1156 | 528 | 144 | 48 | 29 | 13 | 8 | 4 | 6 | 2 | 4 | 1 | 1 | 1 | 2 | ... | 1 | ... | ... | 60 |

| State. | SEX AND AGE. | | | | | | | | | | | | | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | Total. | Under 1. | 1. | 2. | 3. | 4. | Total under 5. | 5 to 10. | 10 to 15. | 15 to 20. | 20 to 25. | 25 to 30. | 30 to 35. | 35 to 40. | 40 to 45. | 45 to 50. | 50 to 55. | 55 to 60. | 60 to 65. | 65 to 70. | 70 to 75. | 75 to 80. | 80 to 85. | 85 to 90. | 90 to 95. | 95 and over. | |
| ght forward | 3698 | 164 | 303 | 428 | 396 | 407 | 1699 | 1156 | 528 | 144 | 48 | 29 | 13 | 8 | 4 | 6 | 2 | 4 | 1 | 1 | 1 | 2 | ... | 1 | ... | ... | 60 |
| | 3 | 1 | 1 | ... | 1 | ... | 3 | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... |
| sippi . . | 10 | 1 | 3 | 2 | ... | 1 | 7 | 2 | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | 1 |
| | 6 | ... | 3 | 2 | ... | 1 | 6 | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... |
| | 7 | 1 | 1 | 1 | 1 | 1 | 5 | 1 | ... | 1 | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... |
| | 115 | 16 | 13 | 19 | 13 | 16 | 77 | 24 | 8 | ... | ... | 1 | ... | ... | 2 | 1 | ... | ... | ... | ... | ... | ... | 1 | ... | ... | ... | 1 |
| uri . . . | 130 | 13 | 22 | 17 | 19 | 12 | 83 | 38 | 5 | ... | 2 | 1 | ... | ... | ... | ... | 1 | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... |
| | 9 | 2 | 2 | 3 | ... | ... | 7 | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | 1 | ... | ... | ... | 1 |
| | 11 | ... | 2 | ... | 1 | 1 | 4 | 6 | 1 | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... |
| ana . . . | 5 | ... | ... | ... | 1 | ... | 1 | 2 | 1 | 1 | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... |
| | 7 | 1 | 2 | ... | ... | ... | 3 | 3 | 1 | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... |
| aska . . . | 137 | 3 | 9 | 16 | 16 | 22 | 66 | 48 | 18 | ... | 1 | ... | 1 | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | 3 |
| | 136 | 4 | 10 | 11 | 11 | 10 | 46 | 52 | 30 | ... | 3 | ... | 1 | 1 | 2 | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | 1 |
| da . . . | 1 | ... | ... | ... | ... | ... | ... | 1 | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... |
| | 2 | ... | ... | 1 | ... | 1 | 2 | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... |
| Hampshire | 52 | 4 | 3 | 7 | 6 | 3 | 23 | 15 | 7 | 3 | 1 | 1 | ... | ... | 2 | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... |
| | 64 | 4 | 3 | 2 | 4 | 3 | 16 | 23 | 12 | 7 | 1 | ... | 1 | 1 | 2 | 1 | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... |
| | 445 | 20 | 40 | 57 | 44 | 36 | 197 | 145 | 62 | 17 | 8 | 1 | ... | ... | 1 | ... | 1 | 1 | ... | 1 | ... | ... | ... | ... | ... | ... | 11 |
| York . . | 418 | 7 | 31 | 44 | 52 | 34 | 168 | 142 | 60 | 16 | 9 | 3 | 3 | 5 | 1 | 1 | 1 | 1 | ... | ... | ... | ... | ... | ... | ... | ... | 8 |
| | 4 | 2 | ... | ... | ... | ... | 2 | 2 | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... |
| | 132 | 15 | 21 | 17 | 11 | 15 | 79 | 30 | 12 | 3 | ... | 1 | ... | ... | ... | ... | ... | ... | 2 | ... | ... | ... | ... | ... | ... | ... | 5 |
| a Carolina . | 139 | 8 | 12 | 16 | 16 | 17 | 69 | 40 | 13 | 5 | ... | 2 | ... | 1 | ... | 1 | 1 | ... | ... | ... | ... | ... | ... | ... | ... | ... | 7 |
| | 18 | 2 | 1 | 3 | 3 | 2 | 11 | 6 | ... | ... | ... | ... | ... | ... | ... | 1 | ... | .. | ... | ... | ... | ... | ... | ... | ... | ... | ... |
| | 19 | ... | ... | 4 | 4 | 3 | 11 | 6 | ... | ... | 1 | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | 1 |
| | 365 | 7 | 28 | 40 | 45 | 42 | 162 | 134 | 40 | 8 | 5 | 2 | ... | 4 | ... | ... | 1 | ... | ... | ... | ... | ... | ... | ... | ... | ... | 9 |
| . . . . . | 346 | 10 | 17 | 35 | 35 | 24 | 121 | 141 | 50 | 13 | 4 | 2 | 1 | 3 | 1 | ... | 1 | 1 | ... | ... | ... | ... | ... | ... | ... | ... | 8 |
| | 3 | 1 | ... | ... | ... | ... | 1 | 1 | 1 | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... |
| n . . . | 21 | ... | 3 | 1 | 2 | 2 | 8 | 10 | 1 | 1 | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | 1 |
| | 24 | ... | 2 | 3 | 3 | 3 | 11 | 9 | 4 | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... |
| | 801 | 33 | 77 | 103 | 90 | 88 | 391 | 258 | 92 | 22 | 3 | 6 | 2 | 1 | ... | ... | 1 | 1 | ... | ... | ... | ... | ... | ... | ... | ... | 24 |
| sylvania . | 878 | 24 | 53 | 67 | 97 | 93 | 334 | 330 | 142 | 34 | 9 | 3 | 2 | 1 | 3 | 1 | ... | 2 | ... | ... | ... | ... | ... | ... | ... | ... | 17 |
| | 2 | ... | ... | ... | 2 | ... | 2 | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... |
| | 6 | ... | ... | ... | ... | ... | ... | 4 | ... | 2 | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... |
| | 19 | 2 | 2 | 4 | ... | 3 | 11 | 6 | ... | ... | ... | ... | 1 | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | 1 |
| e Island . | 24 | 1 | ... | 1 | 6 | 6 | 14 | 8 | ... | 1 | 1 | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... |
| | 2 | 1 | ... | ... | ... | ... | 1 | 1 | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... |
| | 2 | ... | 1 | 1 | ... | ... | 2 | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... |
| | 27 | 2 | 3 | 3 | 6 | 2 | 16 | 8 | 3 | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... |
| Carolina . | 26 | 2 | 1 | 4 | 5 | 2 | 14 | 7 | 3 | ... | ... | ... | ... | ... | ... | ... | ... | ... | 1 | ... | ... | ... | ... | ... | ... | ... | 1 |
| | 9 | 1 | 5 | ... | ... | 1 | 7 | 2 | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... |
| | 11 | 2 | 2 | 2 | 3 | 1 | 10 | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | 1 |
| | 83 | 4 | 11 | 11 | 17 | 12 | 55 | 23 | 4 | ... | ... | ... | 1 | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... |
| ssee . . | 87 | 8 | 8 | 10 | 3 | 13 | 42 | 32 | 11 | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | 2 |
| | 16 | 4 | 1 | 2 | 1 | 1 | 9 | 5 | 1 | ... | ... | ... | ... | ... | ... | ... | 1 | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... |
| | 23 | 4 | 2 | 4 | ... | 5 | 15 | 3 | 3 | ... | 1 | 1 | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... |
| | 25 | 3 | 2 | 6 | 3 | 6 | 20 | 2 | 1 | 1 | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | 1 |
| . . . . . | 23 | 1 | 5 | 5 | 2 | 5 | 18 | 4 | ... | ... | 1 | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... |
| | 2 | ... | 1 | ... | ... | ... | 1 | 1 | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... |
| | 5 | ... | 1 | 1 | ... | ... | 2 | 1 | ... | 1 | 1 | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... |
| | 57 | 3 | 6 | 1 | 6 | 12 | 28 | 22 | 2 | 4 | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | 1 |
| . . . . . | 67 | ... | ... | 5 | 10 | 9 | 24 | 20 | 20 | 1 | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | 2 |
| ont . . . | 39 | 3 | 1 | 9 | 2 | 2 | 17 | 12 | 4 | 2 | 2 | 1 | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | 1 |
| | 34 | 1 | 4 | 2 | 2 | 2 | 11 | 12 | 7 | 4 | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... |
| | 59 | 2 | 10 | 5 | 6 | 6 | 29 | 15 | 10 | 1 | 1 | ... | 1 | ... | ... | 1 | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | 1 |
| hia . . . | 64 | 1 | 6 | 1 | 5 | 5 | 18 | 26 | 12 | 4 | 1 | 1 | ... | 1 | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | 1 |
| | 9 | ... | ... | 1 | 1 | 1 | 3 | 4 | ... | ... | ... | 1 | ... | 1 | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... |
| | 14 | ... | 1 | ... | 3 | 1 | 5 | 5 | 2 | 1 | ... | ... | ... | 1 | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... |
| | 23 | ... | 1 | 3 | 1 | 2 | 7 | 7 | 6 | 2 | 1 | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... |
| ington Ter. | 14 | ... | ... | 1 | ... | 1 | 2 | 2 | 4 | 6 | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... |
| | 2 | ... | ... | ... | 1 | ... | 1 | 1 | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... |
| | 98 | 2 | 9 | 5 | 4 | 20 | 40 | 35 | 16 | 4 | 1 | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | 2 |
| Virginia . | 81 | 2 | 5 | 14 | 9 | 6 | 36 | 28 | 11 | 5 | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | 1 | ... | ... | ... | ... | ... | ... |
| | 3 | ... | ... | ... | 1 | 1 | 2 | 1 | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... |
| | 2 | ... | ... | ... | 1 | ... | 1 | 1 | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... |
| nsin . . | 191 | 7 | 18 | 25 | 21 | 15 | 86 | 56 | 32 | 7 | 5 | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | 5 |
| | 198 | 5 | 10 | 9 | 19 | 14 | 57 | 86 | 35 | 8 | 2 | 1 | 1 | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | 8 |
| Grand total | 9354 | 405 | 778 | 1034 | 1011 | 991 | 4219 | 3057 | 1275 | 329 | 112 | 57 | 28 | 28 | 18 | 13 | 10 | 10 | 4 | 2 | 2 | 2 | 2 | 1 | ... | ... | 185 |

## DISCUSSION.

Dr. SQUIRE, London: In answer to Dr. Airy, I would say that exposure of germs to air oxidates and destroys them. I think the remark of Dr. Jacobi, that infection diminishes rapidly with distance, is well founded. One of his reasons for the predisposition of children to diphtheria is their greater proximity to the infecting source. I would extend this to their nearness to their nurses and to other children, as well as to sewers and to exhalations of the ground. I quite agree with Dr. Jacobi as to the important part played by all the causes of individual predisposition in determining an attack of diphtheria, and am glad to see that he also fully admits the influence of contagion, and the care necessary against its conveyance by clothes and utensils.

Dr. ASHBY, Manchester: I would draw attention to the curious distribution of diphtheria, including its varieties. Thus, in comparing large cities, diphtheria or diphtheritic croup is abundantly common in London and Liverpool, but comparatively rare in Manchester. In the latter city a few cases appear in the suburbs, but very few in the city or adjoining township of Salford. The reason for this is far from obvious. With regard to the question of the conveyance of the diphtheritic poison by means of wind, while no doubt dust, smut, and various vegetable spores are carried long distances in this way, it is contrary to all our ideas concerning the spread of zymotic diseases. Modern treatment of specific febrile affections, and the precautions taken to prevent their spread, very strongly point to the destruction of the poison, whether "vital" or chemical, by the abundant mixture of fresh air.

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## *Ueber den Zusammenhang der Chorea mit dem Rheumatismus in besonderer Beziehung auf die Herzgeräusche, die so häufig Chorea begleiten.*

Dr. STEFFEN, Stettin.

Seit geraumer Zeit haben verschiedene Autoren annehmen zu müssen geglaubt, dass ein bestimmter Zusammenhang zwischen Chorea und Gelenk-Rheumatismus bestehe. Man hat dies zum Theil daraus gefolgert, dass man die eine Krankheit der anderen hat vorausgehen oder derselben folgen sehen. Man hat sogar Fälle zur Unterstützung herangezogen, in welchen Pausen von Monaten und Jahren zwischen beiden Processen bestanden haben. Einen wichtigeren Grund für die Wechselbeziehung beider Processe fand man in der Endocarditis, welche nach der Annahme vieler in der Regel beide Krankheiten begleiten und so in gewisser Weise ein Bindeglied zwischen beiden darstellen solle.

Es scheint mir, dass der Zusammenhang der Chorea mit dem Gelenk-Rheumatismus in verschiedenen Gegenden sehr verschieden ist, in manchen beobachtet man, dass dieselben sehr häufig zu einander in Beziehung stehen, in anderen, wie z. B. in Stettin und Umgegend, wird dies äusserst selten gefunden. Gelenk-Rheumatismus kommt bei uns im kindlichen Alter sehr selten vor, wogegen man Chorea, namentlich in der ärmeren Klasse, ziemlich häufig auftreten sieht, so dass Exemplare dieser Krankheit fast stets in dem, unter meiner Leitung stehenden, Kinderspital zu finden sind.

Was das Verhältniss des Gelenk-Rheumatismus zur Endocarditis betrifft, so

kommen Fälle des ersteren ohne die letztere vor, wenigstens ohne dass diese klinisch nachgewiesen werden könnte. Man hat dann das Recht anzunehmen, dass sie nicht vorhanden gewesen ist, weil man wohl im Stande ist, die Diagnose derselben zu machen.

Andererseits kommen Fälle einfacher Endocarditis zur Beobachtung, in welchen von Rheumatismus gar keine Rede sein kann. Man kann unmöglich in solchen Fällen, in welchen keinerlei Zeichen von Rheumatismus existiren, annehmen, dass die Endocarditis eine rheumatische Ursache habe und in Folge von Erkältung entstanden sei. Wollte man sich solchen Auffassungen anbequemen, so würde man den Rheumatismus als eine äusserst häufige Ursache der verschiedensten Krankheitsprocesse hinstellen können.

Die Häufigkeit des Auftretens einfacher Endocarditis ohne Complication mit Rheumatismus, scheint in den verschiedenen Gegenden verschieden zu sein. Wahrscheinlich würde dieser Process häufiger zur Beobachtung kommen, wenn man es sich zur Regel machte, bei jeder ernstlichen Krankheit das Herz genau und mehrfach zu untersuchen. In dem 3ten Bande, N. F. des Jahrbuchs für Kinderheilkunde 1870, p. 393, habe ich in einem Aufsatze: "Beiträge zur Lehre von den Herzkrankheiten," bereits mehrere Fälle einfacher Endocarditis zusammengestellt, welche ohne vorgängigen Rheumatismus unter meinen Augen im Spital zur Entwickelung gekommen und nach Wochen mehr oder minder rückgängig geworden waren, ohne dass sich irgend ein Symptom von Rheumatismus gezeigt hatte.

Wenn wir also vom Rheumatismus absehen, und bloss die Beziehungen der Chorea zur Endocarditis in das Auge fassen, so findet sich bei aufmerksamer Beobachtung, dass beide Krankheiten häufig vereint vorkommen und dass in unserer Gegend dies vielleicht die Regel ist. Ich habe nie Chorea auf Grundlage von Endocarditis sich entwickeln, sondern letztere nur im Verlauf der ersteren auftreten sehen. Es scheint mir, dass in den Fällen, in welchen man die Endocarditis als die primäre Krankheit gesehen zu haben glaubt, die unscheinbaren Anfänge der Chorea der Beobachtung entgangen sind.

Da die pathologische Anatomie des Processes der Chorea für uns im Dunkeln liegt, so lässt sich eben so wenig ein Grund für die Complication mit Endocarditis nachweisen.

Um die klinischen Beziehungen zwischen beiden Processen nachzuweisen, ist zunächst eine genaue Feststellung der Erscheinungen, mit welchen die Endocarditis auftritt und verläuft, nothwendig. Bekanntlich sind wir in der glücklichen Lage, im kindlichen Alter die Lage und Grösse des Herzens mit der grössten Genauigkeit festsetzen zu können. Bei hinreichender Uebung kann man sogar in den meisten Fällen die Grenze zwischen den Vorhöfen und Ventrikel bestimmen. Die Methode dieser Untersuchung findet sich in dem oben angeführten Aufsatz ausführlich auseinandergesetzt. Hat man diese Untersuchung genau ausgeführt, so ist es nicht schwierig, die pathologischen Veränderungen des Herzens zu constatiren.

Jede frisch auftretende Endocarditis ist von Fieber (40° bis sogar zu 42° hinauf) begleitet. Die örtlichen Erscheinungen differiren, je nachdem die Entzündung von vorn herein die Klappen oder das Endocardium der Wandungen betroffen hat. Es braucht kaum erwähnt zu werden, dass es sich in der Regel nur um Erkrankung der linken Herzhälfte handelt.

Bei der primären Affection der Klappen findet sich der erste Ton über der Herzspitze von einem meist sausenden Geräusch begleitet. Ein präsystolisches Geräusch wird im kindlichen Alter selten beobachtet. Der zweite Pulmonalarterienton ist stärker accentuirt. Nach wenigen Tagen folgt eine Dilatation des Herzens, welche hauptsächlich oder fast ausschliesslich die linke Herzhälfte betrifft. Diese Dilatation wird bedingt durch eine seröse Durchfeuchtung und

Erschlaffung der Herzwandungen, deren Endocardium von dem entzündlichen Process mit ergriffen ist. Mit dem Beginn der Dilatation rückt die Herzspitze mehr nach aussen und auch etwas nach unten. Seltener findet man die Herzdämpfung am rechten Rande des Sternum verbreitert. Diesem Befunde entsprechend, lässt sich eine Zunahme der Breite und Länge des Herzens nachweisen. Entweder erlangt eine solche Dilatation in wenigen Stunden ihr Maximum, oder sie schreitet allmählig von Tag zu Tage fort, bis sie ihren Höhepunkt erreicht hat. Es lassen sich Zunahmen der Herzlänge um 1–3½ C., der Herzbreite um ½–2 C. constatiren. Den geringsten Grad von Dilatation findet man, wenn dieselbe sich in wenigen Stunden entwickelt und damit ihren Höhepunkt erreicht hat.

Wenn der entzündliche Process zuerst das Endocardium der Wandungen ergreift, so kann derselbe in nicht seltenen Fällen auf diese Region beschränkt bleiben. Ich habe einen solchen Fall bei einem Knaben von 13 Jahren beobachtet und die Diagnose durch die Autopsie bestätigen können. Der verstorbene Professor Traube theilte mir mündlich mit, dass er ebenfalls einen derartigen Vorgang post mortem constatirt habe. Bei vorhandener Chorea trat durch einen hinzutretenden Process der exitus lethalis ein. Die Endocarditis hatte sich durch keine Veränderung der Herztöne noch sonstige auffällige Symptome kundgegeben.

Die Fälle, welche ich im Spital sich unter meinen Augen habe entwickeln sehen, zeichneten sich neben lebhaftem Fieber zuerst durch eine Zunahme der Herzdämpfung aus. Die Herzspitze überschritt die, bei der Aufnahme festgesetzte Stelle nach links und etwas nach unten, seltener trat eine Verbreiterung der Herzdämpfung auf der rechten Seite des Sternum auf. In der Regel entwickelt sich diese Dilatation nicht plötzlich, sondern lässt sich schrittweise von Tag zu Tage nachweisen, lässt auch zuweilen nach und nimmt wieder zu. Die Zunahme der Breite und Länge des Herzens hält die gleichen Maasse ein wie in den Fällen, in welchen der Process primaer die Klappen ergriffen hatte. Bei der lebhaften und frequenten Action des Herzens sind die Töne lauter, namentlich der erste, welcher auf der Höhe des Processes so verstärkt sein kann, dass der zweite schwächere kaum gehört wird. Zuweilen ist jedoch schon um diese Zeit, jedenfalls aber mit dem Nachlass des Fiebers der zweite Pulmonalarterienton stärker accentuirt.

Wenn die Entzündung auf das Endocardium der Wandungen beschränkt bleibt, so vergehen mehrere Stunden, in der Regel Tage, bis die Dilatation einen solchen Grad erreicht, dass eine mechanische Insufficienz der Klappen bewirkt wird. Da der linke Ventrikel die allein oder hauptsächlich ergriffene Region ist, so wird man das entstehende Sausegeräusch in der Regel auf den linken Ventrikel und speciell auf die Herzspitze beschränkt finden. In seltneren Fällen findet man es weiter über die Herzdämpfung verbreitet. Selbstverständlich ist es systolisch.

Ich habe in einem derartigen Fall, welcher in meinem Spital beobachtet wurde, die Diagnose durch die Autopsie bestätigen können. Es war dies ein Knabe von 13 Jahren, welcher mit einer jauchigen Periostitis der linken Tibia und der rechten Ulna aufgenommen wurde. Chorea war freilich nicht zugegen, doch instruirt dieser Fall die Verhältnisse der Endocarditis. Der erste Herzton war von einem schwachen sausenden Geräusch begleitet. Die Zunahme der Breite und Länge des Herzens gestaltete sich folgendermassen:

| | BREITE. | LÄNGE. |
|---|---|---|
| Am 1sten Tage . . . | 8¼ | 12¼ |
| Am 3ten Tage . . . | 8¼ | 14 |
| Am 5ten Tage . . . | 8½ | 15 |
| Am 6ten Tage . . . | 10 | 15½ |

Seitdem fand keine Zunahme der Dilatation statt. Der Spitzenstoss, welcher bei der Aufnahme die linke Mamillarlinie kaum nach aussen überschritt, befand sich auf der Höhe der Dilatation $3\frac{1}{2}$ C. ausserhalb dieser Linie. Bei der Autopsie fand sich das Herz dilatirt, die Klappen normal, dagegen war Endocarditis der Wandungen des linken Ventrikel vorhanden, verbunden mit zahlreichen kleinen Apoplexieen.

Wenn sich der Process der Endocarditis von den Wandungen auf die Klappen verbreitet, so findet zunächst keine Veränderung der Symptome statt, aus welcher man diesen Vorgang nachweisen könnte.

Endocarditis, welche sich auf Grundlage von Chorea ohne Complication mit Rheumatismus entwickelt, wird wohl in der Regel rückgängig. Ich habe keinen Fall mit tödtlichem Ausgang beobachtet. Blache (Essai sur les maladies du cœur chez les enfants p. 128) führt den Fall eines zwölfjährigen Mädchens an, welches an Gelenk-Rheumatismus, Chorea, dann secundär an Endocarditis und Pericarditis erkrankte und nahezu geheilt entlassen wurde. Derselbe Autor referirt dagegen über einen analogen Fall bei einem Mädchen von vierzehn Jahren, welches von denselben Processen heimgesucht war und an doppelseitiger Pleuritis zu Grunde ging. Die Autopsie entsprach dem klinischen Befund. Die Mitralklappe war insufficient und geschrumpft.

Man sieht in der Regel mit dem Rückgängigwerden der Endocarditis die Symptome der Chorea allmählig oder auch mehr plötzlich in ihrer Heftigkeit nachlassen. Das Fieber, welches im Beginn der Endocarditis am hochgradigsten war, lässt nach. Die Herzthätigkeit ist in der ersten Zeit eine unregelmässige, wechselt fast jeden Augenblick ihren Rhythmus. Die Herztöne zeigen Verschiedenheiten in Bezug auf ihre Intensität. Nicht selten findet man den ersten Ton gespalten und den zweiten Pulmonalarterienton abwechselnd stärker accentuirt.

Mit dem Rückgängigwerden der Endocarditis nimmt allmählich die Dilatation des Herzens und die Vergrösserung der Herzdämpfung ab. Die Herzspitze kehrt allmählig vollständig oder nahezu an die Stelle zurück, welche sie vor der Endocarditis eingenommen hatte. Die Breite und Länge des Herzens kehren allmählig ebenfalls vollständig oder nahezu zur Norm zurück.

Als Beispiele dienen folgende Messungen von einfacher Endocarditis.

Ein Knabe von 10 Jahren, welcher ohne Chorea an einer frischen Endocarditis erkrankt war, hatte am Tage der Aufnahme in das Kinderspital:

| | BREITE. | LÄNGE. |
|---|---|---|
| 1ster Tag . . . . | $5\frac{3}{4}$ | 8 |
| 2ter Tag . . . . | $6\frac{1}{2}$ | $8\frac{1}{2}$ |
| 7ter Tag . . . . | 7 | 10 |
| 10ter Tag . . . . | $7\frac{3}{4}$ | $11\frac{1}{2}$ |
| 15ter Tag . . . . | 7 | 10 |
| 45ster Tag . . . . | $6\frac{1}{2}$ | $9\frac{1}{2}$ |

Ein Mädchen von 10 Jahren ohne Chorea an Endocarditis erkrankt:

| | BREITE. | LÄNGE. |
|---|---|---|
| 1ster Tag . . . . | 7 | 10 |
| 4ter Tag . . . . | $7\frac{3}{4}$ | $10\frac{1}{2}$ |
| 10ter Tag . . . . | 8 | 12 |
| 13ter Tag . . . . | 7 | 10 |
| 46ster Tag . . . . | 7 | $10\frac{1}{2}$. |

Wenn die Endocarditis nur die Wandungen betraf, die Klappeninsufficienz also mechanischer Natur war, so verliert das den ersten Herzton begleitende

Sausegeräusch mit der Abnahme der Dilatation an Intensität, um allmählig ganz zu schwinden. Gleichen Schritt hält hiermit die Abnahme der verstärkten Accentuation des zweiten Pulmonalarterientones.

Wie vollständig die Erscheinungen der Endocarditis schwinden können, beweisen folgende Fälle.

Ein Mädchen von 9 Jahren ist in diesem Jahr zum 4ten Mal wegen Chorea im Kinderspital aufgenommen worden. In jedem der drei letzten Jahre war die Erkrankung aufgetreten. Die beiden ersten Male hatten sich keine Symptome von Endocarditis gezeigt, nur fand sich bei dem zweiten Mal bei normaler Grösse, Lage und Tönen des Herzens, die Action desselben hier und da unregelmässig und zuweilen etwas verlangsamt. Im dritten Mal war Endocarditis mit deutlicher Dilatation des Herzens aufgetreten und vollständig rückgängig geworden, wie der Befund bei der vierten Aufnahme des Kindes nachweist. Es wurde vollkommen normale Lage und Grösse des Herzens constatirt. Die Action desselben war nicht immer regelmässig, in Folge davon die Intervale zwischen den Tönen oft ungleich, der erste Ton war zeitweise von einem schwachen sausenden Geräusch begleitet. Zwei und eine halbe Woche nach der Aufnahme steigerte sich die Chorea in dem Maasse, dass die Kranke das Bett hüten musste. Zwei Wochen später fand sich der erste Ton zeitweise von einem dumpfen klingenden Geräusch, wie von dem einer Basssaite begleitet. Wenige Tage später, am 5. März d. J., Abends, traten unter lebhaftem Fieber die Zeichen von Endocarditis auf. Die Herzaction war ausserordentlich beschleunigt, der erste Ton sehr stark accentuirt, der zweite kaum zu vernehmen. Am folgenden Tage konnte bereits Dilatatio cordis nachgewiesen werden. Das Herz hatte seine Lage nicht verändert, dagegen hatte sich die grosse Herzdämpfung nach links verbreitert, die Herzspitze stand über 1 Centimeter ausserhalb der linken Mamillarlinie, während sie sich bei der Aufnahme genau in derselben befunden hatte. Bei beträchtlicher Beschleunigung der Herzaction war der erste Ton verschärft, der zweite beinahe ausfallend. Am 7ten März erschienen beide Töne gleich stark. Vom folgenden Tage ab war der erste Ton in der Regel von einem deutlich sausenden Geräusch begleitet und der zweite Pulmonalarterienton stärker accentuirt. Die Herzerweiterung schwankte, indem die Herzspitze bald nur $\frac{1}{2}$, in der Regel aber über 1 Cent. die linke Mamillarlinie überschritt. Die höchste Höhe des Fiebers hatte 400 betragen. Am 4ten Tage der Endocarditis begann das Fieber nachzulassen, schwand aber erst vollständig am 7ten Tage. Mit dem Schwinden des Fiebers mässigten sich die Erscheinungen der Chorea. Allmählig liess die Dilatation des linken Ventrikel nach. Am 31sten März war das Herz zu derselben Grösse zurückgekehrt, welche bei der Aufnahme der Kranken constatirt werden konnte, die Herzspitze stand wieder in der linken Mamillarlinie, der erste Ton war klar, ohne irgend welches Geräusch, und die Accentuation des zweiten Pulmonalarterientones geschwunden. Seitdem sind diese Verhältnisse dieselben geblieben und die Symptome der Chorea immer mehr zurückgegangen.

Ein Knabe von 14 Jahren wird am 28sten Mai 1880 mit Chorea aufgenommen. Die Untersuchung des Herzens weist normale Verhältnisse nach, namentlich steht die Herzspitze etwas nach innen von der linken Mamillarlinie. Die Erscheinungen der Chorea sind sehr hochgradig. Zwei Tage später treten rheumatische Schmerzen ohne wesentliches Fieber auf, welche wechselsweise verschiedene Gelenke befallen, das Herz ganz intact lassen und nach Natr. salicyl. vollkommen schwinden. Etwa vierzehn Tage, nachdem der Rheumatismus vollständig beseitigt war, entwickelt sich Endocarditis der Wandungen des linken Ventrikels unter mässigem Fieber. Die Herzdämpfung ist nach allen Richtungen um $\frac{1}{2}$–$1\frac{1}{2}$ C. vergrössert, der erste Ton ist überall von einem lebhaften Sausegeräusch begleitet, der zweite Pulmonalarterienton verstärkt, die

Herzspitze steht 1 Cent. nach aussen von der linken Mamillarlinie. Nach anderthalb Wochen lässt sich eine allmählige Abnahme der Dilatatio cordis nachweisen. Sechs Tage später hat die Herzdämpfung nach allen Richtungen um 1–1½ C. abgenommen, also etwa die Maasse wieder erreicht, welche bei der Aufnahme des Kranken constatirt worden waren. Die Herzspitze stand in der linken Mamillarlinie, überall konnte man bei dem ersten Ton ein schwaches Sausgeräusch nachweisen. Bis zu der 3 Wochen später erfolgten Entlassung des Kranken waren diese Verhältnisse unverändert geblieben. Mit dem Rückgängigwerden der Endocarditis lassen die Erscheinungen der Chorea schnell nach, um bald gänzlich zu schwinden.

Es bleibt in diesem Fall, der mir später nicht wieder zu Gesicht gekommen ist, unentschieden, ob es sich bloss um eine wandständige Endocarditis gehandelt hat, oder wie weit die Klappen, namentlich die Mitralis, von dem entzündlichen Process betroffen worden sind.

Wie vollständig eine wandständige Endocarditis rückgängig werden kann, beweist schliesslich noch der folgende Fall, in welchem es sich nicht um Chorea sondern um Gelenk-Rheumatismus mit Endocarditis handelt.

Ein Mädchen von 12 Jahren wurde am 7ten Juli 1880 mit heftigem Rheumatismus im linken Hüft- und Knie- und im rechten Hand-Gelenk, aufgenommen. Lebhaftes Fieber. Lage und Grösse des Herzens normal. Schwaches Sausegeräusch bei dem ersten Herzton, der zweite Pulmonalarterienton nicht verstärkt. Allmähliger Rückgang der Erscheinungen bis Ende September, wo die Kranke fällt und sich in Folge davon eine neue heftige Entzündung des linken Kniegelenks entwickelt. Vier Tage nach diesem Unfall lässt sich von Neuem ein schwaches Sausegeräusch constatiren, welches den ersten Herzton begleitet und am deutlichsten über dem linken Ventrikel gehört wird. Vierzehn Tage nach dem Beginn des Recidius lässt sich eine beträchtliche Vergrösserung der Herzdämpfung nachweisen. Der Spitzenstoss, welcher sich innerhalb der linken Mamillarlinie befand, zeigte sich jetzt 2–3 Centimeter ausserhalb derselben, der erste Ton war von einem starken Sausegeräusch begleitet. Nachdem diese Erscheinungen bis Ende October ziemlich constant gewesen waren, trat Anfang November ein allmähliger Nachlass derselben ein, die Herzdämpfung verkleinerte sich schrittweise, bis der Spitzenstoss einen halben Centimeter innerhalb der linken Mamillarlinie sich befand. Gleichzeitig wurde das sausende Geräusch schwächer und war völlig geschwunden, als das Herz seine normale Grösse wieder erreicht hatte. Anfang December waren vollständig normale Verhältnisse des Herzens eingetreten und sind dieselben bis Ende März d. J., innerhalb welcher das Kind mehrfach untersucht worden ist, unverändert geblieben.

Diese Beispiele beweisen, dass eine Endocarditis der Wandungen so vollständig rückgängig werden kann, dass keinerlei von der Norm abweichende Erscheinungen zurückbleiben. Zuweilen hält sich noch, nachdem die Grösse des Herzens wieder normal geworden ist, eine Zeit hindurch ein schwaches sausendes Geräusch, welches den ersten Ton begleitet und allmählig auch vollständig schwindet. Wenn die Endocarditis nicht auf die Wandungen beschränkt war, sondern primär oder secundär die Klappen ebenfalls ergriffen hat, so können dieselben, wenn der Process nicht zu ausgedehnt und zu hochgradig war, wahrscheinlich wieder schlussfähig werden. Wenigstens ist durch Autopsieen bei Erwachsenen festgestellt, dass nach Schrumpfung eines Klappenzipfels wieder vollständiger Schluss des Ostium dadurch bewirkt werden kann, dass die andere oder die anderen Klappenzipfel, wenn drei vorhanden sind, sich allmählig mehr und mehr ausbuchten.

Wenn nach Endocarditis der Klappen der Process rückgängig wird, aber Schrumpfung der befallenen Zipfel eintritt, so sieht man zunächst ebenfalls die

Grösse der Herzdämpfung abnehmen und allmählig mehr oder minder nahezu die normale Grösse erreichen. Später bildet sich dann, wenn die Klappen-Insufficienz bestehen bleibt, allmählig Dilatation und zur Compensation derselben Hypertrophie des Herzens aus.

Als Zeichen der bei Chorea acut auftretenden Endocarditis sind also anzusehen:

Ein plötzlich aufspringendes heftiges Fieber, Zunahme der Grösse der Herzdämpfung in Folge von Dilatatio cordis, lebhafte Herzaction, sausendes Geräusch, welches den ersten Herzton begleitet, Verstärkung des zweiten Pulmonalarterientons. Wenn diese Symptome nicht sämmtlich vorhanden sind, wird man eine Ausbildung von Endocarditis nicht annehmen dürfen. Ist man nicht in der Lage gewesen, den Beginn derselben beobachten zu können, so kann das Fieber vollkommen bereits geschwunden sein, doch müssen die übrigen Symptome vorhanden sein, um die Diagnose auf Endocarditis stellen zu dürfen.

Unter den übrigen Erscheinungen der Endocarditis steht die acute Dilatatio cordis in erster Linie. Ohne diese kann von einer acuten Endocarditis keine Rede sein. Doch muss man im Auge behalten, dass es Fälle von plötzlich auftretender Dilatatio cordis giebt, welche nicht in Folge von Entzündung des Endocardium entstanden sind. Hierher gehören Krankheitsprocesse, welche schnell und in grosser Ausdehnung die Blutcirculation in den Lungen beschränken, wie ausgebreitete croupose Pneumonie, diffuse Bronchitis und Lungenödem. In solchen Fällen betrifft die Dilatation überwiegend das rechte Herz, doch wird ziemlich regelmässig auch das linke in Mitleidenschaft gezogen. Systolische Sausegeräusche werden dabei nicht beobachtet, doch findet sich der zweite Pulmonalarterienton stärker accentuirt.

Einen sehr interessanten Fall von acuter Dilatatio cordis ohne ein den ersten Herzton begleitendes sausendes Geräusch und ohne Verstärkung des zweiten Pulmonalarterientons habe ich bei septischer Infection beobachtet.

Ein Knabe von 11 Jahren, welcher wegen Caries im rechten Kniegelenk in das Kinderspital gebracht worden war, wurde am 27sten October 1880 unter allen Vorsichtsmaassregeln amputirt. Am folgenden Tage lebhaftes Fieber, Spuren von Carbolintoxication im Urin. Am 30sten October Osteomyelitis im Stumpf, Schüttelfrost, Collapsus. Am nächsten Tage war Dilatatio cordis deutlich zu constatiren. Die Herzspitze überschritt die linke Mamillarlinie um mehr als 1 Cent., während sie bei der Aufnahme etwas nach innen von derselben gestanden hatte. Der Puls war unregelmässig, sehr frequent, klein und weich. Am Abend Nachlass des Fiebers. Am 3ten November fieberfrei, die Dilatation bedeutend geringer, der Spitzenstoss in der linken Mamillarlinie, die Arterien gespannter, die Pulsfrequenz geringer. Kein Eiweiss im Urin. Am 6sten November trat ein neuer Schüttelfrost mit den gleichen Erscheinungen wie der erste, auf. Das Fieber war hoch, es entwickelte sich wiederum Dilatatio cordis, welche mit dem Schwinden des Fiebers ebenfalls rückgängig wurde. Als sich am 10ten November derselbe Vorgang wiederholte, betrug am folgenden Tage die Breite des Herzens $7\frac{1}{2}$, die Länge $11\frac{1}{2}$ gegen $6\frac{1}{4}$ und $10\frac{1}{4}$ bei der Aufnahme, die Herzspitze schlug über einen Centim. nach aussen von der linken Mamillarlinie an. Am 13ten wurde ein Theil des Knochenstumpfs resecirt. Am folgenden Tage war die Dilatatio cordis geringer. Am 23sten November wurde constatirt, dass die Grösse des Herzens vollständig wieder die normalen Grenzen erreicht hatte. Die Genesung machte dauernde Fortschritte. Die Grösse und Lage des Herzens zeigte dauernd normale Verhältnisse. Die beiden ersten Schüttelfröste wurden mit Hydrochinon, der dritte mit Natr. salicyl. bekämpft.

Die kurze Dauer des Bestandes der Herzerscheinungen, das schnelle und vollständige Schwinden und das wiederholte Auftreten desselben Vorganges nach kurzem Intervall beweist, dass es sich hier nicht um eine Endocarditis,

sondern um eine Erschlaffung der Herzwandungen in Folge der septischen Infection gehandelt hat. Beweisend dafür ist auch die jedes Mal mit dem Anfall auftretende Kleinheit und Weichheit des Pulses.

Es wird nicht schwer halten, eine septicämische Dilatatio cordis von einer durch Endocarditis bewirkten zu unterscheiden.

Der Unterschied zwischen der letzteren und den durch Behinderungen im Lungenkreislauf entstandenen Dilatationen des Herzens liegt zunächst darin, dass die letzteren zunächst die rechte Herzhälfte ergreifen und an und für sich nicht mit Fieber verknüpft sind, das Auftreten der Dilatation also auch nicht durch die Steigerung eines etwa vorhandenen Fiebers markirt wird. Ferner sichern die nachweisbaren Ursachen der Dilatation die Diagnose und schliesslich werden die Fälle, in welchen sich in Folge von Erkrankung der Lunge eine Dilatatio cordis deutlich constatiren lässt, in der Regel lethal ablaufen.

Wenn keine Dilatatio cordis bei Chorea vorhanden ist, so kann es sich um keine florirende Endocarditis handeln, wenn auch der erste Ton von einem Geräusch begleitet und der zweite Pulmonalarterienton verstärkt ist. Man hat dann eine im Ablauf begriffene Endocarditis vor sich, in welcher die Dilatation geschwunden und das Herz zur normalen Grösse zurückgekehrt ist. Dann wird auch das systolische Geräusch und die Verstärkung des zweiten Pulmonalarterientones allmählig schwinden oder, wenn der Process Klappen ergriffen und zum Schrumpfen gebracht hat, meistens bestehen bleiben und nach und nach secundaere Dilatation und Hypertrophie des Herzens zur Entwickelung kommen.

Nun giebt es aber Fälle von Chorea, in welchen unzweifelhaft keine Endocarditis vorhanden ist. Es fehlt die Dilatation, das Fieber, dagegen ist der erste Herzton, entweder hauptsächlich an der Herzspitze oder auch verbreitet über der ganzen Herzdämpfung von einem sausenden Geräusch begleitet, oft auch der zweite Pulmonalarterienton verstärkt. Charakteristisch ist für diese Fälle, dass diese Erscheinungen nicht dauernd sind, sondern, oft nach ganz kurzem Bestehen schwinden und wieder auftreten. Hier handelt es sich nicht um einen Process, welcher das Endocardium und speciell die Klappen betroffen hat, sondern um eine Störung in der Herzthätigkeit. Man braucht nur wenige Minuten lang das Herz eines an Chorea leidenden Kindes zu auscultiren und zu palpiren, um sich zu überzeugen, wie wechselnd die Herzthätigkeit und wie unregelmässig der Rhythmus der Contractionen ist. Der Grund hiervon liegt theils in den, durch die Chorea mit afficirten Nerven des Herzens, theils in der wechselnden Störung der Blutcirculation. Die Ursache dieser letzteren besteht theils in der krampfhaften regellosen Respiration, theils in den krampfhaften Bewegungen des Körpers überhaupt, so dass also diese Erscheinungen von Seiten des Herzens um so eher zur Beobachtung kommen, je hochgradiger die Chorea entwickelt ist, je beträchtlicher die dadurch bewirkten Störungen der Circulation des Blutes und die davon abhängigen erhöhten Ansprüche sind, welche an die Arbeit des Herzens gestellt werden.

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## *On the Relationship of Chorea to Rheumatism, with Special Reference to the Heart Murmur which so frequently attends Chorea.*

Dr. Stephen Mackenzie, London.

As a means of ascertaining the relationship of chorea to rheumatism, I have carefully examined the records of the cases of chorea admitted as in-patients

into the London Hospital during the last six years (1875 to 1880 inclusive). During this period the records have been kept with precision by successive medical registrars, who have also exercised supervision over the ward-work. The cases possess for the purpose of this examination the advantage that, having been under the care and observation of many different physicians, any personal bias on the point investigated is practically neutralised. At the same time, the conclusions based on such observations can only be regarded as approximately correct. The fifteen minutes to which the reading of this paper is limited obliges me to confine myself strictly to the question on which I was asked to write—namely, the relationship of chorea to rheumatism, with special reference to the heart murmur. I am greatly indebted to Dr. Mills, of Montreal, for valuable assistance in preparing and arranging the material on which this paper is based.

During the six years there were 172 cases of chorea with sufficiently complete records for the purpose of the inquiry.

The sex and ages of the patients is shown in the following table:—

| Sex. | No. of Cases. | Under 5. | 5 to 10. | 10 to 15. | 15 to 20. | 20 to 25. | 25 to 30. | Over 30. |
|---|---|---|---|---|---|---|---|---|
| Males . . | 51 | ... | 11 | 28 | 10 | 2 | ... | ... |
| Females . . | 121 | 1 | 22 | 53 | 31 | 10 | 3 | 1 |
| Total . | 172 | 1 | 33 | 81 | 41 | 12 | 3 | 1 |

It will be seen that most of the cases occurred between the ages of five and twenty. As a matter of fact, there were only thirteen cases eight years of age or younger, and of these eight were eight years old.

Particulars as to the heart were obtained in 164 cases:—

In 89 or 54·26 per cent. there was a distinct murmur.
„ 21 „ 12·8 „ the heart sounds were modified, but there was no distinct murmur.
„ 48 „ 29·27 „ there was no abnormality.
„ 4 „ 2·43 „ the signs were equivocal.

The nature of murmurs was as follows:—

| | |
|---|---|
| A systolic murmur at apex in . . . . | 73 |
| A presystolic murmur at apex in . . . . | 5 |
| A presystolic and systolic murmur at apex in . . | 7 |
| A systolic murmur at base in . . . . | 1 |
| A systolic murmur at base and apex . . . | 3 |
| | 89 |

It will be seen that in the cases in which a murmur was heard, it was at the apex in all except one case; in three cases there was a murmur at the base in addition to the apical bruit. Excluding these last cases, the relative frequency of apical to basic murmurs was as 85 to 1. Under the term "modified heart sounds" are included cases in which the heart sounds were "murmurish" or prolonged, but which the observer hesitated to describe as with a distinct bruit, cases in which the heart sounds were reduplicated, and cases in which the heart's action was irregular or tumultuous. These cases form a debatable ground as to whether the modification of the heart's sounds are indicative of valvular defect or organic lesion of any kind. Dr. Ernest Sansom has brought forward evidence that reduplication of the first sound may be due to mitral stenosis.*

* "On the Causes and Significance of Reduplication of the Sounds of the Heart," *Medical Times and Gazette*, January 9 and 16, 1881.

If to the cases in which there was an undoubted murmur be added the cases in which the heart sounds were modified in some way, we should have 89 + 21 = 110 cases, or 67·07 per cent. in which some cardiac abnormality existed. Keeping, however, to the cases in which a distinct murmur was present, we have 89 or 54·26 per cent. with bruits.* It must be remembered this is a calculation for all ages.

Having shown the frequency and clinical features of the cardiac abnormalities observed in chorea, I shall next proceed to examine into the essential nature of the lesion on which the alterations in the heart sounds depends. For that purpose there is evidence of two kinds:—(1.) The evidence afforded by examination of the heart after death. (2.) The evidence afforded by clinical examination of the heart at a period subsequent to the attack of chorea.

I.—*Condition of the Heart in Fatal Cases.*—Out of the whole number of cases analysed, seven proved fatal, and in six of these a post-mortem examination was made.

I give the briefest possible extracts of the cases:—

1.—Male, æt. 17. First attack. Had had rheumatism. Systolic murmur at apex. *Necropsy.* Vegetations on mitral valve.

2.—Female, æt. 19. Second attack. No history of rheumatism; no murmur. Pregnant. *Necropsy.* Vegetations on auricular surfaces of mitral valve.

3.—Female, æt. 10. Second attack. No history of rheumatism. Systolic and diastolic murmurs. Great anæmia. *Necropsy.* Incompetent aortic and mitral valves, with vegetations on each. Contracted mitral orifice. Hypertrophy and dilatation of left auricle and ventricle.

4.—Female, æt. 19. History as to rheumatism and antecedents not ascertainable. Systolic murmur at apex. Three months pregnant. In consequence of severity of symptoms, abortion induced. *Necropsy.* Pyæmia, heart fatty.

5.—Female, æt. 27. Second attack. Stated never to have had rheumatism. Six months pregnant. No murmur. No anæmia. *Necropsy.* Numerous vegetations on mitral valve.

6.—Male, æt. 13. Had had subacute rheumatism; subcutaneous fibrous nodules. Mitral systolic and presystolic murmurs; pericarditis. The patient died from exacerbation of pericarditis, the chorea having subsided. *Necropsy.* Old endocarditis, and recent vegetations on mitral and tricuspid valves. Acute pericarditis.

7.—Female, æt. 13. Fourth attack. Had had rheumatic fever. Presystolic and systolic murmurs at apex. Pneumonia. *No necropsy.*

Thus in all except one in which a post-mortem examination was made, vegetations were found on the mitral valve. In the case in which no vegetations were present, though pyæmia was the actual cause of death, the patient died *with* chorea. The heart, it will be observed, was fatty, and probably during life allowed of regurgitation, for a systolic mitral murmur was present. In the case in which no post-mortem examination was made, the patient had, whilst living, unmistakable physical signs of mitral stenosis.

It will thus be seen that heart disease of an organic kind existed in all the cases dying of or with chorea. Further, that there was endocarditis affecting the mitral valve in all except one case, where the heart was fatty. In two of the fatal cases with endocarditis no bruit was heard during life. From

* It is instructive, as showing that this is probably a very nearly correct estimate of the frequency of heart murmur in chorea in hospital patients at all ages, and not a biassed calculation of the author, to compare these figures with those of successive registrars during the same period. In 1876 Dr. Herman gives 57·14 per cent.; in 1877 Dr. Warner gives 47·7 per cent.; and in 1880 Dr. Gabbett gives 56·09 per cent. as the frequency of bruits. The mean of these is 53·79.

this it may be inferred that absence of murmur is no proof that the heart is unaffected with organic disease. This raises an important point. In chorea occasionally a murmur which is not present at first develops in the course of the disease. In such cases it is from the above possible that the disease of the valve, which ultimately causes the murmur, may have been present from the first; and also, that had the fatal cases in which no murmur was heard lived longer, a bruit might have become developed. In the case of the fatty heart without endocarditis, the patient was a pregnant female, and it might be assumed from this that vegetations are not found in the chorea of pregnant women. This conclusion is by no means warranted by the present series of facts, however, for in the two other fatal cases of chorea occurring in the pregnant, vegetations were found on the mitral valve.

II.—The evidence afforded by physical examination of the heart at some period subsequent to the attack of chorea.

This is a line of inquiry which, as far as I know, has been but little investigated. There are great practical difficulties in following up hospital cases after their discharge, but the trouble is repaid by the results. Those engaged in family practice might collect some really valuable information on this point. With the assistance of Mr. George Greenwood and Mr. Grün,* students of the London Hospital, I have been able to follow up thirty-three cases of chorea at periods varying from one to five years subsequent to their leaving the Hospital. I give in a table, as an Appendix, the results of my examinations of these thirty-three patients, but these results may be briefly summarised.

Condition of the heart in cases of chorea, examined at periods from one to five years subsequent to the attack for which the patients were in the Hospital:—

| | |
|---|---|
| Cases with a distinct murmur | 17 |
| Cases with some cardiac abnormality otherwise than a murmur | 7 |
| Died of heart disease | 3 |
| Died of unknown causes | 3 |
| Heart normal | 3 |
| | 33 |

It will be seen that there was a distinct murmur (as well as other signs of heart disease) in 20 (17 + 3) cases, or in 60·6 per cent. If to these cases with unquestionable heart disease be added the cases in which the heart sounds were distinctly altered, but in which there was no well-marked murmur, it gives 20 + 7 = 27 cases, or 81·81 per cent., in which some cardiac derangement persisted after the attack of chorea observed. If, as is possible, two, or all three, of the other fatal cases died of or with heart disease, the percentage of permanent cardiac abnormality would be still further increased.† Objection may reasonably be made to considering cardiac abnormalities other than distinct murmurs as evidence of organic heart disease. I would therefore be content with the statement that indisputable heart disease persisted in 60·6 per cent. of the series of chorea cases examined. But I think that hearts that show such abnormalities as the following at periods of from one to five years after chorea may fairly be regarded as at least suspicious of some organic lesion. These abnormalities were —1. Occasional reduplication of first sound, which is rather prolonged at apex. 2. Reduplication of first sound at apex. 3. Murmurish first sound at apex, especially on exertion; accentuated second sound. 4. First sound a little pro-

* Mr. Greenwood and Mr. Grün visited the patients at their homes, and sent them to see me at the Hospital, where I examined and noted the condition of the heart.

† The condition of the heart whilst in Hospital in the three fatal cases of which the cause of death was not ascertained was: 1. Systolic and presystolic murmurs. 2. Systolic murmur at apex; dilated heart. 3. Mitral systolic murmur.

longed, especially after exertion. 5. Excited action of heart; second pulmonary sound accentuated. 6. Intensified first sound; heaving beat. 7. Slight accentuation of pulmonary second sound.

The number of cases examined is small, however, and requires corroboration.

With reference to the PRE-EXISTENCE OF RHEUMATISM, the cases are divided into four classes :—

CLASS I.—Cases in which it was distinctly stated that the patient had suffered from rheumatic fever, or in which the patient had been laid up in bed with swelling of, and pain in, the joints.

CLASS II.—Cases in which the patient had suffered from "rheumatic pains," "pains in the joints," "growing pains," &c., but had not been laid up in bed with the attack; probably subacute rheumatism.

CLASS III.—Cases in which the history of rheumatism was doubtful, *i.e.*, the patient had had some illness, the nature of which could not be ascertained owing to the parents' or friends' ignorance, or to their want of opportunity of obtaining information on this point. It will be conceded that, in hospital practice in this metropolis, this class will embrace a good many cases in which the possibility of rheumatism having occurred will be considerable.

CLASS IV.—Cases in which no history of rheumatism could be obtained, even on inquiry on this point. The remark made on the last class applies almost equally to this. It cannot be safely assumed that rheumatism has not existed in cases where the parents and friends (of this class of society) are unaware of its occurrence.

Dividing, now, the cases into these four classes, we have :—

| | | | |
|---|---|---|---|
| I. With a distinct history of rheumatism | 47 | or 27·32 | per cent.* |
| II. With a history of pains of various kinds | 30 | ,, 17·44 | ,, |
| III. With a doubtful history of rheumatism | 25 | ,, 14·53 | ,, |
| IV. With no history of rheumatism | 70 | ,, 40·69 | ,, |
| | 172 | ,, 99·98 | ,, |

If we separate the first attacks of chorea from the remainder, we have the following figures :—

| | | | |
|---|---|---|---|
| CLASS I. . . . . | 22 | cases or 21·76 | per cent. |
| ,, II. . . . . | 23 | ,, 22·54 | ,, |
| ,, III. . . . . | 14 | ,, 13·72 | ,, |
| ,, IV. . . . . | 43 | ,, 42·15 | ,, |
| | 102 | ,, 100·17 | ,, |

in which there was a history of rheumatism. It must be borne in mind that deduction should be made for what may be termed the age incidence of rheumatism, as well stated by Dr. Octavius Sturges.† This observer has, in dealing with a considerable number of cases, separated those below from those above eight years of age. The number of cases in this collection below eight years of age are so few (thirteen) that conclusions based on so small a number might mislead. A certain deduction should, however, doubtless be made for the natural incidence of rheumatism—one or two per cent.

Dealing with the whole number, without reference to the number of the attack, it has been shown that there was unmistakable evidence of rheumatism in 27·32 per cent. To these, I think, without straining the figures, the cases in

* The calculations of successive registrars agree very closely with my figures as to the frequency of a distinct history of rheumatism. Thus Dr. Herman, in 1876, gives 28·57; Dr. Warner, in 1877, gives 27·27; Dr. Gabbett, in 1880, gives 29·26 per cent. The mean of these numbers is 28·36 per cent. of cases of chorea with a distinct history of rheumatism.

† *Lancet*, 1879, vol. ii. p. 791; *Brain*, July 1881, p. 169.

Class II.—the patients who had suffered from pains of various kinds, probably subacute rheumatism—ought to be added. We should then have 47 + 30 = 77 cases, or 44·76 per cent. (nearly one-half) in which rheumatism was directly associated with the chorea. And treating the cases of first attacks of chorea in a similar manner, 22 + 23 = 45 or 44 per cent. (nearly) in which rheumatism *preceded* the chorea.

But I believe that the 44 per cent. in which rheumatism had *almost* certainly (27·32 certainly) existed, by no means represents the probable prevalence of rheumatism as an antecedent to the first or later attacks of chorea, and that in a very considerable (though not ascertainable) proportion of cases rheumatism must have occurred, though no history of its occurrence was obtained. I base my belief on this point mainly on the following grounds :—

1.—The difficulty of obtaining from the friends or parents of chorea patients of the class admitted into hospitals the nature of previous illnesses unless of a very definite character.

2.—The ill-marked characters which rheumatism in children, especially when subacute, often presents.

3.—The frequency with which heart murmurs were found in cases of first attacks in which rheumatism could not be ascertained to have pre-existed.

The last reason, I admit, seems dangerously like begging the question under discussion ; but seeing the proved connection between rheumatism and chorea, and the marked influence of the former in producing valvular disease of the heart apart from chorea, it appears to me a legitimate reason, until it is shown conclusively that chorea independently of rheumatism causes similar cardiac disease. Thus it is instructive to compare the condition of the heart in the four classes into which I have divided the cases with reference to rheumatism :—

Class I.—47 cases with a distinct history of rheumatism.

| | |
|---|---|
| With systolic murmur at apex | 26 |
| „ systolic at base and apex | 1 |
| „ presystolic and systolic at apex | 7 |
| „ murmur supposed to be hæmic (excluded) | 2 |
| „ heart sounds modified, no distinct murmur | 3 |
| „ no murmur | 8 |
| | 47 |

With a distinct murmur of some kind 34 or 72·34 per cent.

Class II.—28 cases with a history of pains (probably rheumatic).

| | |
|---|---|
| With systolic murmur at apex | 11 |
| „ presystolic at apex | 1 |
| „ murmur supposed to be hæmic (excluded) | 1 |
| „ heart sounds modified (no bruit) | 8 |
| „ no murmur | 7 |
| | 28 |

With a distinct murmur of some kind 12 or 42·85 per cent.

Class III.—23 cases with doubtful history of rheumatism.

| | |
|---|---|
| With systolic murmur at apex | 10 |
| „ presystolic at apex | 2 |
| „ murmur supposed to be hæmic (excluded) | 1 |
| „ heart sounds modified | 1 |
| „ no murmur | 9 |
| | 23 |

With a distinct murmur 12 or 52·17 per cent.

Class IV.—66 cases with no history of rheumatism.

| | |
|---|---|
| With systolic murmur at apex . . . . | 26 |
| „ systolic murmur at base and apex . . . | 2 |
| „ systolic murmur at base . . . . . | 1 |
| „ presystolic murmur at apex . . . . | 2 |
| „ murmur supposed to be hæmic (excluded) . | 2 |
| „ heart sounds modified . . . . . | 9 |
| „ no murmur . . . . . | 24 |
| | 66 |

With a distinct murmur of some kind 31 or 46·96 per cent.

Comparing these classes we find :—

| | | | | |
|---|---|---|---|---|
| Class I. | 47 cases with murmur | in | 34 or 72·34 | per cent. |
| „ II. | 28 „ | „ | 12 „ 42·85 | „ |
| „ III. | 23 „ | „ | 12 „ 52·17 | „ |
| „ IV. | 66 „ | „ | 31 „ 46·96 | „ |

It will thus be seen that whilst the frequency of heart disease was greatest amongst the most distinctly rheumatic, it was very high in the cases in which rheumatism was probable, in which it was doubtful, and in which no history of rheumatism was obtainable.

Again, separating the first attacks of chorea from the rest of the cases, we have :—

| | | | | |
|---|---|---|---|---|
| Class I. | 22 cases with murmur | in | 16 or 72·72 | per cent. |
| „ II. | 23 „ | „ | 11 „ 47·82 | „ |
| „ III. | 14 „ | „ | 6 „ 42·85 | „ |
| „ IV. | 43 „ | „ | 20 „ 46·51 | „ |

It is thus shown that in primary attacks of chorea a murmur existed in something approaching 50 per cent. in cases in which a history of rheumatism was doubtful or denied. In *five* only of the cases, all first attacks, did the murmur develop *during* the attack. In none of these five cases was there a history of rheumatism. In all the remaining cases a murmur was present when the patient came under observation, presumably early in the attack ; and as a murmur only developed under observation in such few instances, it was probably in the remainder caused by some antecedent condition, or some influence acting at the commencement of the attack. Chorea, *per se*, has not been shown in these cases to produce a murmur at all frequently. As we know of no influence likely to produce heart disease with anything like the frequency with which it occurred in Classes III. and IV., I think the conclusion is almost irresistible that rheumatism must have obtained in a very considerable proportion of cases in which no history of its occurrence could be elicited. I have inquired into the histories of the cases for other occasional causes of endocarditis, such as scarlet fever, measles, smallpox, anæmia, &c., but taken collectively, they do not equal the frequency of rheumatism as an antecedent, and they are nearly equally distributed amongst the rheumatic and the non-rheumatic. This latter statement is of importance, as it may be presumed that on failure to elicit a history of rheumatism, additional care would be taken to trace other antecedents.

In some of the cases "subcutaneous nodules," as described by Dr. Barlow and Dr. Warner, were present.

The condition of the heart in old and recent cases is the same as that seen in rheumatism old and recent. It is, of course, admitted that endocarditis from various causes assumes the same form.

Finally, I believe that from the analysis of these cases the following conclusions may be drawn :—

1.—That a murmur was present in more than half the cases of chorea.

2.—That the murmur, in all except one case, was due to endocarditis affecting almost exclusively the mitral valve.

3.—That in 60 per cent. for certain (and in over 80 per cent. possibly) the heart lesion persisted.

4.—That absence of murmur is no proof of absence of organic heart disease.

5.—That rheumatism had pre-existed in nearly half the cases, and that there are strong grounds for believing that it had been an antecedent in a very much larger proportion of cases.

6.—That no other very frequent exciting cause of endocarditis is shown to have pre-existed, or to have been more frequent amongst the non-rheumatic than the rheumatic.

7.—That the form of heart disease is that seen in connection with rheumatism.

8.—That rheumatism is in nearly all cases the cause of the heart murmur which so frequently attends chorea.

## APPENDIX.

CASES OF CHOREA examined with reference to the condition of the heart at periods of from one to five years subsequent to the attack for which they were in the London Hospital.

| No. | Name. | Age. | Condition of Heart on Leaving. | Present Condition of Heart. |
|---|---|---|---|---|
| | LEFT HOSPITAL IN 1876—(*Five Years*). | | | |
| 1 | M. L. . . | 29 | Systolic at apex. | Occasional reduplication of first sound, which is rather prolonged at apex; soft blow over pulmonary artery. |
| 2 | A. L. . . | 12 | Mitral diastolic. | No bruit; reduplication of first sound at apex. |
| 3 | A. W. . . | 15 | Mitral systolic. | No bruit; apex beat in 5th space, diffused, moderately strong. |
| 4 | F. A. . . | 16 | Mitral systolic. | First sound murmurish, occasionally developing into bruit; apex in nipple line. |
| 5 | G. F. . . | 9 | Systolic at apex. | Sounds normal, beat normal. |
| 6 | K. A. . . | 8 | Mitral systolic. | Systolic at apex, second sound intensified at base; beat in 4th and 5th spaces nipple line. |
| | LEFT HOSPITAL IN 1878—(*Three Years*). | | | |
| 7 | S. N. . . | 14 | Systolic at apex. | Soft systolic at apex, intensified on exertion; apex beat in 4th space and nipple line, slightly heaving. |
| 8 | A. W. . . | 10 | Mitral systolic. | Murmurish first sound at apex, especially on exertion; accentuated second sound. |
| 9 | C. W. . . | 16 | Systolic at apex. | Died of heart disease in 1881. Had subcutaneous nodules. |
| 10 | H. B. . . | 9 | Mitral systolic, which disappeared. | Died of heart disease in 1881. |
| 11 | M. A. W. . | 10 | Systolic and (?) presystolic at apex. | Loud systolic at apex; maximum intensity in 3d space; second sound intensified and occasionally reduplicated. |

CASES OF CHOREA—*continued.*

| No. | Name. | Age. | Condition of Heart on Leaving. | Present Condition of Heart. |
|---|---|---|---|---|
| | LEFT HOSPITAL IN 1878—(*Three Years*)—*continued.* | | | |
| 12 | P. K. . . | 10 | Normal. | First a little prolonged, especially after exertion ; no murmur. |
| 13 | E. A. . . | ... | Systolic and presystolic. | Dead. |
| 14 | C. T. . . | ... | Systolic at apex. | Systolic at apex. |
| 15 | W. B. . . | ... | Systolic at apex ; transient suspicion of presystolic. | Systolic at apex ; reduplicated second sound ; apex heaving. |
| 16 | G. K. . . | 12 | Systolic at apex ; dilated heart. | Died of "blood tumour of brain," 1880. |
| 17 | A. L. . . | 14 | Soft basic systolic. | Excited action of heart, second pulmonary sound accentuated ; no bruit. |
| 18 | A. P. . . | 9 | Presystolic at apex. | Loud blowing systolic at apex, heaving beat. |
| | LEFT HOSPITAL IN 1880—(*One Year*). | | | |
| 19 | H. M. . . | ... | Mitral systolic. | Dead. |
| 20 | C. H. . . | ... | Mitral systolic. | Mitral systolic, second sound intensified. |
| 21 | W. W. . . | ... | Systolic at apex. | Systolic at apex. |
| 22 | A. P. . . | 11 | Systolic at apex. | Systolic at apex, heaving beat. |
| 23 | E. S. . . | 16 | Systolic at apex. | Intensified first sound, heaving beat. |
| 24 | L. B. . . | 16 | Systolic at apex. | Normal, except for slight accentuation of second sound. |
| 25 | A. S. . . | 13 | Systolic, (?) presystolic. | Loud systolic at apex, heaving beat. |
| 26 | M. O. . . | 21 | Systolic at apex. | Blowing systolic at apex. |
| 27 | A. L. . . | 16 | Systolic at apex. | Loud blowing systolic at apex, heaving beat. |
| 28 | F. G. . . | 16 | Systolic and presystolic. | Systolic at apex ; palpitation. |
| 29 | M. A. J. . | 13 | Systolic at apex, disappeared. | No bruit ; very anæmic. |
| 30 | M. M. . . | 15 | Systolic at apex. | Blowing systolic ; short presystolic. |
| 31 | A. N. . . | 9 | Systolic at apex. | Soft systolic at apex ; beat outside nipple. |
| 32 | A. H. . . | 11 | Systolic at apex. | Died of heart disease. |
| 33 | C. H. . . | 16 | Systolic at apex. | Systolic at apex ; second sound slapping. |

## *On the Modes of Origin of Chorea in Relation to the Question of its Pathology and Treatment.*

Dr. STURGES, London.

During the last five years I have had charge of 177 children and young adults suffering from chorea, and have taken note especially of the following points :—(1.) *The morbid associations of the disorder.* (2.) *Its mode of origin and of progress in the several groups of muscles which it affects.* (3.) *Its cause* (near and remote), and *its final issue* (whether in recovery or otherwise). The precise conclusions under these several headings must be stated numerically,* but as figures and percentages do not convey a very vivid impression, I shall here speak of the results in general.

These 177 examples of chorea are made up of 132 received from Great Ormond Street into the hospital at Highgate devoted to chronic cases, and

* The cases are detailed *seriatim* in an appendix of my work on "Chorea and Other Movement Disorders of Early Life," just published.

forty-five admitted into the Westminster Hospital. The former series consists of children at or under the age of eleven; the latter series includes as many as ten who are over thirteen. In the first series of 132, the girls are to the boys in proportion of three to one. Of the whole 177, something less than a third (fifty-two) have had previous attacks, but only ten of the whole number have had two previous attacks, and only four more than two.

I.—As regards my first point, the *morbid associations of chorea*, it must be remembered that the *brief lives* of these little children afford but small opportunity for judging of nervous temperament or proclivity for particular diseases. But amongst infantile affections, there is one which has at least this alliance with chorea, that it is (in parts at least) a nervous disorder, and that it shows a marked preference for girls—I mean whooping-cough. It is worthy of remark, therefore, that in 120 cases of the Children's Hospital series, where this point was inquired into, only twenty, or one in six, had escaped whooping-cough (viz., sixteen of the ninety-two girls, and four of the twenty-eight boys).

In order to ascertain how this proportion of whooping-cough amongst choreic children might compare with its proportion amongst children in general, I take from the records of Cromwell House, during my connection with it, three independent series, each consisting of 120 patients taken consecutively. The proportion of children who escaped whooping-cough in these three series respectively varies but little. It is very nearly expressed by saying that two in every five have so escaped.* *It would thus appear that whooping-cough has more than double the frequency in choreic children that it has in others, the proportion being as twelve to five.*

II.—The *mode of origin* of chorea may be considered from many different points of view. It has been here observed in reference, first, to those *parts of the body which the disorder selects by preference*, whether as its sole seat, or as the starting-place whence it spreads to other limbs; and, secondly, with reference to the *cause or causes on which the disorder seemed immediately to depend.* The first inquiry—viz., that which concerns the starting-place of chorea—while it is the more manageable and reliable of the two, is also the one which has received least attention.

*The mode of onset of chorea*, in the sense just mentioned, was made the subject of particular inquiry in 132 of the 177 cases here discussed. In (forty-one) *nearly a third* of these, the muscular disorder was represented as being *general* from the first. Yet with the probability of partial chorea for a while escaping notice, it would be hardly safe to conclude that in so large a proportion of instances it is from the first a general invasion. Greater weight attaches to the remaining examples, where the first onset of the disorder was distinctly observed in a particular place, and the results were, I think, remarkable. In seventeen cases chorea is represented as *right-sided*, the meaning being that the arm and leg of that side were first or more especially affected. In nineteen, or what is practically the same number, it is represented as *left-sided.* Right and left sided chorea, therefore, thus generally described, may be taken as equal. But here equality ends; and I now quote figures as showing the disparity better than proportions. *Chorea, in its commencement, affected the upper parts of the body alone in forty-five cases; it affected the lower part of the body alone in five (or less than a tenth of the former).*

To particularise: Chorea *began in the hands* (one or both) in as many as thirty-seven cases, or more than a third of the entire number in which its starting-place was noticed. In about half of these (eighteen) it began *in both hands;* but when this was not so, it was *right-handed* in fourteen, and *left-handed* in only five. On the contrary, we have the *right* arm, the first to

* *i.e.*, 1 in 2·9 in first series, 1 in 2·9 in second series, 1 in 2·15 in third series.

be unsteady with less than half the frequency (four) of the left (ten). The affection began in *both legs* in but two cases; in the left leg in two; in the right leg in one. It began in the face, according to the observation (which is probably faulty in regard to so mobile a part), nine times.

Thus *the order in which the several muscular groups yield to chorea is the following:*—The hands most, the right hand, the left arm, the face next (with a sudden fall in frequency, but little difference between themselves), the left hand, the arms, the right arm, the legs, the left leg, the right leg, these last appearing so seldom that it is obviously impossible to rate and compare one with the other.

On the whole, and making abundant allowance for error, the muscles which chorea affects, both first and most, are the same that are concerned in those higher uses which are not only the most intricate, but which, apart from civilisation, are never reached at all; while the parts which the disorder affects later and more rarely, concern movements which are so far *natural* and removed from any special process of education that savages perform them as well, and, indeed, far better than civilised men.

And there are other points to be kept in view in reference to the relative stability of different parts of the body, which need no support from statistics, since they are matters of common knowledge. Notice, for example, *the several distinct modes of development of chorea;* the fact that choreic disturbance (often, it is true, not so accounted, because sensitive facial movement is easily excused) will appear in the face and spread no further, whilst, on the contrary, the infrequent chorea which begins in the legs will almost always spread to the upper limbs. And the mode of recession is as remarkable as the onset. A general chorea mainly recovered from elsewhere will sometimes linger in the hand,* and exhibit extreme severity in this one place; but the comparatively rare chorea of the feet and legs is both the last to come and the first to go.

One other matter may be mentioned apart from the direct sanction of the tables. It is that *in the most violent chorea the muscles still maintain their accustomed concert.* The muscular over-movement, except for its exaggeration and disregard of the will, is the same as the natural movement. Putting aside the paresis, which is sometimes conjoined with chorea, it is not true to say (as has been said), that it will select certain muscles out of a group and move them after a manner of which mere will is incapable.

That this conjoint action of the muscles remains intact is shown especially in this: that in those parts of the body where the several muscular uses are most distinctly differentiated, there, in exact proportion to the degree and perfection of such differentiations, do we find a more or less perfect separateness of choreic movement. Thus, for example, the hand and the fingers are concerned in many tasks in which the arm takes no direct share, and the hand and fingers are often choreic without the arm. On the other hand, the simple movements of the foot are commonly those which it shares with the leg. Foot movements alone are as rare as foot chorea alone. It is the same in regard to the two sides of the body. Movement of one arm is common enough without need or suggestion of movement in the other, but the movement of one leg almost implies movement of the other. Similarly, chorea of a single arm is very common, and chorea of a single leg is very rare. But most of all do we find this rule exhibited in the face. One-sided chorea of the face is the exception; one-sided paralysis is the rule.

To return to the tables. I have taken account of *rheumatism* in regard only to the 132 cases furnished by the Hospital for Children; the others may

* In this, as in some other features, chorea is not only an affection commonest in childhood; it is *peculiar* to that time of life.

be supposed to derive a bias from the preconceptions of the narrator. These 132, let it be remembered, came to me with their histories already taken, and I have only put down what was told me by a succession of observers, the several medical registrars, that is, acting in conjunction with the three physicians of the Hospital. The rheumatic account thus derived is as follows:—In the whole 132, six were doubtful, and in five the particular fact was not ascertained; fourteen or fifteen had had pains, probably rheumatic; seven had had rheumatic fever. Or, stating the facts in somewhat different form and less open to error, there are 97 cases out of the 132, or, excluding eleven where the point was not ascertained, there are 97 *out of* 121, *free from any rheumatic association.* In other words, *chorea has nothing to do with rheumatism in three-fourths of the cases.* And when we come to inquire what is the *nature* of the rheumatic association, the *degree* of its being what we see, it appears that in six out of the 132 (two of these six being cases of *acute* rheumatism) the chorea arises directly out of the rheumatism in such manner as to make the one seem the direct *cause* of the other.

I know that these figures are very much out of accordance with those of others, and that it is said by many that we are not sufficiently alive to the various forms of child's rheumatism, and especially to the transient character of the joint symptoms, and the frequent implication of the heart in the rheumatism of children. In reply to such contention I would only say this—First, that it must be a matter of extreme difficulty and doubt to accept, on the evidence of mothers and nurses, such indefinite signs as these of bygone rheumatism; and, secondly, that if a latent form of rheumatism is to be reckoned in this way, we are in fact maintaining an old hypothesis upon a new basis; for the old doctrine of the rheumatic origin of chorea asserted plainly an alliance between it and the ordinary power of articular rheumatism. However the truth may be, it is at least certain that *no one in this country has succeeded in finding any large proportion of cases immediately connected with rheumatism*, while as to those to whom a rheumatic history is assigned, it is obvious that *until we agree upon the particular signs and symptoms which are to be accepted as valid evidences of rheumatism, we have no common factors to deal with, and may expect the remarkable discrepancies in result which actually appear.*

My own belief, derived not from these tables alone, but from ampler grounds, to which I have referred elsewhere, is that *acute articular rheumatism*, although rare in the history of chorea, occurs *in such association with it as to justify the assumption of some direct relationship* existing in a very small proportion of examples; while, on the other hand, *chronic rheumatism* is so difficult of identification, and so easily confused with the joint and limb pain which properly belongs to chorea, that (although on this very account it is easy enough to impute it) *no such connection can be asserted on behalf of rheumatism generally.*

Very different must be the statement in regard to the *mental or psychical causes of chorea.* In the first series of 132 cases, the *immediate* cause was thought to be ascertained in 112, and in 97 (or 93) of these, say *in about half*, that cause was some mental shock or strain. In the second series of 45 cases there were as many as 28 (not to speak of those where the cause was unknown), *say two-thirds, where the cause was either fright or something allied to fright.* It is only by a perusal of the tables that the very real nature of this, the great, at all events the great *exciting*, cause of chorea can be adequately appreciated. "Pursued by a drunken man," "Sent for a policeman to quell a family disturbance," "Frightened by drunken father," "Locked up in a dark cellar"—is there anything in any suggested pathology of chorea which will

explain half so well as such an enumeration as this the undoubted fact that chorea is a disease of poor, ill-cared for children, and especially of timid girls?

I do not suppose that the immediate cause of chorea is *always* a mental one. Yet, supposing that were so, we should hardly expect, I think, to find it actually appearing in our records in much larger proportion than it does. There are other causes of mental distress, both for children and for men and women, besides those which openly appear—terrifying dreams, night terrors, sights in the street, incidents of school life, unequal competition with stronger and cleverer companions. Chorea is not more often to all appearances spontaneous than is the sudden and causeless change of disposition which will affect young children, and especially girls, at an age too early to be attributed to sexual development, disappearing in the same unaccountable way that it came. Considering, I say, that for half, or more than half, the instances of chorea that come before us we have a distinct cause in some great terror or such like, it would not be asking too much if we said of the rest that the many hidden, undiscovered causes of mental distress which lie in wait for little children, and especially *poor* little children, sufficiently account for them.

How are *the heart symptoms of chorea* to be reconciled with such a view of its causation as this? In 132 choreic children of the first series there was heart defect in one-third (45); in 45 children and young adults of the second series there was heart defect in one-third (16); "heart defect" being understood to mean either a mitral systolic murmur or an uneven action. But the observations hitherto made upon this point need, as I believe, to be supplemented by others having reference not merely to the cardiac sounds, but to the *rate* and *variability* of the cardiac action. Fully admitting the obscurity of this part of the subject, I would venture to repeat here the substance of some remarks I have sought elsewhere to justify.* The heart, I would submit, suffers in chorea (apart from the rheumatic association altogether), because chorea affects the same parts and organs which ordinary emotion affects; because, to use bolder language, it is an affection of the emotional centres. The commonest heart symptom in chorea, as in emotion, is increased frequency, but the most obvious and most observed symptom is systolic murmur, due to blood reflux through the auriculo-ventricular orifices, the consequence of fatigue *paresis* on the part of the papillary muscles.

I come to speak, in the last place, of *the final result*. Here are nearly 200 examples of chorea of varying severity, taken without selection from public practice during a period of five years. Excluding some few of the elder ones, and especially youths between thirteen and sixteen, in whom the disorder is very intractable, *they all recovered*. It cannot be said that they recovered altogether without drugs, because a large proportion of them, variously treated at an early stage of their disorder, were on their way to recovery before they came under my care; but it *may* be said of the cases which came to the chronic wards, and of the Westminster cases from first to last, that no medicinal agents had any share in their treatment.

*Chorea in children is a self-recovering affection*, and if it be urged that the question still remains as to the means by which it may be cured the quickest, I would venture to answer that we have yet to learn in what sense the term "cure" is applicable to an affection which no one professes to be able to alleviate immediately and out of hand, and which is of such uncertain duration that it is impossible to say what is a long and what a short term for it. Putting aside, indeed, extreme chorea, or about one case in a hundred, it seems of small importance whether the limbs of a young child return to such control as they are likely to endure this week or next. Any manifestation of anxiety for a

* "The Heart Symptoms of Chorea," *Brain*, July, 1881.

speedy recovery is likely to defeat its own object, in the injurious effect it produces in the case of children of sensitive temperament.

And while this much is to be said of the ordinary run of chorea—of the chorea which is too often over-noticed and over-treated—we must be careful of too readily altering our views when we come to consider the violent and sometimes fatal chorea of puberty. Such instances, as is well known, very soon reach their term. The extreme violence of the jactitation, the mental disturbance, the inability to take food or to get sleep—these are symptoms which so combine to reduce the strength, that they are of themselves too often the cause of death. Yet with all these symptoms, and even actual signs of sinking, the patient will sometimes revive, and quite suddenly return to health. It is true that some profound cerebral disturbances—such, for instance, as an embolic shower—will sometimes commence with choreic-like restlessness, soon to develop into clonic convulsions, and destined from the first to inevitable death—not from chorea, but from what at the first may easily pass for it. Yet we are to remember that the chorea which by its recovery sufficiently shows its alliance with the child's disorder may at the first exhibit symptoms as formidable as the other, and only in the event distinguishable from it.

In conclusion, the following propositions seem to be legitimate inferences from the statistics :—

1.—The muscles chiefly affected by chorea are the same which are devoted to the higher intellectual uses, and such as children (the favourite subjects of the disorder) have but imperfectly acquired the use of.

2.—Hence the parts selected by chorea are not those which depend upon a common motor centre, but those which habitually combine in purposeful and emotional movements.

3.—Psychical disturbance far outweighs all other immediate causes of chorea put together.

4.—The heart symptoms proper to chorea (independent, that is, of any rheumatic connection) are probably due to the fact that the heart is concerned along with the other muscles in a disorder which follows the pattern of emotion.

5.—The chorea of childhood, although it may recur several times in the same patient, is almost always eventually recovered from without the employment of drugs. The question, therefore, of the service of medicinal agents relates to the possibility of shortening attacks, and cannot be settled until it has been first determined what is a short and which a long term for an affection of very variable duration.

6.—The main facts of chorea—viz., those which concern its subjects, mode of origin, movement-phenomena, and security of spontaneous recovery—are all in favour of the hypothesis that it is best described as a functional disorder.

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## *The Relationship of Chorea to Rheumatism, with especial Reference to the Nature of the Heart Murmur which so frequently attends Chorea.*

Dr. John W. Byers, Belfast.

Although chorea has been a well-recognised affection since the days of Sydenham, and although in recent years, owing to the very great interest taken in the investigation of diseases of the nervous system, the attention of physicians has been directed to it with renewed vigour, still there is at the present time not only no unanimity, but the greatest diversity of opinion as to its nature.

In regard to chorea there are many points well worthy of study, but the two features of it which stand out prominently, and which the Committee of this Section have chosen for discussion to-day, are :—

1.—Its relationship to rheumatism.

2.—The nature of the heart murmur which so frequently attends it.

Having had an opportunity of observing a number of cases of chorea during the past two years at the Belfast Hospital for Sick Children, I have embodied their clinical features, especially as they relate to the question of rheumatism and the nature of the heart murmur, in the following table :—

## Analysis of Cases of Chorea from the Belfast Hospital for Sick Children.

| Name. | Age. | No. of Attacks. | Rheumatism. | Heart. | General Remarks. |
|---|---|---|---|---|---|
| Maggie M. . | 11 | 1 | Acute rheumatism two weeks previous to admission into hospital. Three days after the rheumatism commenced chorea set in. | Soft systolic apical murmur during the attack, which gradually ceased to be heard. | Case of right hemichorea commencing during the course of an attack of acute rheumatism. Slight anæsthesia and muscular weakness of right side. The choreic movements specially severe in face and arm. During recovery the murmur passed away. Child seen again four months after attack and no murmur heard. |
| Ellen M. . | 9 | 1 | Sister of No. 1; admitted on the day that her sister was discharged, suffering from acute rheumatism. | On admission systolic murmur at apex. | Acute rheumatism commenced four days before admission to hospital. On the seventh day after admission the chorea began. Both sides affected, but especially the right. Removed home by friends on the eleventh day (in hospital) and died three days afterwards. No. p.m. A cousin of this child's mother had an attack of severe general chorea after a fright. |
| Agnes C. . | 14 | 2 First attack followed a fright when she was nine years of age. No cause of second attack assigned. | Subacute rheumatism during decline of second attack. No history of rheumatism with first attack. | Mitral systolic murmur during second attack. Condition of heart during the first attack not known. | Was in hospital during second attack, which was general. As she was getting better, pains came on in the joints accompanied by extreme anæmia, œdema, and purpuric spots (Purpura rheumatica?). She was seen two years after this, when mitral murmur was still heard. |
| Margt. J. . | 12 | 2 Rheumatism and fright before the first attack. Cause of the second not known. | Rheumatism before the first attack. | Apical systolic murmur heard four years after the first attack. Condition of heart during the attack not known. | Child brought to out-patients' room complaining of pains in joints (subacute rheumatism). Mitral systolic murmur heard. Mother stated that the child had had chorea four years before, which was preceded by rheumatism, but which set in immediately after a fright (chased by a dog). There was another attack a year after first, but no cause could be given. |

CASES OF CHOREA—*continued.*

| No. | Name. | Age. | No. of Attacks. | Rheumatism. | Heart. | General Remarks. |
|---|---|---|---|---|---|---|
| 5 | Mary T. . . | 11 | 1 | Rheumatic pains came on a fortnight after scarlatina, followed by chorea. | Very faint systolic murmur occasionally present at apex. | Choreic movements not very severe and confined to the right side. On discharge from hospital heart normal. |
| 6 | Maggie E. . | 10 | 1 | Rheumatic pains before the attack. | Double murmur at apex; a soft systolic heard to left of apex and also in axilla; a rough presystolic heard to right of apex, and accompanied with frémissement cataire. | Case of right hemichorea. Both murmurs persisted on recovery from the chorea. |
| 7 | Thomas J. S. | 13 | 1 | Rheumatism two years before the attack of chorea. | Condition of heart during attack not known. | Was brought to out-patients' room suffering from bronchitis (secondary to heart affection). Mother stated that he had had a severe attack of chorea five years before. When seen, he had a rough systolic cardiac murmur at the base, and a systolic murmur at the apex. |
| 8 | Catherine B. | 12 | 2 First attack came on suddenly after a fright. Second attack, three years afterwards, came on gradually. Attributed to overstudy. | No history of rheumatism. | Action of heart irregular; no murmur. | Severe case of general chorea; the movements, however, more marked on left side. Great emotional excitement. Present attack (second) came on gradually. |
| 9 | Sarah J. D. . | 8 | 1 | None. | Soft mitral systolic murmur. | Attack commenced abruptly after a fright (chased by a cow). Movements general, but commenced in right side, and were always more severe in it. When she left hospital the murmur still present. |
| 10 | Ellen O. . . | 10 | 1 | None. | Nothing abnormal detected. | Attack came on gradually. Face and arms principally affected. Child very emotional. No cause assigned for the chorea. |
| 11 | Sarah S. . . | 7 | 1 | None. | Irregular action of heart; no murmur. | No history of rheumatism or fright. Attack sudden in its onset. Movements general and very severe. |
| 12 | Annie E. S. . | 6 | 1 | None. | Nothing abnormal detected. | Very protracted and severe case of general chorea, which came on suddenly, but no cause could be given. No history of fright or of rheumatism. |

Of the twelve cases of chorea, the details of which are given in the annexed table, eleven were girls and one a boy. Cases 1 and 2 were sisters, and a cousin of their mother is also stated to have had chorea.

In five of these cases there was no history of rheumatism, while in the remaining seven there was. In five of these seven the chorea was preceded immediately by the rheumatism; in one case the rheumatism occurred two years previous to, while in another it set in during the decline of, the chorea.

With reference to the condition of the heart, it was noted that during the choreic attack there were cardiac murmurs in five cases out of the twelve; in two others the action of the heart was irregular. In other two the state of this organ during the attack is not known, but there were valvular murmurs in one (Case 4) four years afterwards, and in another (Case 7) five years afterwards. So of the twelve cases, in three only did the evidence afforded by physical examination warrant the statement that the condition of the heart was normal.

In five of the seven cases with cardiac murmurs the murmur was single, apical, and systolic; in the remaining two there was also in each a mitral systolic murmur; but in one (Case 7) this was accompanied with a rough systolic murmur at the base, and in the other (Case 6) with a mitral obstruction with presystolic thrill.

Of these seven cases with heart murmur there was in six a rheumatic history; in the remaining one this was absent, the chorea having come on after a fright. In one of the six cases complicated with rheumatism there was also a history of fright.

In four cases the murmur persisted on recovery from the chorea; in three of these there was a history of rheumatism; in the fourth there was no history of rheumatism, but of fright.

The evidence afforded by these cases warrants the conclusion that a mitral systolic murmur is of frequent occurrence in chorea, and that in many cases there is a close relationship between chorea and rheumatism.

How then is this heart murmur to be explained? I think it will be generally admitted that in the great majority of cases this murmur is caused by some organic change affecting the mitral valve: this seems to be rendered tolerably certain by the following facts:—

1.—The clinical fact that the murmur which was present during the chorea frequently persists after the attack has passed away.

2.—The evidence afforded by post-mortem examination. Raymond in his article (Danse de Saint Guy) in the "Dictionnaire Encyclopédique des Sciences Médicales" has given a table of seventy-nine fatal cases of chorea in which a post-mortem examination was made. All these cases, which he has brought together (in his table) from various sources, were observed between the years 1850 and 1878, and the autopsies made in them show that, as far as the heart is concerned, the predominating lesion is valvular endocarditis.

Again, Dr. Wilks, one of our ablest English pathologists, states that he has never seen a fatal case of chorea in which there was not some evidence of previous endocarditis, "the inner surface of the mitral valve being lined by a narrow row of bead-like vegetations."

Admitting, then, that in many cases the murmur is organic, what is the cause of the endocarditis which gives rise to it? Rheumatism will account for a number of these cases, as it is a well-known fact that in the rheumatism of children, even in cases where few joints are affected, cardiac complications are frequent; but it will not explain cases such as Case 9, where there has been no history of rheumatism, but where the attack took its origin in fright. In this case the murmur remained after recovery, and there seems to be no other explanation of its occurrence than that it was caused by the chorea. But even in some

cases in which no murmur has been heard during life, post-mortem examination has shown a morbid condition of the mitral valve. Thus Wilks gives the history of two fatal cases of chorea, both following a fright. In neither was a cardiac bruit heard during life, in neither was there any history of rheumatism, and yet in both there were found vegetations on the mitral valve.

But there still remain a number of cases in which the murmur, although present for a time, eventually passes away.

In explanation of this evanescent murmur, the following views may be stated:—

1.—It is held by some that the murmur is due to spasmodic contraction of the papillary muscles, producing a temporary insufficiency of the mitral valve. But it may be objected to this view that there is no good proof that the involuntary muscular organs participate in these choreic disorders; and further, if this theory were correct, the murmur should vary from time to time in its character, whereas we know it will remain often for a lengthened period without any change.

2.—Even in these cases many think that there is some lesion of the valve, and that the explanation of the cessation of the murmur is that the recent lymph is either washed away or absorbed.

3.—It is well known to careful clinical observers that murmurs heard at the mitral area do not always indicate that there is any organic change in the valve. Thus in severe cases of anæmia, a mitral systolic murmur may sometimes be detected, which, with the restoration to health of the patient, gradually disappears. The same phenomenon is occasionally met with in enteric fever; and Hayden has observed it in purpura, masturbation, and in excessive tobacco-smoking. Bristowe has given a series of cases, and my former teacher, Dr. Cuming, has described two very interesting cases, in both of which there was a mitral systolic murmur. In one case the murmur ceased under tonic treatment, but in the other, in which there was anasarca and congestion of the lungs, the patient died; but post-mortem examination showed nothing wrong with the mitral valve. In all such cases there is regurgitation at the mitral valve; and while it is difficult to give an explanation that will apply to all the different cases in which this phenomenon has been met with, it is probable that in the majority, if not in all, there is a weakened condition of the cardiac muscle, or of the *musculi papillares*, which, by preventing the normal closure of the valve, causes regurgitation, and thus gives rise to the murmur. With the gradual return to health the weakened muscle is restored to its normal condition, the valve again acts as in health, and the murmur disappears.

The same explanation has been applied to the evanescent murmur of chorea?

We conclude, then, that the cardiac murmur met with in chorea, while in certain exceptional cases it may be functional, is in the great majority of cases, whether there has been a history of rheumatism or not, due to some lesion of the mitral valve.

There still remains the question of the relationship of rheumatism to chorea.

In cases in which the rheumatism precedes the chorea, and in which endocarditis is present as shown by the cardiac murmur, the theory that the endocarditis is the connecting link between the two (the chorea being caused by capillary embolism) is certainly a very attractive one.

Cases 1 and 2 support this view. Thus the child, Case 2, was lying in bed suffering from acute rheumatism, and with a mitral systolic murmur present, when suddenly without any other exciting cause violent chorea set in. Here all the factors that produce capillary embolism were present.

This embolic theory is also supported by cases of hemichorea, as, *i.e.*, Case 1,

where there was also anæsthesia and weakness on the same side as that on which the movements were present. Cases of hemichorea have also been known to pass on to hemiplegia. Further, Hughlings Jackson says that embolism is the very process which could cause that unstable or excitable condition of the nervous system present in the disease.

The very great frequency of endocarditis, whether there has been a history of rheumatism or not, lends additional weight to this embolic theory.

Although Tuckwell, Broadbent, and Jackson have found emboli in fatal cases of chorea, still the cases in which emboli have been met with are few; and on the other hand, Dickinson, Wilks, and Moxon, and others have failed to find any microscopic emboli in the numerous cases which they have examined. It is difficult, moreover, to understand why chorea is not more common in adults, in whom embolism frequently occurs.

But even granting that this theory may be proved to be true, it will not explain the relationship of the rheumatism to the chorea when the former succeeds the latter (as in Case 3), nor cases where the chorea has followed the rheumatism, but in which no cardiac bruit has been detected. Further, there are still a large number of cases with no history of rheumatism at all. What we want, then, is some theory that will account for all cases of chorea, as well those in which there is a rheumatic history as those in which this is absent, and which will also explain the frequent presence of the cardiac complications.

Impressed with the necessity of giving an explanation that will cover all cases of chorea, some look upon a morbid condition of the blood as the cause, but it is more probable that the nervous system is the part primarily at fault, a view which receives additional weight from the fact that there is considerable evidence to show that rheumatism itself is a disease of the nervous system, and in this way can be explained the frequent association of rheumatism with chorea.

From the foregoing I would draw the following conclusions:—

1st.—That in those cases of chorea in which there is a cardiac murmur, this murmur is generally organic.

2d.—That the murmur is usually heard at the mitral area, and is systolic.

3d.—That in some cases, however, this murmur may be inorganic.

4th.—That in those cases in which the murmur persists after recovery from the chorea, there has often been a rheumatic history.

5th.—While in a large number of cases a close connection exists between rheumatism and chorea, and while there is considerable evidence to support the view that rheumatic endocarditis is the connecting link between the two (the chorea being due to emboli), that this embolic theory will not explain all the cases.

6th.—That there are cases of chorea without any cardiac bruit and without any history of rheumatism.

7th.—That in many fatal cases of chorea, some in which there had, and others in which there had not, been a history of rheumatism, and some in which there had, and others in which there had not, been a cardiac bruit, post-mortem examination has demonstrated a morbid condition of the mitral valve.

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## *On Subcutaneous Nodules connected with Fibrous Structures, Occurring in Children the Subjects of Rheumatism and Chorea.*

Dr. Thomas Barlow, London, and Dr. Francis Warner, London.

In the study of rheumatism and chorea we have observed during the last six years a number of cases in which subcutaneous nodules have occurred in different parts of the body. The subjects of our observations have been either children or young people, not more than eighteen years of age; but we find that similar lesions have been noted (although rarely) in persons of middle life.

These nodules being often inconspicuous, may be overlooked alike by the patient, the friends, and the doctor, and as they are mostly painless, and spontaneously subside, they are of little therapeutic import. But we venture to think that their natural history is of interest (1.) as showing their conformity to the general type of rheumatic inflammation, and (2.) as helping to establish the existence of the rheumatic diathesis in a given case where, as is so often the case, in childhood the evidence of well-marked arthritis may be wanting, and (3.) as helping to establish in some cases of chorea the relationship with rheumatism, obscure and empirical though that relationship may be.

We propose to give (1.) a summary of the facts which we have observed in regard to these nodules; (2.) a tabular account of 27 of the cases which we have separately or conjointly investigated; (3.) an abstract of the observations of French and German authors on the subject.

1.—The nodules in these cases varied in size from that of a pin's head to that of an almond. They were strictly subcutaneous; the skin over them being simply raised without infiltration. In no case was there any local heat or spontaneous pain.

In one case (XVI.) the child had cried out on account of pains referred to joints in the neighbourhood of which nodules existed; but pressure on the nodules elicited no complaint.

In another case (VII.) nodules situated under the scalp were slightly tender on pressure, and combing the hair gave rise to distress. With this exception there was no tenderness.

In all but two (XVI. and XVIII.) there was no redness of skin over the nodules, and in these it seemed possible that local pressure or friction had induced the redness.

The nodules were separable from the skin, and to a varying amount movable on the subjacent structures.

The back of the elbow, the malleoli, and the margins of the patella were the commonest sites. Other situations were the neighbourhood of the vertebral spines, the spine of the scapula, the crista ilii, the extensor tendons of the foot and hand, the temporal ridge, the forehead, the superior curved line of the occiput, the pinna of the ear.

They were often symmetrical.

They occurred discrete or in clusters.

In total number present in a single patient they varied from one (IV.) to fifty (XVIII.)

The small nodules were often more palpable than visible.

To demonstrate their existence it was often necessary to stretch the skin over them; those in the neighbourhood of the knee and elbow joints were best brought into relief by flexing the said joints.

In regard to evolution, the following varieties were noted :—

1.—Nodules appeared in one crop, *i.e.*, several nodules appeared simultaneously, in different parts of the body.

2.—Successive nodules appeared.

3.—Individual nodules when first noted had attained their maximum ; they remained stationary for a time and then gradually subsided.

4.—Individual nodules greatly increased in size under observation (III., XV.)

5.—Duration of the nodules varied considerably ; the shortest period noted was three days from eruption to disappearance (XII.) The longest period that they were noticed to persist without diminution in size was five months (I.) In another case (XIV.) some persisted for five months ; others had diminished in size, and others had disappeared. The duration varied between these limits.

6.—Nodules partially subsided and then underwent recrudescence (I.)

7.—After complete subsidence, so far as manipulation could determine, a new crop appeared (VIII.)

8.—A nodule which, so far as palpation could determine, had subsided entirely, was nevertheless found post-mortem still to exist though very small (XVII.)

9.—In many cases the temperature was normal when the eruption of nodules occurred.

10.—In some cases there was pyrexia, but there were other conditions present, such as pleurisy, pericarditis, &c., which might possibly have determined the pyrexia.

11.—($\alpha$) In no case were the nodules found to become bony, or ($\beta$) infiltrated with urate of soda ; and ($\gamma$) in no case did suppuration occur.

By the first of these characters the nodules were distinguished from the "nodi digitorum" of Heberden, which according to Charcot are always bony (*vide* "Maladies des Vieillards").

By the second character they were distinguished from gouty nodules ; by the third from scrofulides. From erythema nodosum they were distinguished by the absence of skin implication, and from syphilomata by there being no infiltration of surrounding structures.

We had the opportunity of making post-mortem examinations on three cases (V., VII., XVII.)

In regard to anatomical site, we found that they were attached to tendons, to the deep fasciæ, and in one case (XVII.) to the pericranium.

The cellular tissue was not involved. In Case XVII. they were numerous, and each about the size of a pin's head.

Corresponding with many of the nodules, there was a minute pit in the subjacent bone, apparently due to pressure. In no other case was there any evidence of the bone being implicated. The nodules found over one of the vertebral spines and near the knee and elbow joints had no relation to the periosteum, but entirely belonged either to the deep fascia or to tendinous expansions. They were round or oval, semi-transparent fibrous bodies, in appearance somewhat like boiled sago grains.

A nodule was removed during life by Mr. R. W. Parker from over one of the dorsal spines in Case XVIII., and Mr. Parker kindly made some microscopic sections of it. It was also attached to the deep fascia. On section it presented wavy strands of fibrous tissue, with caudate, spindle-shaped, nucleated cells and abundant vessels. It had, indeed, many of the characters of organising granulation tissue.

We shall now consider some of the clinical features of the subjects of these nodules. They were either children or adolescents, the limits of age being four and a half years and eighteen years. Seventeen were female, ten were male.

With regard to evidence of *rheumatism*, in two cases there was no history

of joint affection to be obtained. One of the two had suffered from a fever lasting for three weeks, but no other information was forthcoming. Of the remaining twenty-five :—

Eleven had under observation either unquestionable rheumatic fever, or at least arthritis with effusion in one or more joints.

In eight others there was a history of rheumatic fever, or of some joint trouble in which there was swelling present.

In six there was arthralgia (without swelling).

Other evidences of rheumatism were stiff neck (I.), thickenings along tendons, with contraction (XVIII.), acrid sweating (XXII.), and an attack of hyperpyrexia, which subsided within a fortnight, and during which a crop of miliaria rubra appeared (V.)

In a large number of the cases the skin was dry and harsh.

We believe that as a general rule it may be stated that in the rheumatism of childhood the skin is much less moist than in that of adults.

We adhere strongly to the view which has been held, that some forms of erythema are rheumatic manifestations. We think it important, therefore, to note that seven of our patients had erythema marginatum and erythema papulatum whilst under observation at or near the time of eruption of the nodules, and that another at a subsequent period developed this rash.

One had urticaria and another purpura.

With regard to the heart, we believe that in all these cases there was some morbid condition.

In one the only physical sign was an altered ("murmurish") first sound. This was the only case about which there could be any doubt, in all the rest the evidence of disease was abundant and conclusive.

In eight cases pericarditis developed under observation, and in two, in addition to extensive valvular disease, it was believed that there was adherent pericardium. In twelve the cardiac disease was progressive—that is to say, the valvular murmurs altered, and the dilatation increased under observation, in spite of the employment of digitalis and rest. Severe dyspnœa was a common symptom. Eight out of the twenty-seven patients died from the result of heart disease. Autopsies were obtained on five. In all there was mitral disease with dilatation ; in four there was disease of mitral and tricuspid valves ; in three, of mitral, tricuspid, and aortic valves ; in four, pericarditis.

With regard to chorea, ten of our patients were the subjects of this neurosis whilst under our observation, and another had suffered from it after a previous attack of rheumatic fever.

We submit in conclusion :—

1.—That subcutaneous nodules having such a life history as we have described truly conform to the general type of rheumatic inflammation, especially in their spontaneous tendency to subsidence more or less complete, and in their proneness to relapse.

2.—That they may be considered as in themselves indicative of rheumatism, even in the absence of pain.

3.—That when found associated with chorea and heart disease, although no history of antecedent rheumatic fever can be obtained, nevertheless, their presence gives a presumption that the chorea is rheumatic.

4.—That in regard to prognosis and treatment, although the nodules are unimportant in themselves, they are nevertheless of serious import, because in several cases the associated heart disease has been found actively progressive.

5.—That such nodules belong strictly to the fibrous tissues, and in nature are probably homologous with the inflammatory exudation which forms the basis of a vegetation on a cardiac valve.

## TABULAR ACCOUNT OF CASES OF SUBCUTANEOUS NODULES.

### DR. BARLOW'S CASES.

| Name and Age. | Nodules. | Skin. | Temperature. | Heart. | Chorea. | Evidence as to Rheumatism. |
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| Case I. Percy S. 8½ years. July, 1875. Fatal. | On extensor tendons of first and second fingers, both sides. These were left after first attack of rheumatic fever. They partially subsided. Patient was seen again sixteen months afterwards. At this period (three months after a second attack of rheumatic fever) he had nodules on tendons of extensor indicis and of palmaris longus of both sides. These diminished, but did not disappear. One month afterwards a nodule appeared in front of left knee, and four months afterwards many fresh nodules about the metacarpo-phalangeal joints. | Frequently moist. No erythema observed. After one of the febrile attacks he had some desquamation. | Elevated during the attacks of rheumatic fever; but on many occasions when the nodules were present there was no pyrexia. | Hypertrophy and dilatation. Mitral regurgitation(?). Adherent pericardium. Death from cardiac dropsy. | None. | History of two febrile attacks, which lasted about three weeks. There was pain in the joints and sweating. After the first attack there was a stiff neck, and the first crop of nodules appeared. After the second attack the knuckles were a little swollen, and the second crop of nodules appeared. The knuckles gradually enlarged a little. |
| Case II. Mary C. 8 years. Aug. 1877. | Movable nodules over vertebral spines of middle dorsal region; over olecranon and metacarpo-phalangeal joints of ring and little fingers of both sides. Over both knees. | No eruption. Skin dry. | 101° F.; but observe there were some signs pointing to tubercular disease of right apex, which might have accounted for the pyrexia. | Mitral regurgitation. | None. | No history of arthritis. |
| Case III. Charles C. 15 years. Feb. 1878. | Right elbow:—One above external condyle, one on external condyle, one below external condyle, five around olecranon. Right hand:—Three connected with extensor tendons on back of hand; one on each knuckle. Left upper limb—nodules as on right. Lower limbs—nil. Three days after first eruption, several small nodules appeared over the styloid process of the left ulna. Also some on the dorsum of each foot. Eighteen days after first eruption a group of three nodules, each the size of a hempseed, appeared on the posterior surface of the pinna of the right ear. Twenty-three days after first eruption there were no fresh nodules, but several had increased in size. One hundred and nine days after first eruption the nodules had entirely disappeared. | Had two attacks of erythema marginatum whilst under observation. | Pyrexia during one attack of erythema marginatum, 100°—101°. Probably no pyrexia at efflorescence of nodules; certainly none during considerable time when they were under observation. | Mitral and aortic regurgitation. Dilatation. Probably adherent pericardium. Much cardiac dyspœna. Bruits altered. Was readmitted on account of severe cardiac dropsy. | None. | Stated to have had rheumatic fever two years ago, and to have been laid up five months with it. Since then breath short, and has frequently suffered with painful swellings of ankles, knees, and knuckles. Admitted with slight swelling around both knee-joints; and eight days after had some swelling of the ball of left great-toe. This was followed by an attack of erythema marginatum, and seven weeks afterwards by the eruption of nodules. |

CASES OF SUBCUTANEOUS NODULES—*continued.*

| Name and Age. | Nodules. | Skin. | Temperature. | Heart. | Chorea. | Evidence as to Rheumatism. |
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| CASE IV. Elizabeth R. Under 10 years. Sept. 1878. | One nodule over one internal malleolus. This remained unchanged during the fortnight that the child was under observation. | No erythema. Skin dry. | Not noted. | Murmurish first sound. | Slight chorea. | History of both ankles being swollen eight months ago. She was not kept in bed for it. No arthritis at present. |
| CASE V. Henry R. 10 years. Nov. 1878. Fatal. | On the back of each hand connected with extensor tendons; over both elbows; on both knees, and on both external malleoli; on the occipital region. These nodules had been noticed by the boy's mother three weeks before admission, and five weeks after the onset of pains in large joints. She believes they did not appear all at once. Partial subsidence during the eighty-two days that patient was under observation. One examined post-mortem found to be solid and fibrous in structure, the size of a hempseed, intimately blended with the tendon of the triceps at its insertion into the olecranon. | Dry and harsh. During a febrile attack, which lasted about a fortnight, there was a small crop of miliaria rubra. | Normal on admission, when the nodules were present. Subsequently became febrile. Temperature, 105°, without fresh joint manifestations, but with increase of heart mischief, and miliaria rubra. Temperature lowered by cold packing. Became nearly normal again in about a fortnight. | Mitral regurgitation and obstruction. Pericarditis. Post-mortem:—Pericardium coated with recent lymph, but just above apex some organised lymph of older date. Left auricle dilated, and endocardium thickened. Mitral valve much thickened, and posterior curtain shortened. Also thickening of tricuspid valve. Recent vegetations on tricuspid and mitral. | Moderately severe. Came on about six weeks after onset of joint symptoms, and a fortnight after appearance of nodules. Had almost entirely disappeared in eight weeks after admission. | History of pains in large joints for two months before admission, which pains had not compelled his being kept to bed. When admitted, no redness over joints, but some effusion in right knee, and the boy complained of pains in both knees. Temperature was normal, but in a few days began to rise, and reached 105° (*vide ante*). This was believed to be an attack of masked rheumatic fever. Afterwards the temperature was nearly normal, and the patient seemed moderately comfortable. Died from a fresh attack of pericarditis eighty-two days after admission. |
| CASE VI. Kate B. H. 6 years. Dec. 1878. Fatal. | Over external condyle of humerus on both sides; over right external malleolus, three—of which one is attached to the tendon of the peroneus longus, and two are free from it; one over right internal malleolus; one connected with tendon of right extensor proprius hallucis; two over left external malleolus connected with the peroneus longus. These nodules had not been noticed by the parents, so that the period of their onset cannot be fixed. During the three months that the child was under observation they diminished in size. No fresh ones appeared. | No erythema. Dry. | Irregular hectic temperature for at least two months. Morning, 97°; evening, 101°—102°. But observe, during this time she had pericarditis and pleurisy. | Mitral obstruction and regurgitation. Pericarditis. Bruits varied much under observation. A high-pitched musical murmur developed, which subsequently disappeared. Extreme dyspœna. Died with fresh access of pericarditis. Post-mortem refused. | Moderately severe at first, but rapidly subsided after admission. | History of rheumatic fever, with swelling of knee and elbow joints four months ago, succeeded by St. Vitus's dance. |

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| CASE VII. Martin D. 8 yrs. 11 mos. April, 1879. Fatal. | One over each superior curved line of the occiput (these were tender on pressure: the boy cried when his hair was combed); one over left external condyle of the humerus; two over left olecranon; one attached to the extensor tendon of the right middle finger over the metacarpo-phalangeal joint; one over the upper portion of the external condyle of each femur; some thickening of the right tendo-achillis and over the left external and internal malleoli. The nodules above his knee-joints had been noticed by the boy's mother for one month before admission. During the eighty-seven days that he was under observation no fresh nodules appeared. Several subsided, but many remained. Post-mortem: the nodules about the elbows and knees were found attached to tendinous expansions; they were semi-transparent fibrous bodies, in appearance somewhat like boiled sago grains, but less defined. | No erythema. Skin generally dry. | For three weeks normal. Subsequently, normal in the morning; not more than 100·6° in the evening. | Presystolic and systolic murmurs. Rapidly progressive dilatation. Post-mortem: — Slight recent pericardial effusion, with thin shreds of lymph. Great dilatation of all the cavities. Vegetations on all the valves, except those of the pulmonary artery. Large nutmeg liver. Congested kidneys. Slight serous effusion right pleura. Small caseous mass in middle of upper lobe of right lung. An adherent thrombus in the left anterior cerebral artery. A great number of small cortical extravasations on the surface of the brain, behind the fissure of Silvius and over the tempero - sphenoidal lobes, (?) the result of capillary embolisms. | Had existed for eighteen days before admission; not severe, bilateral. Entirely disappeared in nine weeks after admission. | History of pains in knees for one month before admission. No effusion when admitted. |
| CASE VIII. Emma D. 12 years. July, 1879. | Over upper part of both patellæ. One over the right internal malleolus; two over the right external malleolus, of which one is attached to the tendon of the peroneus longus; one over the left external malleolus; one over the left olecranon. These nodules appeared twenty days after admission, and after the subsidence of the rheumatic symptoms, with the exception of slight effusion in the sheaths of tendons on the back of the left hand and a soft cardiac murmur. Twenty-one days after their eruption all had subsided except one small nodule over the right internal malleolus. | Erythema papulatum and erythema marginatum on admission. This continued for ten days. No copious sweating at any time, but subsequently skin a little moist. | Marked hectic for the first twenty-one days —99° morning, 101°–102° evening. The day when the nodules appeared there was a morning temperature of 99° and evening temperature of 101·6°. During week afterwards, 99° morning, 100° evening. | Pericarditis, systolic murmur heard at both base and apex, which afterwards cleared up. | None. | Said to have had rheumatic fever four times; the last attack was four months ago, for which she was in hospital two months. She had pyrexia, slight sweating, effusion in knee-joints, one shoulder, and in sheaths of tendons on back of each hand and about the ankles. She had also two nodules over the right elbow and two on the back of one hand. In the present attack there were effusions in the sheaths of the tendons on the back of each hand and around the ankles. |
| CASE IX. Alice H. 12 years. Aug. 1879. | One over spine of right scapula; one over olecranon. | No erythema. | Not taken. Skin cool. | Mitral regurgitation. | None. | History of pains in limbs for six weeks, and of swelling in ankles and wrists. Child poorly, and off her appetite. Tongue furred. Has pains in ankles now, but no swelling. |

## Cases of Subcutaneous Nodules—*continued.*

| Name and Age. | Nodules. | Skin. | Temperature. | Heart. | Chorea. | Evidence as to Rheumatism. |
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| Case X. Mary G. 5 years. Aug. 1879. | Two over right olecranon. | No erythema. | Not taken. Skin cool. | Mitral regurgitation. Hypertrophy. | None. | Brought for debility. Mother gives a history of the child having had a fever one year ago, which lasted for three weeks. No history of joint affection. There is no arthritis at present. |
| Case XI. Arthur E. 7 years 3 months. Aug. 1879. | One over each olecranon; one over each patella. Date of onset not known. Subsided within two months after the boy was admitted into the hospital. | Dry. One year and ten months afterwards was readmitted to hospital with extensive erythema marginatum. | Generally normal. | Mitral obstruction and regurgitation. Dilatation. Probable adherent pericardium. Much dyspnœa. Brought to hospital on that account. | None. | History of a bad attack of rheumatic fever eight months previously, which attack kept him in bed three months, and left him short-breathed and exceedingly pale. No history of arthritis in present attack. *Postscript.* — One year and ten months subsequently was readmitted with erythema marginatum, but without nodules. Some effusion in one knee-joint, and some fresh pericarditis developed whilst under observation. |
| Case XII. Mary A. W. 10 years. Nov. 1879. | One on tendon of right tibialis anticus above internal malleolus. This was first noticed fourteen days after admission. It disappeared in three days. | Dry. No erythema. | Normal. | Mitral regurgitation. | Bilateral. Not severe. Appeared five weeks after onset of rheumatic symptoms. | History of pains in legs and arms ten weeks ago, which continued for five weeks, but were not bad enough to compel her to go to bed. |
| Case XIII. Rosina B. 11½ years. Nov. 1879. | Over right olecranon, and over external condyle of left humerus; over left patella. | No erythema. | Normal. | Mitral regurgitation. | Bilateral. Had existed for three months. | History of rheumatic fever four years ago. |
| Case XIV. Walter B. 11 years 10 months. April, 1880. | Over external condyle of humerus; over olecranon; over lower end of radius on both sides; over both patellæ; above each external malleolus. Size, split pea to hempseed. These diminished but did not entirely disappear during the three weeks that he was under observation. | No erythema. | ... | Mitral and aortic regurgitation. Dilatation. Bruits altered under observation. | None. | History of two previous illnesses, in which the boy did not keep his bed, but had pains in joints, the knees being swollen and subsequently stiff. Present attack began five months ago with pains in joints, palpitation, and short breath. Three weeks after the onset nodules were noticed by his mother about the elbows, wrists, and knees. |

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| CASE XV. Arthur D. 18 years. Feb. 1881. Under the care of Dr. Coupland, Middlesex Hospital. | Over vertebral spines from third dorsal downwards; several along spine of each scapula, and one on each acromion; several along clavicles and beneath subcutaneous tissue of neck; several (20–30) over forehead, temporal regions, and occiput; over external condyle of each humerus; on the back of each hand connected with the extensor tendons; connected with the palmaris longus and flexor carpi radialis tendons; connected with the hamstrings and the tendons of the common extensor on the dorsum of one foot. The nodules on the back of each hand were first noticed three weeks after admission. During the forty-four days afterwards that he was under observation several fresh crops appeared; those noted on the forehead increased from day to day. Some of the individual nodules grew to be as big as Barcelona nuts, but the average size was that of a split pea. Three months after his discharge, that is to say, nearly five months after onset of nodules, they had entirely disappeared. The patient states that a former severe attack of rheumatic fever (five and a half years ago) left him with lumps like the above, and that they entirely disappeared after a time. | No erythema marginatum. For a day or two the skin was "clammy;" generally dry. | During part of the time when fresh nodules were appearing there was a tolerably constant evening temperature of about 100°, and a morning temperature of 98·4°. It was noted, however, that on two occasions fresh nodules appeared on days when both morning and evening temperatures were normal. | Mitral obstruction and regurgitation, and aortic obstruction. An aortic regurgitant murmur developed whilst under observation. Dilatation. | None. | History of rheumatic fever, with pain and swelling of joints, five and a half years ago. Was ailing for ten months with it. Present attack:—Began fourteen days before admission with pains in many joints, sore throat, and short breath. There was effusion in both knee-joints, swelling about ankles and one wrist, and some thickening of metacarpo-phalangeal joints. Evening temperature 102°. Pain and swelling rapidly subsided, but relapses, especially in the wrist, occurred, and the heart mischief increased. There was much wasting and anæmia, and slight œdema of the feet appeared whilst in the hospital. This cleared up, and then the patient was discharged with still a number of nodules present. |
| CASE XVI. Mary I. V. 5 years 2 months. May, 1881. | One on external condyle of right humerus; two over right olecranon; one on knuckle of right index finger; one on external condyle of each femur; three on right internal malleolus; two on left internal malleolus; one on left external malleolus. A large nodule on outer side of each tendo-achillis, near its insertion; two higher up on the right tendon; one on the left. Over the nodules about the malleoli (and over these only) there was slight redness. There was also a little complaint of pain about the ankles; nevertheless, pressing the nodules did not evoke any complaint. Some of the nodules were noticed by the mother six weeks before admission. The mother spoke positively about the appearance of successive nodules. During one week that the child was an out-patient fresh nodules formed, but during the nineteen days that she was in the hospital in bed no fresh nodules appeared, and those present on admission soon subsided. Thus the total duration was not probably more than two months. There were no black and blue changes in the skin over the nodules about the ankles, where there had been slight redness. | No sweating. No erythema marginatum. Slight redness over some of those nodules which were situated about the ankles. | Normal when nodules were first seen, at which period fresh ones were forming. During nineteen days that the child was in hospital, when the nodules were subsiding, temperature normal. | Slight hypertrophy. Mitral regurgitation. | None. | There is a strong family history of rheumatism on the mother's side. This child five months ago, after a sea-voyage, had complained much of aching in her joints. The joints were not swollen, but they were stiff. For about five weeks before she was brought to the hospital she had complained much of pains in her joints, especially at nights. There had been no swelling or redness except over some of the nodules before described. She had not sweated at all. During the period that she was in the hospital there was no arthritis. |

## Cases of Subcutaneous Nodules—*continued.*

| Name and Age. | Nodules. | Skin. | Temperature. | Heart. | Chorea. | Evidence as to Rheumatism. |
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| Case XVII. Mary L. J. 5 years 11 months. Aug. 1881. Fatal. | One over the eleventh dorsal spine appeared three days after admission into hospital, and twenty-four days after the onset of joint pains. When first felt it was the size of a split pea; During fifteen days it subsided so that it could no longer be felt. But post-mortem a small nodule was found having the appearance of a boiled sago grain, situated in the deep fascia over the eleventh dorsal spine. On the tenth day after admission a nodule appeared on external condyle of the right humerus. This subsided in a few days. Some minute nodules were found post-mortem which had not been felt during life. They were of the size of a pin's head, and were situated in the substance of both temporal fasciæ and the pericranium between them. They were at least thirty in number. Corresponding with many of those situated in the pericranium there were minute pits in the outer surface of the parietal bones, especially on the left side. These pits were apparently caused by pressure. | Erythema marginatum and papulatum for one month before admission. Situated on trunk and lower limbs. Faded in two days, leaving slight staining. Fresh patches appeared on the seventh, eighth, ninth, and tenth days after admission. No sweating. | For eight days the morning temperature was generally normal. During the succeeding ten days it was generally 102·2°. The evening temperature ranged from 100° to 102°. | On admission, systolic and presystolic apex murmurs. On the sixteenth day after admission well-marked pericardial friction was heard. She had had vomiting and much dyspnœa for four days previously. Post-mortem :—Universal pericarditis, with one ounce of serous effusion, Both auricles dilated. Endocardium of left auricle thickened and opaque. Mitral valve thickened; many recent vegetations on it; some small vegetations on one aortic valve. Left ventricle slightly hypertrophied. Heart and pericardium together weighed 10 oz. Slight recent pleurisy left side. Liver nutmeg. Brain and membranes healthy. Nodules *vide ante.* | Slight for two days before death, affecting face, muscles, and speech. | History of rheumatic fever, in which all her joints were affected one year ago. She was in bed for one month with it, and has not been strong since. Present illness:—Has had flying pains in joints for three weeks. When admitted the pains ceased, and there was no arthritis. On the seventeenth day there appeared distinct effusion with pain and redness in the right wrist-joint. No other joints were affected. |
| **DR. WARNER'S CASES.** | | | | | | |
| Case XVIII. Olive B. 9½ years. July 31, 1878. | One over each spinous process; large ones over both scapulæ at spine and acromion, also at the angles. That at angle of left scapula had the skin over it a little reddened; it had been rubbed with some liniment. Others over prominences of elbows and wrists; small ones over knuckles and inter-phalangeal joints. Large ones surrounded the patellæ; others over extensor tendons of ankles. There were forty or fifty in all. They appeared five weeks before admission; no fresh ones developed under observation. They gradually disappeared in ten weeks from the time of onset. The nodule was excised from the back by my colleague Mr. R. W. Parker, to whom we are indebted for microscopic sections of the same. **Vide text.** | No erythema. | No pyrexia. | A distinct mitral regurgitant murmur which did not vary. | Followed the joint pains and development of nodules. Well marked, principally affecting right side. | Father rheumatic. Six weeks before admission joints painful; this pain, much increased by movement, increased and laid her up. When admitted, joints painful but *not swollen.* The fingers of both hands contracted and flexed; the attempt to straighten them caused pain. The sheaths of palmar tendons presented ridges, and were tender; this condition passed away completely from the right hand, but still existed in the left hand when she was discharged. |

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| Jan. 13, 1879. Fatal. | crests, on both olecranon processes, on external condyle of each humerus, over peroneal tendons of each ankle, and on the edge of the left patella. Next day others seen over knuckles. A month later erythema returned accompanied by fresh nodules over the sternum and metacarpal bones. | after admission erythema papulatum and marginatum appeared in successive crops. Patches increased in size and were not painful. Rash and nodules coexisted. | F. with eruption of nodules temperature normal. | heart's action became quick and irregular. Post-mortem:—Complete pericardial adhesion; heart dilated and hypertrophied. Vegetations on mitral and aortic valves. Tricuspid valve thickened. Lungs congested. | | in joints in present illness, no distinct effusion. |
| CASE XX. Selina H. 18 years. Nov. 19, 1880. | At base of first phalanx of left middle finger flexor surface an elongated lump ¼-inch long, not tender, movable laterally but not lengthwise; apparently connected with sheath of tendon; skin over it movable. (This developed early in the case.) Small movable nodule over head of left radius; thickening over extensor tendon of left great-toe; thickening over right and left peroneal tendons. These developed when patient was convalescing. There was a coincident onset of pleurisy and a rise of temperature. | ... | 100°–101° F. It was elevated during the eruption of the nodules, but this was probably due to the coincident pleurisy. | Aortic and mitral disease; probable active endocarditis in hospital. Murmurs subsequently noted as less distinct. Heart's action at times tumultuous with dyspnœa. | None. | Arthritis under observation. Pains in all the joints. In-patient two years ago for rheumatism and endocarditis, a mitral regurgitant bruit then developed. |
| CASE XXI. George I. 14 years. Feb. 3, 1880. | Situated on acromial end of both clavicles; over right olecranon nodule ¼-inch long; left only a slight thickening; others over knuckles. Over each patella 5 nodules size of split pea, symmetrically placed. They were said to be of several weeks' duration, and to have been preceded by pain in the limbs; they were never painful, and became smaller in a fortnight, finally all disappeared. | Skin harsh and dry; no erythema. | 99°–100° F. subsided in a few days. | Mitral regurgitation on admission. Pericarditis subsequently developed in hospital. | None. | Mother had had rheumatic fever. Patient complained of pain in limbs of several weeks' duration. Slight tenderness of several large joints with distinct effusion. |
| CASE XXII. William W. 14 years. Nov. 22, 1880. | First noted a few days after onset of chorea. Situated over knuckles of second and third fingers of right hand. Ill defined over right external condyle of humerus; others over lower part of sternum. They began to disappear in three weeks. | Clammy with sour sweat. | ... | Mitral regurgitation; bruit became more developed. | Present: Three previous attacks—the first, five years ago, lasted three months; the third attack followed on rheumatic fever. | Rheumatic fever two years ago. Sour sweating in present illness. |
| CASE XXIII. Emma H. 12 years. Aug. 2, 1879. | Situated over both elbows and hands on extensor tendons; over external malleoli. They did not appear till the subsidence of the erythema and pericarditis. When patient left hospital, five weeks after their eruption, they were subsiding. | Urticaria and erythema in patches on back and limbs; it came out with itching. A second crop of urticaria was not followed by nodules. Sweating. | 102°–103° F. on admission; a week later normal; so also on the eruption of nodules. | Mitral regurgitation and pericarditis on admission; a week later the bruit at apex became musical. A fortnight later an aortic systolic bruit developed. | None. | Laid up with rheumatism about a year ago. In present illness arthritis in large joints, with sweating. |

CASES OF SUBCUTANEOUS NODULES—*continued.*

| Name and Age. | Nodules. | Skin. | Temperature. | Heart. | Chorea. | Evidence as to Rheumatism. |
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| CASE XXIV. Emma G. 4½ years. Dec. 11, 1878. | Numerous; situated over both olecranon processes; left internal and right external condyle of humerus; external malleoli. Extensor tendons of toes over metatarso-phalangeal joints; over spine of scapula. Most disappeared in a month. | ... | 99 °F. | Mitral regurgitation on admission. No change in bruit. | Slight; commenced after nodules appeared. | Brought to hospital on account of lumps. Has had sub-acute joint pains. Family history:—Father rheumatic; sister has had rheumatic fever and heart disease. From history it appears certain that she also had nodules. |
| CASE XXV. Keziah I. 16 years. Oct. 1880. Fatal. | Right hand: — Over knuckles of first and middle finger, six in all. Left hand:—Eleven in all, distributed over the four knuckles. One over head of left ulna. All appeared in one eruption, along with erythema marginatum, fifteen days after admission, and had completely disappeared within one month. | Erythema marginatum on trunk, and slight itching, along with eruption of nodules. Fresh patches three days later, without additional nodules. Disappeared within a week. Skin moist. | 101° F. on admission; after a few days, normal. No rise with eruption of nodules. Subsequently 102° F., with severe cardiac dyspnœa. | Mitral regurgitation and dilatation; intense dyspnœa, followed by death. Post-mortem:—Left ventricle dilated and full of clot; vegetations on aortic, mitral, and tricuspid valves; lungs œdematous. | None. | Six months ago laid up eight weeks with rheumatic fever, and has been short of breath ever since. On admission this time several joints painful, but no effusion. |
| CASE XXVI. Emma K. 17 years. Nov. 26, 1880. Fatal. | Over spine of right scapula; several over both condyles of right humerus and olecranon; several over both condyles of left humerus; over dorsal and lumbar vertebral spines; two on left patella. Date of onset could not be fixed. During the six weeks that she was under observation there was slight diminution in size, but none of the nodules disappeared. | No erythema. | 98°–99° F. Once it reached 101° F. | On admission, mitral regurgitation and dilatation; dyspnœa on exertion and cardiac pain. Subsequently double aortic bruits. Whilst walking about, sudden syncope set in, and she died. | Had chorea in 1875, after first attack of rheumatism. Not in present illness. | Had rheumatic fever in 1875, and then had mitral regurgitation only. Joint pains, but no effusion in present illness. |
| CASE XXVII. Emma B. 13 years. Nov. 15, 1880. | One over first inter-phalangeal joint, and over knuckle of right index finger (these appeared during relapse of arthritis). A second crop a week later on other phalangeal joints; also five on external condyle of each humerus. Small nodules over left olecranon; several over both patellæ and over each external malleolus. Some of them diminished under observation. | Sweating. Erythema marginatum appeared after the arthritis subsided, and lasted three days; it returned with the nodules. Purpuric spots of legs during convalescence. | On day of admission 105° F.; fell next day. Average, 100°–101° F. | Slight mitral regurgitant murmur, which increased. Thrill developed under observation. | None. | Mother has had rheumatic fever. This patient had rheumatic fever twelve months ago, with pain and swelling of joints. In the present attack she had been laid up with pain, swelling of joints, and sweating. The arthritis had subsided previous to admission, but relapsed under observation in the hospital. |

## BIBLIOGRAPHY.

We find that nodules of the kind which we have described have been referred to by other writers. Our first case was observed in 1875, but Dr. Meynet of Lyons had under his care in 1874 a boy aged fourteen years who presented similar lesions. His case is recorded in the *Lyon Médical*, No. 49, 1875. The patient had had two previous attacks of rheumatic fever, and was the subject of heart disease. He was admitted to the "Charité" after having suffered for six weeks with pains in the joints and palpitation. When admitted he had very little pyrexia, and scarcely sweated at all, but there was some effusion in the sheaths of the tendons about the hands. Around the metacarpal and phalangeal joints there were numerous small deposits which were movable. Connected with the flexor tendons in the forearm and on the inner edge of each ulna were other nodules, varying in size from a hempseed to a Barcelona nut. Other positions for the nodules were :—around the knee-joints, the tendons of both feet, the vertebral spines, the forehead, and the crown of the head.

Dr. Meynet observed several of these growths from day to day, and that they were not affected by the use of iodide of potassium. Many had subsided at the end of a month, but there were still several deposits to be seen.

Dr. Rehn, in his article on Acute Rheumatism in "Gerhardt's Handbook of Children's Diseases" (p. 21, 1st div. 3d vol., 1878), has described similar tumours in a girl aged ten years suffering from relapsing rheumatism. These, at the outset, were decidedly painful, but afterwards became insensitive to pressure, and disappeared without treatment. Since the above article was written, Dr. Rehn has seen another case of nodules in a child aged four and a half years, who was the subject of articular rheumatism, with pleurisy, pericarditis, and peritonitis.

In this case also, at the outset, the nodules were a little painful, but they soon became insensitive to pressure, and diminished in size and number.

Dr. Hirschsprung of Copenhagen has written a valuable and exhaustive paper on the subject in the *Jahrbuch für Kinderheilkunde*, March, 1881.

He records four cases which came under his own observation. The first of these, after the lapse of seventeen months, had a subsequent attack of rheumatism, in which nodules reappeared. Some of them appear to have been distinctly tender on pressure, and the skin over them was a little reddened. Dr. Hirschsprung had an autopsy on one of his cases in which the heart lesions were very severe.

Two nodules removed from the tendon of the triceps brachii at the olecranon were examined microscopically. Besides the strands of fibrous tissue, spindle-shaped and caudate cells, and the abundant vascular supply to which we have referred in one of our cases, Dr. Hirschsprung found in some places multinucleated cells, isolated and in groups, and some areas where there was slight indication of necrobiosis.

In the *Revue de Médecine*, April, 1881, Messrs. Troisier and Brocq have recorded one case of rheumatic nodules which came under their observation.

This was in an adult aged forty-five, who had had two previous attacks of acute articular rheumatism, and was admitted into hospital after a fortnight's illness for a third. He had effusions in the large joints, high fever, and a mitral regurgitant murmur. In eight days he developed double pleuritic effusion, but was convalescing, when, on the forty-third day, some small tumours appeared on the occiput. The patient has been led to notice them on account of the pain caused by resting his head on the pillow. He affirmed that exactly similar tumours had formed on his forehead and on the scalp in the course of his

second attack of rheumatism. There were about ten of these tumours. They varied in size from that of a pea to that of a small hazel-nut. They were not the seat of spontaneous pain, but continued pressure gave rise to distress. They were very slightly movable on the bone, and the scalp was movable over them.

About two months after admission, the patient had a relapse of joint pains and an attack of pericarditis, and three days afterwards some fresh nodules appeared on the forehead. In two days more there was a nodule on each ear, and fresh rheumatic pains in the knees and elbows. After this there was a gradual subsidence in number and size, and about fifteen weeks after admission there were no nodules to be found anywhere.

Messrs Troisier and Brocq, not having had any autopsy, and not being acquainted with the valuable memoir of Dr. Hirschsprung, can only speculate on the anatomical site and nature of these lesions. From our observations, based on post-mortem examination, it will be seen that we agree with the belief of these authors, and also of Dr. Meynet, that the lesions belong strictly to the fibrous tissues and not to the cellular tissue, and that they are attached specially to the sheaths of tendons, to the deep fasciæ, and, in the case of the head, to the pericranium.

It appears from the foregoing memoir that Jaccoud and Besnier have both referred to the rare occurrence of these nodules in rheumatic subjects, and M. Féréol has also recorded the case of a lady who comes of an arthritic stock, but has not herself suffered from rheumatism. This patient is the subject of obstinate pityriasis of the scalp and of severe migraine. She has on many occasions had subcutaneous nodules on the scalp, which have come and gone very rapidly. Whether this is one of the same group must remain, we think, an open question.

## DISCUSSION.

Dr. Ranke, Munich: I wish to point out the great importance of the study of the geographical distribution of disease. I think that future International Congresses might do most useful work if general attention were drawn to this point and followed up. In regard to chorea, I am inclined to think that it is much less frequent in some places than in others. I have lately searched my books, and found that amongst 40,723 children treated by me in the University's dispensary for sick children since 1867, there have been only nineteen cases of chorea, and amongst these nineteen only three have presented a systolic murmur; in all the others the heart sounds were found normal. Amongst the three cases with mitral trouble, there was only one in which an attack of acute rheumatism had previously occurred. Of the nineteen cases, two followed immediately after fright; in all the rest an immediate cause could not be discovered.

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## *Sur l'Erosion des Dents considerée comme Signe retrospectif de l'Eclampsie infantile.*

Dr. Magitot, Paris.

Monsieur le President, Messieurs,—Tous les médecins savent, et en particulier ceux qui s'occupent spécialement des maladies des enfants, qu'on observe fréquemment à la surface des dents permanentes, et plus rarement des dents

temporaires, une certaine altération de tissu de nature congénitale, et consistant soit dans des échancrures du bord libre, soit dans des sillons annulaires plus ou moins profonds et plus ou moins nombreux, et toujours symétriques aux dents homologues d'une même mâchoire. Cette lésion a été désignée sous le nom générique d'*érosion*.

Or l'histoire de cette altération appartient au domaine de la séméiologie générale, car elle est, de l'avis unanime des auteurs, l'indice permanent et indélébile d'un trouble de formation de la couronne dentaire, trouble survenu pendant la période intra-folliculaire, et dont la cause pathologique est nécessairement contemporaine de la période du développement à laquelle correspond le niveau même de l'altération.

Une première assimilation, très-légitime d'ailleurs, rapproche de l'érosion dentaire la production du sillon unguéal de Beau, qu'on pourrait appeler justement l'érosion de l'ongle, mais avec cette différence, que l'érosion de l'ongle se montre de suite à la base de celui-ci et disparaît par le fait de son rapide développement, tandis que l'érosion intra-folliculaire d'une dent n'apparaît que plus tard, à l'époque de l'éruption ; la première est conséquemment fugace, tandis que la seconde est permanente et indélébile.

Une autre assimilation rattache encore à ces deux érosions *unguéale* et *dentaire*, la lésion connue sous le nom de *cataracte zonulaire congénitale*, décrite par les auteurs allemands, et en France par M. Nicati, et ce triple rapprochement est d'ailleurs conforme à la loi physiologique qui classe dans le même ordre de tissus, les *produits*, *l'organe dentaire*, *l'ongle* et le *cristallin*.

Or, pour nous borner ici à l'étude de l'érosion dentaire, et afin de fixer les rapports réciproques entre le lieu exact de cette lésion sur une couronne en voie de formation et l'âge de l'intervention de la cause productrice, il nous faut établir quelles sont les phases du développement de la couronne des dents, et plus rigoureusement quel est, à une époque déterminée, l'état du *chapeau de dentine* pour les différents follicules. C'est ce qui résulte du tableau suivant.

Les indications fournies par ce tableau ne doivent pas toutefois être regardées comme absolues, et les diverses époques fixées ainsi d'après nos études personelles, présentent certains écarts suivant les sujets, les constitutions et même les maladies. Ces variations, qui ne peuvent être que de quelques jours pour la première colonne relative à l'apparition du chapeau de dentine, s'accentuent davantage à la seconde colonne, mais surtout à la troisième ; c'est-à-dire, que l'époque assignée à telle ou telle hauteur d'un chapeau de dentine peut varier de quelques semaines et même de quelques mois. Ce sont là en tout cas des données moyennes.

Quoi qu'il en soit, les conséquences de ces indications à l'égard de l'érosion se résument de la manière suivante :

1° Si une intervention morbide porte son action sur un follicule au début de la formation du chapeau de dentine, l'érosion occupera soit le bord libre s'il s'agit d'une incisive ou d'une canine, soit la surface triturante s'il s'agit d'une molaire. C'est ainsi que nous pouvons montrer un fait d'érosion exclusive à la face triturante des premières molaires dont le chapeau apparaît au sixième mois de la vie fœtale. Cette érosion fut due à des accidents de la grossesse auxquels la mère a d'ailleurs succombé.—(Observation 21 du tableau analytique, No. 3.)

2° Si cette même influence morbide intervient plus tardivement, l'érosion aura pour siège un point plus ou moins distant de ce bord libre, mais toujours précédé d'une région saine dont l'étendue est proportionelle à la durée même de la période de santé qui a précédé les accidents.

Quelques mots maintenant sur la nature exacte de cette lésion et sur ses formes diverses. Nous serons bref, car les auteurs anciens et modernes sont d'accord sur cette question :

L'érosion dentaire est caractérisée essentiellement tantôt par un simple trouble dans la formation simultanée de l'ivoire et de l'émail, tantôt par une suspension complète de cette formation. S'il y a trouble léger sous l'influence d'une cause faible et fugace, l'érosion se traduira par un sillon simple peu profond, parfois même difficile à reconnaître. Si le trouble est intense ou prolongé ainsi que la cause qui l'a provoqué, l'érosion sera étendue en surface ou en profondeur, formant quelquefois une véritable zone annulaire plus ou moins large avec absence congénitale d'émail et altération pénétrante dans l'ivoire. Enfin, si les crises morbides ont été fréquemment répétées et très-rapprochées dans leurs apparitions, une surface très-étendue ou la totalité de la couronne pourra être frappée des altérations caractéristiques.

## TABLEAU No. 1.

*Etat du Chapeau de Dentine pour chacun des Follicules dentaires au Premier Age chez l'Homme.*

| Désignation des Dents. | Apparition du Chapeau de Dentine. | Dimension en Hauteur du Chapeau de Dentine à la Naissance. | Dimension en Hauteur du Chapeau de Dentine au 6e Mois. |
|---|---|---|---|
| **A.—Dentition Temporaire.** | | | |
| Incisives centrales inférieure | 16e semaine de la vie embryonnaire | mm. 3 5 | La couronne est entièrement formée et prête à l'éruption. |
| Incisives centrales supérieure | | | |
| Incisives latérales inférieure | | | |
| Incisives latérales supérieure | | | |
| Canines inférieure | | 3 2 | 6mm. |
| Canines supérieure | | | |
| Premières molaires inférieure | 17e semaine de la vie embryonnaire | 3 0 | 7mm. |
| Premières molaires supérieure | | | |
| Deuxièmes molaires inférieure | | | |
| Deuxièmes molaires supérieure | | | |
| **B.—Dentition Permanente.** | | | |
| Incisives centrales inférieure | 1er mois de la naissance | ... | 2mm. |
| Incisives centrales supérieure | | | |
| Incisives latérales inférieure | | | |
| Incisives latérales supérieure | | | |
| Canines inférieure | Du 3e au 4e mois de la naissance | ... | 1mm. |
| Canines supérieure | | | |
| Premières prémolaires inférieure | Du 5e au 6e mois de la naissance | ... | ... |
| Premières prémolaires supérieure | | | |
| Deuxièmes prémolaires inférieure | | | |
| Deuxièmes prémolaires supérieure | | | |
| Premières molaires inférieure | 6e mois de la vie fœtale | 2mm. | 6mm. |
| Premières molaires supérieure | | | |
| Deuxièmes molaires inférieure | 3e année | ... | ... |
| Deuxièmes molaires supérieure | | | |
| Troisièmes molaires inférieure | 12e année | ... | ... |
| Troisièmes molaires supérieure | | | |

C'est ainsi que se classent les formes diverses d'érosion de la manière suivante :

1° Erosion *en échancrure ou en coup d'ongle* du bord libre (incisives).

2° Erosion en rugosités ou mamelons de la face triturante (molaires).

3° Erosion *en sillon simple ou pointillé,* unique ou multiple. L'existence de rainures multiples superposées a été désignée par J. Tomes, sous le nom d'érosion *en étages ou en escalier.*

4° Erosion en nappe avec absence congénitale d'émail et état spongieux de

l'ivoire. Elle indique de la part de l'intervention morbide une durée proportionelle à l'étendue de la lésion.

5° Enfin, érosion totale de la couronne de certaines dents, avec durée correspondante de la cause productrice, altération désignée par le même auteur sous le nom de dents en *gâteau de miel.*

Cette dernière forme, par l'intensité et la généralisation de son état, nous paraîtrait même devoir être distraite de la classe des *érosions* proprement dites. Ici, en effet, il n'y a plus de sillons ou d'échancrures symétriques et parallèles ; la couronne est tout entière désorganisée et elle disparaît d'ordinaire très-vite par suite de carie consécutive. La durée de la cause morbide a dû être considérable, et nous pourrions citer maintes observations dans lesquelles, en dehors de toute diathèse syphilitique ou autre, un état maladif de la première enfance a pu causer une telle lésion.

Voici, par exemple, une observation dans laquelle les premières molaires et les incisives des deux mâchoires ont présenté la forme *en gâteau de miel* et une destruction ultérieure complète, consécutivement à une entérite chronique qui a duré depuis les premiers mois de la vie jusqu'à la deuxième année.

Nous en donnerons simplement le sommaire :

*Observation :* Homme de 23 ans ; entérite chronique grave, ayant débuté au 2° mois et ayant persisté jusque la deuxième année, pendant tout la durée et à la suite d'un allaitement artificiel—aucun soupçon de syphilis, aucune trace de rachitisme; pas d'accidents scrofuleux, pas d'attaques éclamptiques. Au moment de l'éruption de la seconde dentition, les incisives, les canines, et les premières molaires apparurent dans un état de désorganisation complète, dans la forme dit *gateau de miel.* Elles furent très rapidement détruites par la carie, plusieurs prémolaires présentent aussi des caries moins généralisées et qui n'ont pas être mises en traitement. Les deuxièmes molaires, dent d'évolution, répondent à une epoque où la santé de l'enfant s'était rétablie, ne presentent aucune lésion.

Tels sont les aspects divers de l'érosion.

Notre savant ami, M. le professeur Parrot, a tenté de modifier cette classification et en a proposé une autre dans les termes suivants :

1° *Atrophie cuspidienne* portant sur la partie la plus saillante de la dent : elle serait très-commune aux premières molaires permanentes, ce qui impliquerait une origine fœtale, aux prémolaires de la seconde dentition, et plus atténuée à celle de la première dentition.

2° *Atrophie cupuliforme,* avec absence de l'émail et mise à nu de l'ivoire, c'est la forme en nappe associée ou non à la précédente.

3° *Atrophie sulciforme ou en sillon,* c'est la forme en *rainure* simple ou multiple.

4° *Atrophie en hache.* Celle-ci, de l'aveu de M. Parrot, ne serait pas congénitale, mais secondaire et pathologique. C'est de toute évidence une carie consécutive. Elle ne saurait donc, à aucun titre, entrer dans le cadre de l'érosion qui est avant tout une lésion congénitale, une anomalie de nutrition intra-folliculaire.

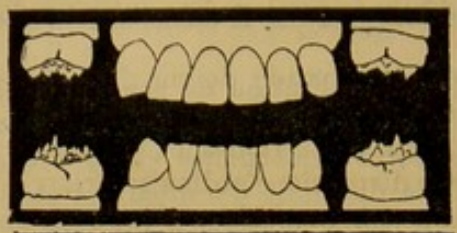
5° *Atrophie Hutchinsonienne.* C'est la variété en coup d'ongle, échancrure du bord libre.

Nous n'entrerons pas dans la discussion de cette nouvelle classification, et nous demandons la permission à M. Parrot de conserver l'ancienne qui nous paraît plus simple et plus physiologique.

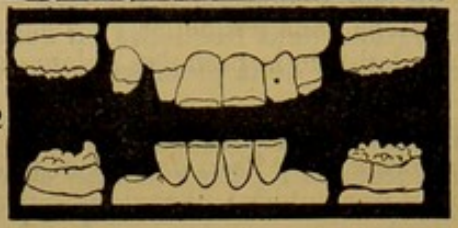
Voici au reste un tableau représentant les types principaux qu'affecte l'érosion. Nous avons fait représenter dix formes non seulement au point de vue de leurs caractères, mais aussi à l'égard des dents affectées et des point qu'elles occupent. Les exemples pris de nature sont d'ailleurs empruntés à la série d'observations que nous publions d'autre part dans un autre tableau d'ensemble.

## TABLEAU No. 2.

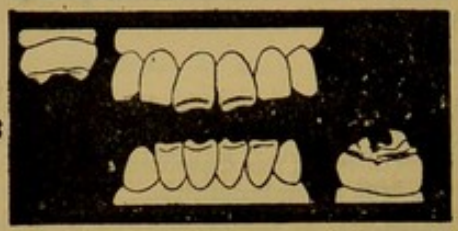
*Principaux types d'érosion.*



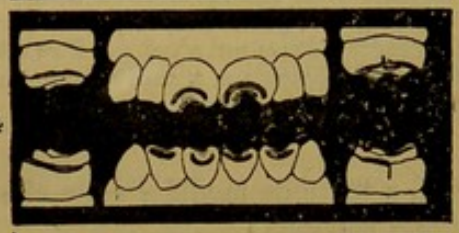
1 Erosion en *mamelons* et rugosité exclusive aux premières molaires permanentes (face triturante). (Observation 21 du tableau analytique.)



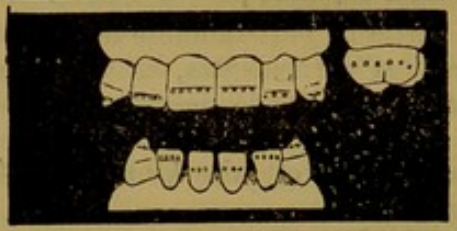
2 Erosion en *mamelons* des molaires en *sillon simple* près du bord libre des incisives et vers le milieu des canines *temporaires* supérieures persistantes. (Observation 22.)



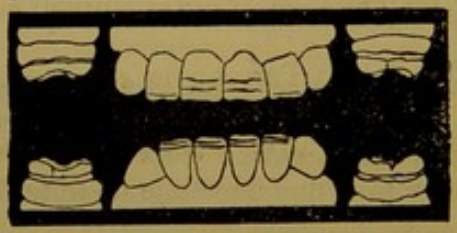
3 Erosion en *mamelons* des molaires et en *sillon simple* des incisives. (Observation 23.)



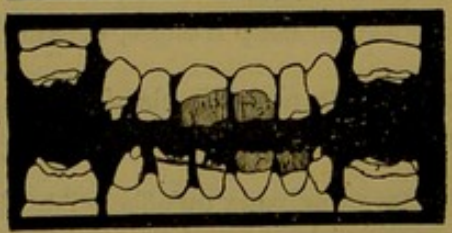
4 Erosion en *sillon* des molaires et en *coup d'ongle* des incisives. (Observation 24.)



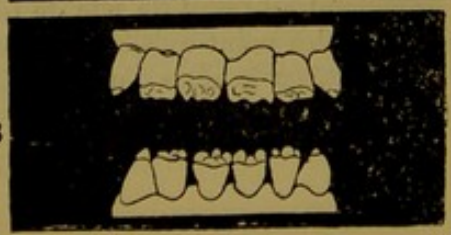
5 Erosion en *sillon pointillé* des incisives et des molaires. (Observation 25.)



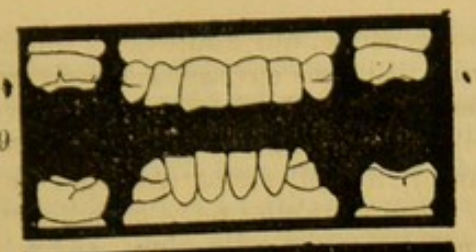
6 Erosion en *double sillon* aux incisives et aux molaires. (Observation 26.)



7 Erosion en *nappe* aux incisives, en *sillon* aux canines et aux molaires. (Observation 27.)



8 Erosion en *nappe* avec végétations mamelonnées. (Observation 28.)



9

Erosion en *sillon simple* portant exclusivement sur la partie moyenne des canines permanentes. (Observation 29.)

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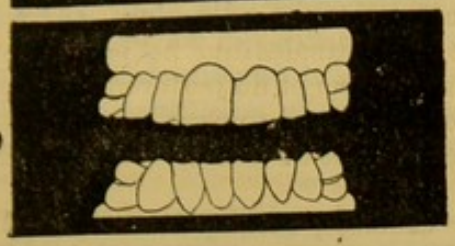

10

Erosion en *sillon simple* portant exclusivement sur la partie moyenne des premières prémolaires. (Observation 30.)

Dans tous les cas, ce qui ressort évidemment de ces considérations, c'est qu'il existe d'abord un rapport étroit entre les trois termes suivants :—

1° Cause morbide produisant la lésion ;

2° Le niveau anatomique de la lésion ;

3° Son étendue.

Les trois termes peuvent ainsi être considérés comme constituant une équation pathologique.

Une autre forme qu'il importe de déterminer ici : c'est *l'identité de nature* de ces différentes formes quels que soient d'ailleurs leur niveau et leur étendue.

L'érosion est une, et les diversités d'aspect résultent simplement de l'époque d'apparition de la cause productrice, de la durée d'intervention de celle-ci, et de l'état du chapeau de dentine au moment de l'invasion des accidents. Les variétés en sillon simple ou multiple s'expliquent ainsi par une ou plusieurs apparitions de la cause ; la variété en nappe, est due à la persistance prolongée de son action, et quant à la forme spéciale, en coup d'ongle ou Hutchinsonnienne, suivant M. Parrot elle semble due à un mécanisme particulier qui est le suivant :

Un trouble de formation du bord libre d'une incisive a pour résultat d'amener au dehors une couronne dont le bord est aminci et très friable. Cette partie désorganisée est limitée par un bourrelet soit rectiligne soit demi-circulaire, et au delà duquel la constitution anatomique de l'organe redevient normale ; or, c'est la fracture de cette partie amincie pendant les premiers temps qui suivent l'éruption qui donne dans la suite l'aspect spécial qu'à décrit M. Hutchinson ; l'érosion en coup d'ongle n'a pas d'autre origine.

Ces faits étant une fois fixés dans leurs rapports réciproques, et reliés ainsi d'un façon systématique, arrivons de suite au but spécial de cette étude, la recherche étiologique de l'érosion.

Or, les opinions émises par les auteurs anciens et modernes peuvent se grouper sous trois chefs :

1° L'opinion qui attribue la production de l'érosion aux *affections quelconques de l'enfance.*

2° Celle qui considère l'érosion comme signe constant de *syphilis héréditaire.*

3° La théorie qui rattache l'érosion à *l'éclampsie infantile.*

Nous allons les passer rapidement en revue.

L'explication qui attribue l'érosion aux affections quelconques de la première enfance est celle des auteurs anciens, et l'on peut dire celle de tous les temps. Ambroise Paré la mentionne ; de même après lui, Bunon, Fauchard, &c. ; plus récemment, Duval, Oudet.

Aujourd'hui encore, mais dans une certaine mesure, elle est soutenue par Tomes en Angleterre et par Broca en France.

Nous pensons qu'une affirmation aussi générale, qui s'adresse à toutes les maladies de l'enfance, ne saurait se soutenir. Si nous envisageons par exemple une grande classe des affections de la première enfance, les fièvres éruptives, il est aisé de se convaincre, par un nombre illimité de faits, qu'elles n'apparaissent guère dans la première ou la deuxième année, et qu'elles sont d'ailleurs impuissantes à produire l'érosion caractéristique, à moins qu'elles ne se compliquent de troubles graves du système nerveux dont nous parlerons plus loin. Il en doit être de même des affections catarrhales ou intestinales qui ne présentent que bien rarement ces deux caractères d'invasion brusque et de perturbation profonde de la nutrition, qui sont les circonstances essentielles du mécanisme de l'érosion. M. Parrot a d'ailleurs developpé lui-même ce point de vue et nous sommes ici tout-à-fait d'accord avec lui.

Quant aux diathèses héréditaires ou acquises, rachitisme et scrofule par exemple, nous leur avons reconnu depuis longtemps une influence considérable sur l'organisation des dents.* Mais cette influence, qui par sa permanence même doit porter sur la totalité de l'organe, se traduit par des altérations de forme générale et de structure intime qui n'ont aucun rapport avec l'érosion. Nous savons, il est vrai, que M. Parrot considère la scrofule et le rachitisme comme des manifestations syphilitiques, mais c'est là une opinion très-personnelle, contre laquelle s'élèvent à la fois le sentiment général des pathologistes et des faits nombreux.

Quoi qu'il en soit, il est certain que les dents des rachitiques, par exemple, sont petites, atrophiées, difformes même, et frappées de défectuosités générales de structure et de constitution chimique. Il y a là sous nos yeux certaines pièces qu'on serait en droit de classer dans cette catégorie, si nous n'avions retrouvé dans les antécédents des troubles nerveux graves, dont la trace est marquée par l'érosion. De la sorte on reconnaît chez le même sujet le vestige du rachitisme et le témoin de l'éclampsie, c'est-a-dire la preuve de la coexistence des deux affections.

La seconde opinion, celle qui attribue l'érosion dentaire à la syphilis héréditaire, est née en Angleterre, d'un travail de M. Hutchinson, publié en 1863,† et elle est défendue en France avec une grande autorité par le professeur Parrot.‡

Cette doctrine n'a point encore de partisans, du moins en France, à l'exception toutefois de M. Lannelongue qui semble disposé à s'y rallier.

En ce qui concerne M. Hutchinson, son travail est, comme on sait, consacré à l'étude de la kératite interstitielle dans ses rapports avec la syphilis héréditaire, et ce n'est qu'un peu incidemment qu'il mentionne chez les mêmes sujets une forme particulière d'érosion, dont il attribuerait en réalité la cause à la stomatite qui accompagne le traitement mercuriel.

M. Hutchinson publie ainsi une série de 102 faits de kératite syphilitique, parmi lesquels nous relevons les points suivants :—

Dans 63 cas, l'auteur mentionne la coexistence de l'échancrure du bord libre des incisives, mais, chose suprenante, sans signaler l'altération des molaires qui est, comme on sait, constante. Dans 39 cas, les conditions des dents n'ont pas été notées ou ont été trouvées normales.

Sur la question des affections concomitantes on reconnaît dans les observations du mémoire des troubles divers se rattachant à la syphilis, mais aucune

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* "Traité des anomalies de l'appareil dentaire," 1877, p. 256.

† "A Clinical Memoir on Certain Diseases of the Eye and Ear consequent on Inherited Syphilis."

‡ "De la Syphilis dentaire, Communication faite au Congrès de l'Association française de Reims, 1880." Clinique de l'Hospice des Enfants-Assistés, *Gaz. des Hôpit.*, 1881, Nos. 74, 78, 80, et 82.

mention de la coexistence ou de l'absence de l'éclampsie. Deux sujets seulement sont dits *épileptiques*.

Tels sont les points principaux que nous avons relévés dans le mémoire de M. Hutchinson, mais il nous semble que les idées de l'auteur se sont notablement modifiées depuis lors et qu'il est disposé à regarder certaines formes d'érosion comme absolument caractéristique de la syphilis.

Pendant notre séjour à Londres, M. Hutchinson a bien voulu nous engager à l'accompagner au *London Hospital* on il désirait nous montrer quelques malades affectés des lésions dont il s'agit, et concurremment avec la kératite interstitielle et la syphilis héréditaire. Cette visite a été pour nous d'un très-grand intérêt, et nous ne saurions trop l'en remercier. Elle nous a permis en effet d'observer plusieurs sujets offrant une certaine forme d'altérations dentaires et particulièrement cette forme que nous avons indiquée comme se rapprochant beaucoup plus des lésions d'ensemble dues aux diathèses que de l'érosion proprement dite. C'étaient des dents mal conformées, des incisives ayant en quelque sorte l'aspect de moignous difformes, de cônes tronqués, de véritables monstruosités morphologiques.

Or ces sujets portaient d'autre part des traces manifestes de rachitisme ancien ou actuel, de scrofule, de syphilis même, et ils ne pouvaient en outre fournir sur leur première enfance aucun témoignage au sujet de l'éclampsie.

Nous n'insisterons pas sur cette discussion en ce que concerne M. Hutchinson, et nous avons hâte d'arriver à M. Parrot qui formule très nettement son opinion en donnant à l'érosion des dents le nom de *syphilis dentaire*.

Les arguments de M. Parrot reposent sur la coexistence avec l'érosion dentaire de certaines lésions osseuses du crâne, des mâchoires ou d'un autre point du squelette ; sur des macules cutanées, traces d'ulcérations éteintes ; sur des ostéophytes, et enfin sur le rapport entre le siège de l'érosion et l'âge d'élection des accidents syphilitiques qui évoluent, suivant lui, depuis le sixième mois de la vie fœtale, jusqu'à la quatrième année.

Or voici nos objections à cette théorie :

1° M. Parrot ne nous a fourni jusqu'à présent aucune observation d'érosion caractéristique avec altérations osseuses spécifiques, accidents cutanés indéniables, et *en l'absence de toute autre intervention morbide ;* en un mot, un diagnostic absolu de syphilis héréditaire. Tout au contraire, M. Parrot, entraîné par l'ardeur de sa conviction, arrive à affirmer *à priori* la syphilis héréditaire sur la seule existence de l'érosion. Une mâchoire d'adolescent, trouvée dans un cimetière gallo-romain, porte deux sillons dentaires superposés ; M. Parrot conclut à la syphilis préhistorique. Un sujet vivant présente la même lésion, il le déclare syphilitique bien qu'on ne puisse saisir dans les antécédents aucune manifestation de cette diathèse. Ce sont là des affirmations dont la gravité n'échappera à personne.

2° Nous pourrions apporter un nombre considérable de faits, dans lesquels des sujets notoirement syphilitiques héréditaires ne présentent aucune trace d'érosion. Nous invoquerons à cet égard le témoignage de M. Alfred Fournier, qui, dans une longue pratique, n'a jamais saisi cette relation. Mentionnons en outre une observation personelle que nous avons faite récemment dans les populations kabyles d'Algérie, chez lesquelles la syphilis serait endémique et héréditaire depuis un temps indéfini, tandis que l'érosion des dents est extrêmement rare. Enfin des sujets affectés d'érosion très-manifeste ont été trouvés porteurs de chancres indurés, alors que la majorité des syphiliographes se refusent à admettre qu'un sujet affecté héréditairement de syphilis puisse la contracter à nouveau.

3° Un grand nombre de sujets, chez lesquels l'observation la plus minutieuse n'a pu retrouver aucune trace de syphilis infantile, présentent l'érosion à ses divers degrés.

4° M. Parrot affirme l'existence de la syphilis aux temps préhistoriques.

Assurément ce n'est pas nous qui nous refuserons à admettre l'ancienneté de la syphilis ; mais sur quels faits raisonne M. Parrot ? Est-ce sur cette mâchoire gallo-romaine déjà signalée tout à l'heure et dont nous donnons le dessin ? (fig. 10).

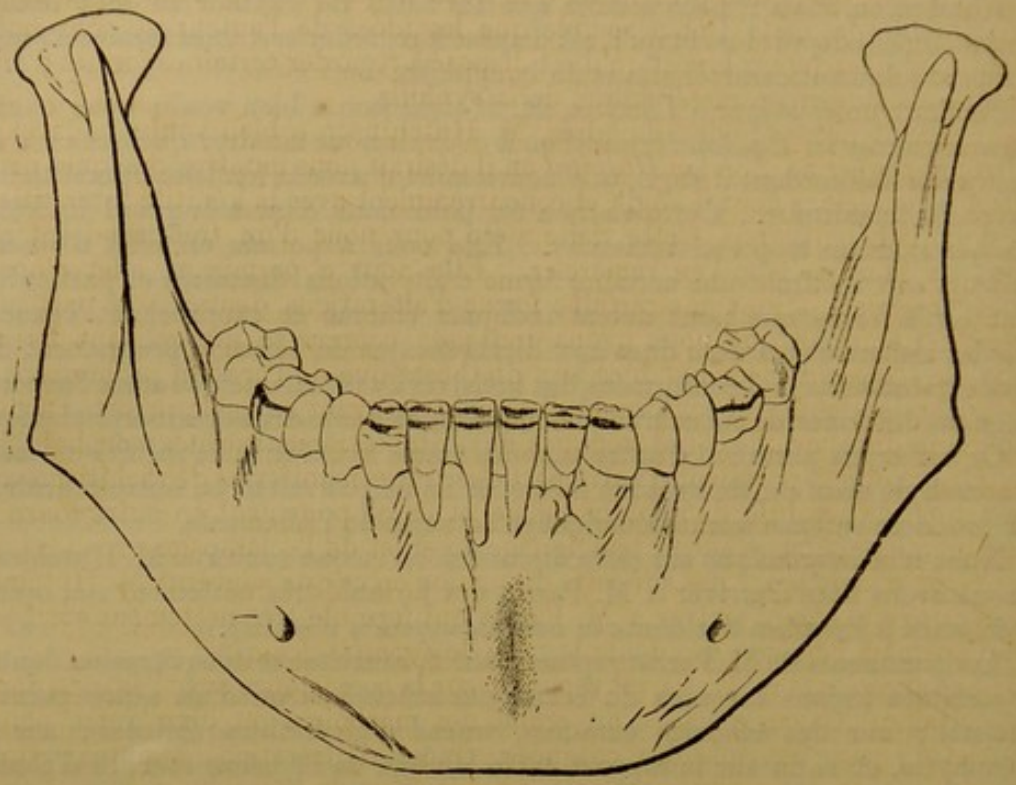

Fig. 10.—Mâchoire d'un jeune Franc de l'époque mérovingienne, et trouvée dans les cimetière de Breny (Aisne) par M. Moreau (Musée de la Société d'Anthropologie de Paris).

Est-ce sur ces pièces trouvée, dans les cavernes de l'époque de la pierre polie et représentant des dents frappée, d'érosion en étages? (fig. 11).

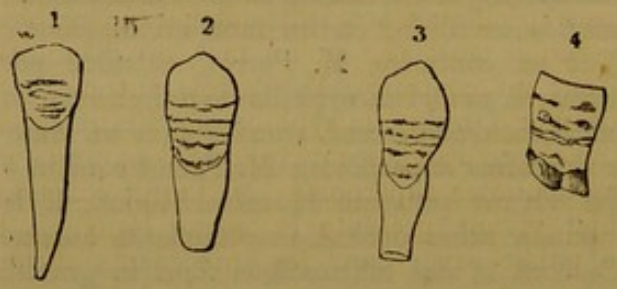


Fig. 11.—Quatre dents frappée d'érosion en sillons multiples superposés et trouvées dans les dolmens de la Lozère—collection de M. le Dr. Prunières.

Elles ont en effet les sillons caractéristiques. Mais ces pièces sont muettes sur le fait de la syphilis, et nous serions tout aussi bien fondé à affirmer ici l'existence de l'éclampsie préhistorique. C'était d'ailleurs, comme on sait, l'opinion de Broca, quand il émit cette hypothèse que la trépanation crânienne, qui s'adressait aux enfants de l'âge de la pierre polie, avait pour objectif la guérison des affections convulsives. Or la présence des dents frappées d'érosion trouvées dans les mêmes gisements ne vient-elle pas singulièrement confirmer cette hypothèse ?

5° Enfin le dernier argument que nous voulons opposer à M. Parrot, argument péremptoire, c'est l'existence de la lésion qu'il qualifie de syphilitique ailleurs que chez l'homme. Voici, en effet, une mâchoire de bœuf, dont les deux pinces centrales sont frappées d'érosion symétrique.

Nous réprésentons (fig. 12) l'une des incisives qui présentait de la manière la plus manifeste une érosion en nappe occupant le quart de la hauteur de la couronne et limité par un bourrelet d'émail.

Nous n'avons point, il est vrai, sur le premier âge de cet animal de renseignement pouvant prouver qu'il à été atteint de maladie convulsive ou autre. Nous nous bornous à conclure que cette lésion n'est pas syphilitique en ajoutant toutefois que certains états convulsifs ont été observés pendant le premier âge chez quelques herbivores, le mouton par exemple.

Tel est l'ensemble de raisons sous lesquelles tombe, suivant nous, la théorie de la syphilis dentaire.

Abordons maintenant la troisième et dernière partie de cette argumentation, c'est-à-dire la démonstration que l'érosion dentaire, dans ses formes d'*échancrure*, de *sillon*, de *nappe*, &c., est le signe rétrospectif constant de l'éclampsie infantile.

Et d'abord voici un tableau dans lequel nous avons réuni quarante observations d'érosion dentaire dans leurs rapports étroits et exclusifs avec l'éclampsie. Ce tableau présente en colonnes parallèles :

1° La lésion dentaire, son lieu d'élection, le niveau des dents affectées, soit de la dentition temporaire, soit de la permanente ;

2° Dans un autre colonne, l'âge des attaques éclamptiques, leur durée, leur intensité ;

3° Dans la dernière colonne, l'étude des antécédents et des maladies intercurrentes.

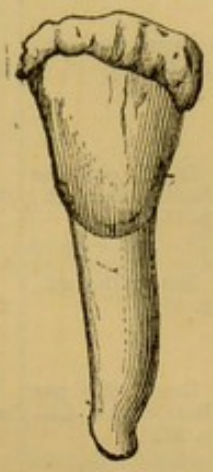

Fig. 12.—L'une des deux pinces centrales d'un bœuf Durham, âge de 4 ans.

Qu'on veuille bien étudier une à une ces quarante observations saisissantes par leur netteté et leur précision. Nous aurions pu les multiplier à l'infini, mais il nous semble qu'elles doivent suffire à la démonstration mathématique de l'équation pathologique que nous avons formulée tout à l'heure.

Ce rapport rigoureux entre l'éclampsie infantile et l'érosion est d'ailleurs pour nous un fait très-anciennement démontré. Sans parler de nos recherches personnelles antérieures, nous pouvons rappeler plusieurs monographies qui défendent la même opinion : telles sont les thèses des docteurs Quinet (de Bruxelles), Castanié et Rattier (de Paris), qui renferment des faits conformes.* Broca, ainsi qu'on sait, s'est également rallié à cette théorie.

Si nous interrogeons les auteurs étrangers, nous trouvons que Arlt, dans ses recherches sur la cataracte zonulaire et l'érosion des dents, invoque dans leur production les attaques éclamptiques ; de même Horner et Davidson, bien que moins affirmatifs. Enfin, M. Nicati n'hésite pas à rattacher à cette même cause à la fois l'érosion de la dent et la cataracte zonulaire qu'on pourrait presque nommer *l'érosion du cristallin.*

Cette opinion, appuyée aujourd'hui sur des faits scientifiquement observés, se retrouve d'ailleurs empirique et en quelque sorte banale dans tous les temps. Je fais appel ici aux médecins qui veulent bien m'écouter. Beaucoup d'entre eux, en présence d'une érosion dentaire bien constatée, n'ont ils pas posé cette affirmation : " Ce sujet a eu des convulsions dans son enfance ? " Et la mère de répondre invariablement : " Cela est vrai."

Aujourd'hui, nous ne nous bornons plus, en présence de la lésion dentaire, à affirmer l'éclampsie ; nous allons plus loin, et du niveau de cette échancrure ou de ce sillon, nous remontons à la date des crises, à leur durée, à leur intensité. Le procédé est fort simple : il est tout entier formulé dans ce tableau (No. 1).

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* Voy. Quinet, " Apropos des Dents syphilitiques," *Bulletin de l'Académie Royale de Méd. de Belgique*, t. xiii. 3e série, No. 1, 1879. Castanié, " De l'Erosion ou des Altérations des Dents permanentes à la suite des Maladies de l'Enfance," Thèse de Paris, 1874. Rattier, " Contributions à l'Etude de l'Erosion dentaire," Thèse de Paris, 1879.

# TABLEAU No. 3.

*Résumé analytique de Quarante Observations d'Erosion dentaire due à l'Eclampsie infantile.*

| Nos. d'Ordre. | Noms des Auteurs. | Sexe et Age des Sujets. | Forme et Niveau de l'Erosion. | Date et Durée des Attaques Eclamptiques. | Observations. |
|---|---|---|---|---|---|
| 1 | Broca, cité par Castanié (loc. cit., p. 49). | H 17 ans. | Sillon circulaire aux trois quarts de la couronne des incisives, faible aux canines. | Attaques d'éclampsie répétées à la 2e année et durant environ 2 mois. | ... ... ... ... |
| 2 | Broca (loc. cit., p. 50). | H 20 ans. | Sillon circulaire d'un millimètre au milieu de la couronne des incisives, au tiers des canines. | Crises éclamptiques ayant duré 3 mois à l'âge d'un an et demi. | L'éclampsie à laissé après elle une contracture des membres supérieurs; rougeole à 7 ans; variole à 10 ans. |
| 3 | David, cité par Rattier (loc. cit., p. 41). | H 28 ans. | Sillon courbe à 2 millimètres du bord libre des incisives; sillon droit aux premières molaires. | A l'âge de six semaines, accès convulsifs durant 8 jours et compliquant une bronchite. | Aucun soupçon de syphilis. |
| 4 | Rattier (loc. cit., p. 40). | H 22 ans. | Echancrure du bord libre des incisives; érosion profonde des premières molaires. | Du 12e au 13e mois, série d'attaques éclamptiques. | A 6 ans, rougeole légère. |
| 5 | Broca, cité par Rattier (loc. cit., p. 38). | H adulte. | Erosion en nappe occupant le tiers de la hauteur des incisives centrales, le quart des latérales, le sommet des canines et la moitié des premières molaires. | Crises convulsives fréquentes et violentes pendant les 18 premiers mois de la vie. | Pas trace de rachitisme; les convulsions ont laissé à leur suite un tic de la face. |
| 6 | Quinet (loc. cit., p. 52). | H 30 ans. | Sillon simple des incisives centrales à 4 millimètres du bord libre, à 2 millimètres aux latérales, près du sommet aux canines; grosses molaires profondément cariées. | Vers l'âge de 3 ans, crises éclamptiques violentes se répétant pendant 3 jours. | Aucune autre affection infantile quelconque. |
| 7 | Quinet (loc. cit., p. 54). | H 18 ans. | Erosion en nappe des incisives centrales près du bord libre, moindre aux latérales et aux canines, très marquée aux premières molaires. | Série d'attaques convulsives du 10e au 15e mois. | Strabisme convergent et surdité consécutifs à l'éclampsie; aucun soupçon de syphilis. |
| 8 | Quinet (loc. cit., p. 57). | H 40 ans. | Sillon pointillé aux incisives centrales, moins marqué aux latérales, altération des sommets des canines; très-marquée aux premières molaires. | Attaque d'éclampsie grave au 14e mois. | ... ... ... ... |
| 9 | Magitot (inédite). | H 40 ans. | Erosion en coup d'ongle des incisives centrales supérieures, des 4 inférieures; les 4 premières molaires manquent. | Eclampsie grave dans le 2e mois de la vie. | Aucun soupçon de syphilis; le sujet étant médecin s'est prêté à l'examen le plus complet. |
| 10 | Combe (inédite). | H 24 ans. | Erosion en sillon au voisinage du bord libre des incisives; rien aux canines; altération profonde des premières molaires. | Série d'attaques convulsives dans le premier mois de la vie. | Aucun soupçon de syphilis; le sujet à un frère aîné et une sœur cadette très-bien portants, sans érosion. |
| 11 | Castanié (loc. cit., p. 50). | F 20 ans. | Erosion en sillon à 2 millimètres du bord libre aux incisives centrales, à 1 millimètre aux latérales et aux canines; au voisinage du collet aux premières molaires. | Série d'attaques éclamptiques occupant une période de 6 mois, du 18e au 34e mois. | Aucun soupçon de syphilis. |
| 12 | Castanié (loc. cit., p. 51). | F 16 ans. | Erosion en échancrure du bord libre des incisives centrales supérieures, des 4 incisives inférieures; rien aux latérales supérieures; érosion très-marquée aux premières molaires. | Une seule série d'attaques éclamptiques au 9e mois et mettant pendant plusieurs jours la vie en danger. | Aucune autre affection infantile quelconque jusqu'à l'âge de 9 ans. |
| 13 | Rattier (loc. cit., p. 39). | F 17 ans. | Erosion en dentelures du bord libre, et sillon de la couronne aux incisives des deux mâchoires; sillon circulaire aux premières molaires. | Au 6e mois, attaque d'éclampsie grave; du 12e au 18e mois, série d'attaques répétées et moins graves. | ... ... ... ... |

TABLEAU No. 3—*continué.*

| Nos d'Ordre. | Noms des Auteurs. | Sexe et Age des Sujets. | Forme et Niveau de l'Erosion. | Date et Durée des Attaques Eclamptiques. | Observations. |
|---|---|---|---|---|---|
| 14 | Magitot (inédite). | F 15 ans. | Erosion en échancrure du bord libre des incisives supérieures centrales, des 4 inférieures; rien aux latérales supérieures; érosion profonde des molaires au voisinage de la face triturante. | Série d'attaques convulsives au 3e mois et ayant duré 24 heures. | Aucune autre affection infantile quelconque jusqu'à l'âge de 7 ans. |
| 15 | Magitot, traité des anomalies, p. 270. | F 9 ans. | Aucune altération des dents temporaires restantes; érosion en nappe avec amincissement extrême au voisinage du bord libre des incisives; érosion très-marquée aux molaires. | A un an, série d'attaques éclamptiques graves. | Aucune autre affection infantile quelconque. |
| 16 | Magitot, eod. loc., p. 271. | H 14 ans. | Erosion en sillon simple de 1 millimètre de hauteur au milieu de la couronne des incisives centrales, plus rapproché du bord libre aux latérales, près du sommet aux canines; sillon aux molaires. | Au 15e mois, série d'attaques éclamptiques durant de 12 à 15 jours. | Aucune autre affection infantile quelconque. |
| 17 | Magitot, eod. loc., p. 270. | H 20 ans. | Sillon très-fin et de niveau identique au milieu de la couronne des 4 canines; les deuxièmes molaires sont détruites par carie; les autres dents sont normales. | A 8 ans, méningite grave avec accès convulsifs pendant 15 jours environ. | Aucune affection infantile quelconque avant cette époque. |
| 18 | Magitot (inédite). | H 13 ans. | Sillon simple au milieu des incisives centrales, au tiers des latérales, au quart des canines; sillon aux premières molaires. | Au 18e mois, série d'attaques éclamptiques durant 1 mois. | Aucun soupçon de syphilis; aucune autre affection infantile jusqu'à 12 ans, époque d'une rougeole. |
| 19 | ,, | H 18 ans. | Sillon pointillé vers le tiers des incisives centrales, au quart des latérales, près du sommet, aux canines, au milieu des premières molaires. | Trois séries d'attaques éclamptiques durant plusieurs jours: la première au 7e mois, et les autres à une semaine d'intervalle. | Aucun soupçon de syphilis; famille nombreuse très-bien portante; frères et sœurs plus jeunes, n'yant jamais été malades et ne portant pas trace d'érosion. |
| 20 | ,, | F 20 ans. | Sillon large avec bourrelet très-saillant au milieu de la couronne des incisives, moins élevé aux latérales et aux canines; les premières molaires manquent. | Série d'accès convulsifs du 8e au 20e mois. | ... ... ... ... |
| 21 | ,, | H 24 ans. | Erosion portant exclusivement sur la surface triturante des premières molaires qui sont couvertes d'aspérités aiguës. (V. tableau no 2, fig. 1.) | Accidents graves de la mère pendant les derniers temps de la grossesse; accouchement au 8e mois, suivi de mort, quelques heures après la délivrance (éclampsie?) | L'enquête au sujet de la mère n'a pu été poussée plus loin. Pour le sujet, aucune affection infantile quelconque; aucun soupçon de syphilis héréditaire, ainsi qu'il resulte de l'examen très-approfondi des antécédents; pas de traces de cicatrices cutanées sur aucun point du corps. |
| 22 | ,, | H 9 ans. | L'érosion a le caractère d'un sillon simple siégeant à la partie moyenne des canines supérieures temporaires, persistantes, d'une échancrure sur le bord libre des incisives centrales supérieures et sur les 4 inférieures. Aux molaires, elle occupe la face triturante qui est rugueuse et mamelonnée. (V. tableau no 2, fig. 2.) | Une première série d'attaques éclamptiques s'est produite au début du 2e mois et a duré trois jours avec dysurie et même rétention complète nécessitant le cathétérisme. Cette première attaque a été suivie de cinq autres se produisant au début de chacun des mois suivants avec le même caractère et la même durée. | Aucune affection quelconque jusqu'à l'âge de 10 ans; parents très-bien portants, sans aucun soupçon de syphilis; pas de fausse couche de la mère; le sujet a un frère cadet âge de 7 ans qui n'a jamais été malade et n'a aucune érosion sur les premières molaires qui sont en place, ni sur les incisives centrales supérieures qui ont achevé leur sortie. |

## Tableau No. 3—*continué.*

| Nos. d'Ordre. | Noms des Auteurs. | Sexe et Age des Sujets. | Forme et Niveau de l'Erosion. | Date et Durée des Attaques Eclamptiques. | Observations. |
|---|---|---|---|---|---|
| 23 | Magitot. | F 18 ans. | Erosion en échancrure du bord libre des incisives centrales supérieures et des 4 inférieures; sillon simple des premières molaires, immédiatement au-dessous de la surface triturante; deux de ces molaires sont détruites par la carie consécutive à l'érosion. (V. tableau nº 2, fig. 3.) | Au 3e mois, séries d'attaques éclamptiques ayant duré 24 heures avec menaces d'asphyxie; état grave; imminence de mort. | Aucune affection intercurrente jusqu'à l'âge de 7 ans; aucun soupçon de syphilis. |
| 24 | ,, | F 15 ans. | Exagération de l'état précédent; érosion en échancrure profonde du bord libre des incisives centrales supérieures, des 4 inférieures, très-marquée aux centrales, moindre aux latérales; rien aux incisives latérales supérieures ni aux canines des deux mâchoires. (V. tableau nº 2, fig. 4.) | Au 6e mois de la vie, série d'attaques convulsives ayant duré 24 heures. | Aucune affection intercurrente de la première enfance, jusqu'à l'âge de 7 ans, époque d'une rougeole bénigne; aucun soupçon de syphilis. |
| 25 | ,, | F 18 ans. | Erosion en sillon pointillé au tiers supérieur des incisives centrales, au quart inférieur des latérales, au cinquième des canines et à la moitié de la première molaire gauche; même niveau aux dents antéro-inférieures; les 3 autres molaires sont détruites par la carie consécutive à l'érosion. (V. tableau nº 2, fig. 5.) | Attaques d'éclampsie très-graves survenues au 7e mois en trois séries avec intervalle de 2 ou 3 jours, l'ensemble des crises ayant duré conséquemment environ 9 jours. | Aucune autre affection infantile; aucun soupçon de syphilis. La famille, composée de: père, 50 ans; mère, 40 ans, et 3 enfants: 1º celui qui fait l'objet de l'observation; 2º F., 16 ans, ayant eu à 10 ans une rougeole simple; 3º H., 12 ans, ayant eu à 2 ans une fièvre typhoïde très-grave ayant duré 6 semaines. Chez ces deux derniers, aucune érosion. La famille, habitant la campagne, a été observée de tout temps par le médecin de la localité, qui donne sur la question de la syphilis les assurances les plus formelles. |
| 26 | ,, | F 21 ans. | Erosion en double sillon au tiers inférieur des incisives centrales supérieures, en sillon unique près du bord libre des latérales, en double sillon aux incisives inférieures, et au quart supérieur, en nappe profonde à la partie moyenne des 4 premières molaires; rien aux canines dont l'angle est cependant émoussé. (V. tableau nº 2, fig. 6.) | Deux séries d'attaques éclamptiques: la première survenue au 6e mois est caractérisée par dix attaques se produisant pendant 24 heures suivies de coma et laissant après elles divers désordres de la santé. La seconde apparaissant au 9e mois à peu près de même nature et de même durée totale. | Aucune affection infantile quelconque jusqu'à l'âge de l'éruption. Une enquête très-minutieuse ne permet aucun soupçon de syphilis. Le sujet a deux frères plus jeunes que lui, dont l'un n'a jamais été malade dans son enfance et l'autre a eu une scarlatine grave à 3 ans. Aucun d'eux n'a d'érosion. |
| 27 | ,, | F 20 ans. | Erosion en nappe des douze dents antérieures et des quatre molaires, sauf les deux incisives inférieures gauches où la lésion a le caractère d'échancrure par suite de la fracture du bord libre. (V. tableau nº 2, fig. 7.) | A 4 mois, série d'attaques convulsives dont l'ensemble a duré 18 heures. Etat très-grave, menaces d'asphyxie; à la suite, contracture permanente des deux membres du côté gauche; réapparition 2 jours après d'une nouvelle série d'attaques durant 15 jours et caractérisées par 2 ou 3 attaques par jour suivies de coma; troubles de l'intelligence jusqu'à l'âge de 18 mois où la santé se rétablit. | Aucun soupçon de syphilis ainsi qu'il résulte de l'examen approfondi de la famille: père et mère vivant encore, frère cadet très-bien portant, sans érosion. |
| 28 | ,, | F 20 ans. | Erosion en nappe occupant à la mâchoire supérieure à peu près la moitié de la hauteur des incisives centrales, le tiers des latérales, et le sommet des canines. A la mâchoire inférieure le tiers de la hauteur des incisives centrales, et le quart des latérales, le sommet des canines; le quatre premières molaires sont détruites par la carie. (V. tableau nº 2, fig. 8.) | Nombreuses séries d'attaques éclamptiques comprenant dans leur ensemble la période entre le 8e et le 30e mois, et consistant en crises graves suivies de coma durant de 4 à 6 heures. | Le sujet a présenté dans son enfance des signes certains de rachitisme; mais l'examen approfondi de la famille composée de père et mère, 2 sœurs aînées et un frère cadet, tous vivants et bien portants. L'état de ce sujet a été attribué à une variole grave de la mère pendant sa grossesse. |

TABLEAU No. 3—*continué.*

| Nos. d'Ordre. | Noms des Auteurs. | Sexe et Age des Sujets. | Forme et Niveau de l'Erosion. | Date et Durée des Attaques Eclamptiques. | Observations. |
|---|---|---|---|---|---|
| 29 | Magitot. | F 22 ans. | Erosion consistant en un sillon unique siégeant au même niveau vers la partie moyenne des quatre canines. (V. tableau nº 2, fig. 9.) | A l'âge de 2 ans et sans aucune affection antérieure, méningite grave ayant mis en danger pendant 3 semaines les jours de l'enfant. | Aucune autre affection infantile quelconque ; aucun soupçon de syphilis. |
| 30 | ,, | F 15 ans. | Erosion consistant en un sillon unique siégeant vers la partie moyenne des 4 premières molaires ; rien à aucune autre dent. (V. tableau nº 2, fig. 10.) | A 3 ans et 4 mois, série d'attaques convulsives ayant duré 56 heures et suivie d'un coma pendant 10 jours avec perte de connaissance. | Aucune autre affection infantile; aucun soupçon de syphilis. |
| 31 | ,, | H 17 ans. | Sillon simple vers le tiers des incisives centrales, au quart des latérales, au cinquième des canines ; sillon au milieu des molaires. | Au 18e mois, méningite avec 3 jours de gravité et convulsions. | ... ... ... .. |
| 32 | ,, | F 14 ans. | Erosion en nappe de 1 millimètre 1/2 de hauteur aux incisives, aux canines, et aux premières molaires. | Série d'attaques convulsives du 10e au 18e mois. | Aucune autre affection infantile quelconque. |
| 33 | ,, | F 18 ans. | Erosion en sillon pointillé au milieu des incisives centrales supérieures et des 4 inférieures, près du sommet des canines, et vers le milieu des premières molaires. | Une seule série d'attaques éclamptiques avec coma profond durant 4 heures. | Aucun soupçon de syphilis ; parents, frère aîné et frère cadet bien observés et ne présentant aucune trace morbide. |
| 34 | ,, | F 22 ans. | Sillon de 1 millimètre de largeur au milieu des incisives centrales, au tiers des latérales, au quart des canines. Les molaires manquent. | Au 13e mois, série d'attaques éclamptiques occupant une période de 3 semaines. | Observation très-minutieuse de la famille ; aucun soupçon de syphilis. |
| 35 | ,, | H 25 ans. | Erosion en sillon pointillé tout à fait au-dessous du bord libre des incisives centrales supérieures, des 4 inférieures ; rien aux latérales supérieures ni aux canines ; érosion profonde des molaires. | Dans le cours du 4e mois, deux séries d'attaques convulsives le même jour et durant ensemble 24 heures. | Ce jeune homme, qui est fils de médecin et étudiant en médecine, donne sur ses antécédents les renseignements les plus précis qui éloignent toute idée de syphilis. |
| 36 | ,, | F 18 ans. | Erosion en échancrure du bord libre des incisives centrales supérieures, des 4 inférieures ; sillon au tiers des premières molaires. | Au 6e mois, une seule série d'attaques convulsives ayant duré 4 heures | Aucune autre affection infantile, quelconque ; aucun soupçon de syphilis. |
| 37 | ,, | F 20 ans. | Erosion en nappe du tiers de la couronne des incisives centrales, au quart des latérales ; sillon annulaire au sommet des canines ; les molaires manquent. | Au 4e mois, série d'attaques convulsives durant 3 semaines et laissant à sa suite des accidents divers, graves, jusqu'au 18e mois. | L'examen très-attentif de la famille et de deux frères plus jeunes ne permet de retrouver aucune trace de syphilis héréditaire. |
| 38 | ,, | H 11 ans. | Erosion en escalier comprenant 6 ou 8 sillons vagues superposés aux incisives, aux canines, et et aux premières molaires. | Au 6e mois, début d'une série d'attaques éclamptiques durant plus de 6 mois à 8 à 10 à chaque série ; coma consécutif. | Ces deux sujets qui sont frères appartiennent à une famille composée de père et mère très-robustes et de six enfants : deux qui font l'objet de ces observations et quatre autres n'ayant jamais été malades et ne présentant aucune érosion ; aucun soupçon de syphilis héréditaire. |
| 39 | ,, | H 8 ans, frère du précédent. | Erosion en sillon des 2 incisives centrales supérieures ; les latérales manquent ; sillon aux 4 incisives inférieures, et aux 4 premières molaires. | Série d'attaques éclamptiques au 7e mois de la vie. | |
| 40 | ,, | H 25 ans, étudiant en médecine. | Erosion en échancrure du bord libre des incisives centrales supérieures, des centrales et latérales inférieures, du sommet des canines et d'une certaine étendue des premières molaires. | Au 6e mois, à la suite d'une chute d'un 1er étage sur la tête, série d'attaques convulsives durant 5 jours. | Le sujet, très-préoccupé des opinions qui rattachent l'érosion dentaire à la syphilis héréditaire, se prête au plus minutieux examen des antécédents et de l'état actuel ; aucun soupçon de syphilis. |

On voit donc sur quel ensemble de considérations repose la théorie que M. Parrot désigne lui-même sous le nom de *théorie des dents éclamptiques;* mais il nous reste cependant une dernière tâche, celle de répondre en quelques mots à plusieurs objections, fort sérieuses en apparence, que nous a posées M. Parrot.

Ces objections se réduisent aux deux suivantes :

1° L'éclampsie infantile est incapable, dit-on, de produire l'érosion dentaire.

2° Tout sujet porteur d'érosion présente des traces de syphilis héréditaire.

Examinons brièvement ces deux points :

Une attaque d'éclampsie, n'ayant qu'une durée de quelques minutes, ne peut interrompre la formation des tissus de l'émail et de l'ivoire pendant un temps suffisant pour produire une trace indélébile, et M. Parrot ajoute qu'en donnant, par exemple, à une attaque la durée d'une minute, il faudrait, pour produire certaines érosions en nappe, un nombre énorme de convulsions, 42,000 ; 600,000.

Assurément, si nous considérons l'attaque éclamptique en elle-même, nous reconnaissons son impuissance à produire un tel résultat. Mais ne sait-on pas que cette attaque convulsive n'est que la manifestation extérieure, le reflet d'un état profondément troublé du système nerveux et de la nutrition générale ? On n'en saurait douter en considérant que l'éclampsie, quand elle n'amène pas la mort, a parfois laissé à sa suite des lésions graves telles que la perte de l'intelligence, le strabisme, la surdi-mutité, le bégaiement, des contractures des membres, &c.

D'autre part, on sait que l'éclampsie ne se manifeste jamais par une seule attaque, mais par des séries plus ou moins nombreuses et plus ou moins rapprochées, de sorte que l'influence perturbatrice correspond, non à l'attaque isolée, mais à la série. Or, cette série, d'après maintes observations, s'étend parfois à dix, quinze, vingt heures, chaque attaque réapparaissant toutes les demi-heures ou toutes les heures.

Un auteur récent, Kien (de Strasbourg), cite le cas d'un enfant qui a présenté une série d'attaques se reproduisant toutes les heures pendant quatorze heures et suivies d'une période de coma durant six jours, et interrompue encore par des crises plus faibles et plus rares. C'est donc une période de perturbation grave de la nutrition ayant duré chez cet enfant près de sept jours, pendant lesquels on a noté le pouls à 140 et la température à 40°.*

On voit donc que les troubles de nutrition, sous l'influence de l'éclampsie, peuvent durer un temps fort long. Nous possédons des faits dans lesquels ils se sont prolongés pendant des mois et des années.

Mais ce n'est pas tout, et nous avons voulu établir expérimentalement quelle quantité de dentine et d'émail se produit normalement pendant un temps donné dans un follicule et par suite quelle étendue et quelle profondeur présentera la zone de tissu frappée d'interruption.

Voici l'expérience :

" Sur un chien de quinze jours, allaité par sa mère, nous avons enlevé par une incision légère le chapeau de dentine de la canine gauche. L'animal est aussitôt remis à sa niche et continue de téter sans trouble apparent.

" Dix jours plus tard, à la même heure, le chapeau de dentine de la canine droite est extrait à son tour. Ces deux préparations sont lavées et séchées. Les voici sous une petite cloche de verre. Elles présentent une différence de hauteur qui se chiffre par 6 millimètres ; le petit chapeau ayant 6, le grand 12 millimètres."

Six millimètres en dix jours nous donnent 6 dixièmes de millimètres par jour. Par heure, nous obtenons 20 millièmes de millimètres plus une fraction. Négligeons cette fraction et faisons le calcul suivant :

Si une cause morbide était venue interrompre dans ce petit chapeau la

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* *Gazette Médicale de Strasbourg*, 1880, No. 7, p. 73.

formation de l'ivoire et de l'émail pendant seulement cinq heures, le sillon produit aurait plus d'un dixième de millimètre de largeur, c'est-a-dire qu'il serait visible à l'œil nu.

Voici sur des moulages pris sur le vivant des sillons de cette étendue et dus à des séries relativement courtes d'attaques éclamptiques. Voir dans le tableau No. 2, les figures 1, 6, 9, et 10.

Poursuivons ce calcul, et nous verrons qu'une série de crises durant dix heures donnera un sillon de deux dixièmes de millimètre, vingt heures quatre dixièmes, et ainsi de suite.

Qu'on juge dès lors de l'étendue d'une érosion sous l'influence d'un état morbide durant plusieurs mois.

Mais ce n'est pas tout, et M. Parrot s'étonne que certaine dents ne soient jamais atteintes, la seconde et la troisième molaire par exemple, et il trouve la théorie de l'éclampsie incompatible avec ce fait.

Le tableau No. 1 publié ci-dessus me paraît répondre complètement à cette objection. On y voit en effet que le chapeau de dentine de la seconde molaire n'apparaît qu'à $3\frac{1}{2}$ ans en moyenne; or il est notoire que l'éclampsie devient fort rare à partir de cette époque. On sait même que la plupart des médecins en attribuent, empiriquement il est vrai, la production à la première dentition qui est achevée à cet âge. Quant à la troisième molaire ou dent de sagesse l'apparition de son chapeau à la 12$^e$ année la met hors de cause.

C'est en raison de ces rapports chronologiques entre l'évolution des dents et l'âge d'élection de l'éclampsie que la première molaire permanente se trouve constamment frappée à des degrés ou à des niveax divers. Son chapeau de dentine débutant, en effet, au sixième mois de la vie intra-uterine tandis que son éruption n'a lieu qu'à la sixième année, elle est ainsi soumise pendant plus de six ans à toutes les perturbations morbides qui atteignent la première enfance.

L'érosion de cette première molaire en particulier porte en outre très souvent à la surface triturante elle-même ce qui implique un trouble fœtal. Il en est de même de l'érosion des dents temporaires, et M. Parrot nous demande d'expliquer ici une telle lésion. Croyons nous à l'existence de l'éclampsie intrautérine?

Nous avouerons à cet égard notre ignorance complète sur ce point de pathologie fœtale. Mais il est fréquent de voir des accidents ou des maladies maternels retentir assez gravement sur l'embryon pour en troubler la nutrition et jouer ainsi à cette période des influences si faciles le rôle que joue l'éclampsie dans le première enfance.

Dans un cas de ce genre où les premières molaires étaient seules atteintes d'érosion (voy. tableau 2, No. 1), l'étude des antécédents nous a permis de retrouver dans l'histoire de la mère des accidents graves de la grossesse survenus vers le 7$^e$ et le 8$^e$ mois, amenant l'accouchément prématuré au 8$^e$ mois et la mort dans les premières heures qui suivirent. Toutefois la question posée de l'éclampsie maternelle n'a pas reçu de solution par insuffisance des souvenirs.

Pour les dents temporaires, on sait qu'elles sont très rarement frappées d'érosion, et dans une de nos observations (obs. 22, tableau No. 2), le mécanisme en a été facilement reconstitué. Mais si M. Parrot croit l'avoir trouvée frequemment c'est qu'il a pu, ce nous semble, confondre avec l'érosion des altérations cadavériques.

On reconnaît en effet que si l'on enlève, dans un follicule clos un chapeau de dentine qu'on abandonne à la dessication spontanée, l'émail incomplètement calcifié se couvre de félures de craquelure par la dissociation et la destruction des prismes. Cet accident simule parfaitement l'érosion et nous l'avons reconnu sur plusieurs des pièces que M. Parrot a présentées l'année dernière au Congrès de Reims.

Arrivons enfin à la dernière objection de M. Parrot, la voici:

Tout sujet porteur d'érosion dentaire présente des vestiges de syphilis

héréditaire. Nous renverrons pour toute réponse à l'analyse des quarante observations que nous venons de résumer. On y trouvera un grand nombre de relations dans lesquelles l'examen le plus minutieux des parents, des frères et sœurs, et du sujet lui-même, n'autorise aucun soupçon de syphilis. La publication détaillée des observations de ce genre qui composent notre dossier personnel allongerait indéfiniment ce travail et n'apprendrait rien de plus que les resumés que nous en avons donnés.

*Conclusions.*—De l'ensemble des documents et des considérations qui précédent, nous déduirons les conclusions suivantes :

1° L'éclampsie infantile produit constamment dans la nutrition intra-folliculaire une perturbation qui se traduit par une des lésions caractéristiques de l'érosion dentaire.

2° Le *niveau*, le *nombre* et l'*étendue* de la lésion de la couronne correspondent à l'*époque*, à la *durée* et à l'*intensité* des crises éclamptiques ; l'érosion est *une*.

3° Les autres maladies de l'enfance, fièvres éruptives, affections catarrhales, intestinales, &c., sont impuissantes à produire l'érosion. Toutefois quelques maladies graves et de très-longue durée peuvent amener une désorganisation totale de la couronne des dents en voie d'évolution, mais non l'érosion proprement dite.

4° La syphilis héréditaire, dont on ne peut nier l'influence sur la constitution générale des tissus osseux et dentaires en voie de formation ne se traduit jamais par les lésions caractéristiques de l'érosion.

Et comme corollaire :

Tout sujet, notoirement affecté de syphilis héréditaire, ne présentera jamais d'érosion que si, dans ses antécédents, on retrouve simultanément la trace de l'éclampsie.

5° L'érosion dentaire constatée dans les gisements préhistoriques serait, d'après les raisons invoquées par Broca et par nous-mêmes, la preuve de l'éclampsie infantile, laquelle constituait le but déterminant de la trépanation crânienne.

6° L'érosion dentaire, dans ses formes les plus caractéristiques, n'est pas exclusive à l'homme et peut se rencontrer chez des animaux domestiques que la syphilis n'atteint pas.

## DISCUSSION.

Mr. Moon, London : In discussing this subject, it may be taken for granted that all medical men will admit that hereditary syphilis is very apt to be productive of a deformity of certain among the permanent teeth ; but there are not a few in the ranks of medicine and surgery who consider that such malformation may be due to other causes besides syphilis. Now this doubt is the outcome, I believe, of a widespread want of distinctness that prevails as to what a typical syphilitic tooth really is. Mr. Jonathan Hutchinson having, years ago, shown the way, it of course is comparatively easy for others to follow out details of this subject. The diagnostic value of a syphilitic tooth depends greatly on this fact—viz., whether such malformation is distinctive and due to syphilis alone, or whether other causes may bring about the like malformation? Some years ago I collected cases and took models of a large number of cases bearing on this subject, and became thereby convinced that there is a special kind of malformation which may be depended on as indicating hereditary syphilis and nothing else. In order to properly appreciate divergences from the normal form of teeth, it is necessary that some features of what may be called the architecture of a normal tooth should be known. To elucidate this subject and to lessen the probability of my remarks wearying

you, I have brought down some models and put up some drawings which will, I hope, clearly bring out the normal form of syphilitic teeth, and the shape of such teeth as are apt to be confused with the latter.

A description was then given of the differences between normal and syphilitic teeth, and the modes of distinguishing the latter from other malformed teeth. The different kinds of defective enamel formation were then touched on, and the question suggested that perhaps "mercurio-syphilitic tooth" might be the right term for certain teeth which presented a want of enamel over a semi-lunar space in the centre of the first-formed portion, leading often to the breaking down of the unprotected dentine and the formation of a semi-lunar notch.

Mr. C. S. Tomes, London: exhibited a model of teeth presenting in a marked degree M. Magitot's "erosion," remarking that the history of the patient was perfectly known to him, and that no attack of convulsions had ever occurred. The patient had, however, suffered from a severe attack of inflammatory croup at such an age as to exactly correspond with the lesion of the teeth. During this attack she was salivated.

Mr. Coleman, Streatham: It was my good fortune to be a colleague of Mr. J. Hutchinson at the period when that gentleman's attention was first directed to this subject. I thus had the opportunity of seeing a considerable number of Mr. Hutchinson's earliest cases, and to his first communication—viz., that to the Pathological Society—was appended a report by me of th peculiarities such teeth presented. I confess I was in the first instance a sceptic with regard to the syphilitic origin of these teeth, but as time wore on I can only come to the conclusion of the correctness of Mr. Hutchinson's views. My observations have now been extended over some twenty years, and from holding hospital appointments I have had large opportunities of inspecting the teeth of all classes, and my firm and settled conviction is, that between cause and effect there cannot in my mind be the least doubt. Persons the subjects of inherited syphilis may have well-formed and excellent teeth, but in my belief no person can have the type of teeth described by Mr. Hutchinson who has not inherited syphilis.

Dr. Blache, Paris: In a paper which I published in 1879 on "Malformation of Teeth in Children as a Symptom of Hereditary Syphilis," I quoted five instances of cases having reference to the first dentition. In taking part in the interesting discussion opened to-day, I claim to contribute towards the establishment of the fact of "erosion" taking place in the milk-teeth, not only in cases of syphilis or in the children of syphilitic parents, but sometimes also quite independently of this condition. Thus I have had under observation for four years a child at whose birth I was present, and whose father and mother I know and believe to be absolutely free from syphilitic disease. This child has marks of "erosion" on its lower incisor teeth and on the two canines, which I cannot refer either to syphilis or to those convulsions which my friend and colleague M. Magitot deems to be the origin of this affection.

Dr. Hayward, Liverpool: I wish to contribute a practical case. The parents were perfectly healthy, having six quite healthy children; after this the father had occasional intercourse with a lady with tertiary syphilis during daytime, and at night with his own wife. After this twins were born; both with very definite signs of inherited syphilis, one dying within three months, the other struggling through, but absolutely without mercurial treatment. Its primary teeth were black and soft, the permanent incisors some defective of dentine, with small

cutting edges and large at the neck, and others defective of enamel and showing honeycomb appearance. There was scarcely any medicinal treatment, and certainly not mercurial. The child never had convulsions or any similar disease ; only a great liability to bronchitis. This would show marked teeth originating without nervous disease or mercury, but certainly from syphilis ; this view being further supported by the fact that both parents afterwards had cutaneous syphilis.

Dr. Dally, Paris : It is many years since M. Magitot first acquainted me with his views on the origin of erosions of the teeth. Since then I have made extended inquiries in my out-patient practice amongst the children whom I attend for deformities of the organs of locomotion, and my inquiries have very rarely failed to elicit a history of convulsions. At the same time I would add that I have never been able to entirely exclude the suspicion of syphilis. The difficulty is that people are ready enough to admit convulsions, but will always endeavour to conceal syphilis. Why must I exclude one of these two causes ? Because hereditary syphilis does not cause disturbances of the nerve centres ? Convulsions are only a symptom. There is some morbid cause behind them, and do we know of any *causa morborum* more active, more profound, and more protean than syphilis ? Erosion of the teeth is evidence of a violent and passing disturbance of general nutrition. True ; and the general conclusion which, as it appears to me, is to be deduced from this discussion is, that all the causes which are capable of interfering with general nutrition—accidental and physiological causes included—are capable of producing erosions of the teeth.

Mr. Hutchinson, London : I avail myself with some pleasure of the opportunity which this debate affords me of attempting to correct some misapprehensions in reference to the clinical symptology of the teeth. M. Magitot makes the denial that honeycomb teeth or eroded teeth are indicative of inherited syphilis one of the main points of his paper, and he seems to think that in saying this he is controverting an opinion of mine. But in point of fact we are in accord, for this is what all along I have most carefully asserted. The honeycombed eroded teeth have always for me been indicative of infantile stomatitis (mercurial or other), and never of syphilis. I am aware that mistakes have been made, and that others besides M. Magitot have supposed that this condition of erosion was what I had described as indicating syphilis. Thus, for instance, most of the casts which M. Parrot shows in our museum as syphilitic are not what I should regard as syphilitic at all, but simply "mercurial." This error has been the more frequent because, as a fact, we very frequently—indeed I may say usually—meet with the malformations due to syphilis and those due to stomatitis in the same mouth ; for most syphilitic children have taken mercury in infancy. But I cannot myself plead guilty to any responsibility for such mistakes, for both in pictorial representation and description, I have always carefully noted the differences and distinguished the two conditions. Even where the two are co-existent there is no real difficulty in discrimination. Permit me to repeat what I have said over and over again on former occasions, that the dental malformations which denote syphilis are not of the nature of erosion, but consist in very peculiar arrests of development. The only teeth to which I venture to attach much importance are the upper central incisors of the permanent set. In these an arrest in the growth of the middle denticle, leaving a single central notch, is the commonest and most trustworthy condition, but, in addition to it, there is usually a dwarfing of the tooth in all its dimensions, and sometimes a

screw-driver form of tooth is almost as characteristic as a notched one. The defects are usually symmetrical, but sometimes not so. Peculiarities are often to be observed in the other teeth, and are often of much help in corroboration of diagnosis, but in the absence of the peculiarity described in the upper central incisors they are not to be trusted, and for fear of occasioning more mistakes I prefer not to allude to them further. Whilst these malformations of the upper central incisors (notches and dwarfing) are the characteristics of syphilitic teeth, those which imply former stomatitis consist chiefly in defective formation of the enamel. The teeth which show these most constantly are the first permanent molars, the two bicuspids being remarkable for their exemption. The explanation of this is easily given if we remember the dates of the calcification of the several teeth. The first permanent molars may be regarded as the test teeth as regards infantile stomatitis, just as the central incisors are for syphilis, but they are by no means the only teeth affected. Next to them in importance come the incisors, which are almost as constantly pitted, eroded, and of bad colour, often showing a transverse furrow which crosses all the teeth at the same level. M. Magitot, if I have understood him correctly, inclines to the belief that these enamel defects are the consequences not so much of stomatitis as of some interference with development brought about through the nervous system in connection either with infantile convulsions or severe illness in early life. He explains them as we explain the furrows on the nails, so often seen as the record of past disturbances of health. It may be that to some extent he is right. I think it very probable that he is. I have myself long ago published facts bearing upon this very point, and have discussed in some detail the question as to whether, in the cases of enamel defects, so frequently seen with lamellar cataract, and in those who have had convulsions, the latter ought to be regarded as the direct cause of the former. The conclusion to which I have arrived is, that even in cases in which defects are in association with neurosis, still there almost invariably intervenes a stage of inflammation which is the immediate cause of the defects. So it is very possible that some stomatitis teeth may be caused by mercurial stomatitis or that of thrush, and that others may have been induced by a stomatitis due to nervous influences. Still, in both the local process which causes the defect is inflammation. Unquestionably, in the case of the nails, local diseases, certainly inflammatory eczema, &c., produce markings just like those which follow general illness. We are now, Mr. President, I think, in a fair position to discuss the questions, Are the teeth which have been described as syphilitic really due to syphilis? are those which are assigned to stomatitis really in association with that cause? The evidence on the latter point is of a far less conclusive character than that as to the former. I have seen many sets of so-called mercurial or stomatitis teeth in which I could get no evidence that mercury had been given, or that any form of infantile illness had been experienced likely to be attended by stomatitis. Still, although not uncommon, this class of facts has been, in my experience, exceptional, and those which have illustrated what I believe to be the rule have been far more abundant. About the time that we were first noticing syphilitic teeth, and were busy examining the mouths of our young patients, the observation was made that in most cases of infantile cataract the teeth were very defective. I do not know whether Professor Horner of Zurich, Mr. Teale of Leeds, or myself first noted this point, but we were all at work on it together. To Professor Horner, I believe, belongs the credit of having drawn attention to the fact that the kind of infantile cataract usually so attended was almost always "lamellar," and that there was usually also a history of convulsions in infancy. Then comes the problem as to the mutual relationship between these three conditions—convulsions in infancy, lamellar cataract, and defectively

developed teeth. Upon this I wrote a paper which was published in the Pathological Society's *Transactions* in 1875.* I must not weary you by recapitulating the arguments of that paper, more especially as it is accessible to all who feel sufficient interest in the subject to refer to it. Briefly, my conclusion was, that the cataract was due to the convulsions, and the dental defect to the mercury given for the convulsions. It may easily be seen that I was to some extent wrong on this latter point, and I am quite willing to join with M. Magitot in the suspicion that the illness coincident with the convulsions may, in some cases without the help of mercury, produce defects in the enamel, &c. It is clear that we have here an interesting field for further observation. Let those who have the opportunities for knowing the facts accurately, that is, without trusting to hearsay evidence, tell us whether convulsions without mercurial treatment and without any form of stomatitis are often followed by defective teeth. Conversely let those who possess facts as to children who, during the first year, have taken much mercury (as "teething powders" or otherwise), tell us whether in such defective development of the enamel is ever absent in the permanent teeth. There must be plenty of facts of both classes extant if we could only get at them. That mercury given in infancy is one of the causes of eroded teeth I have not the least doubt. Many very convincing facts have occurred within my own observations. My friend Mr. Moon and others have also carefully investigated cases of this kind, and have arrived at the same conclusion. Here, then, I leave this part of our subject; it requires further work, but the main conclusions which I have formerly published are, I still believe, well sustained. Respecting the so-called "syphilitic" teeth, I am not prepared to admit the slightest doubt or hesitation. It is now more than twenty years since I submitted to the Pathological Society, and through it to the profession, the evidence which I had obtained on this point. Since then I have made daily use of these conditions as an aid in diagnosis, and their value has been acknowledged by a host of independent observers in all parts of the world. Indeed I may, I think, without presumption, claim that considerable advances in clinical knowledge have been made in various departments by their aid. Almost simultaneously with their recognition came the observation that "interstitial keratitis," when well marked and symmetrical, may be regarded as certainly syphilitic. It had formerly been always called "scrofulous." Choroiditis disseminata, a peculiar form of deafness, and a special form of rapidly destructive "lupus," were also soon afterwards assigned with confidence to their proper category. More recently, partly by the evidence of such affections as those just mentioned, but more particularly by that given by the teeth, various observers—Dr. Hughlings Jackson, Dr. Barlow, Dr. Allen Sturge, Dr. Down, and others—have recognised rare forms of disease of the nervous system as certainly due to inherited taint. A large group of bone diseases have in like manner, and on like evidence, been transferred from struma and rickets to syphilis. I produced yesterday, at the London Hospital, for M. Magitot's inspection, about a dozen examples of well-marked syphilitic teeth, occurring in association with one or other, or with several of the maladies I have enumerated. Neither these maladies nor the teeth which have helped us to their recognition are, in certain departments of practice at all uncommon; and I repeat, our clinical knowledge of the one rests to a large extent upon the trustworthiness of the other. Such being the case, and these facts having been in the main recognised, not only in England but over the Continent and in America, I submit that Dr. Magitot is a little late in the day when he comes to tell us that these malformations are not indications of inherited syphilis but of convulsions. I hope I shall not be

* *Transactions of the Pathological Society of London*, vol. xxvi. p. 235.

considered over-sensitive if I admit to a feeling that it is a little hard to be called on now, after my conclusions have been so long admitted, to again go over the old ground of proof. I should have no right to resent this if it came from one who had studied my views, and was prepared with facts to refute them. Before I sit down, sir, let me fully admit that it is not always that the teeth of the subjects of inherited syphilis reveal the diathesis. In very many cases they are not malformed at all. In many others the malformations are too slight or too much mixed to be worthy of confidence, though still possibly very valuable by way of corroboration. The frequent coincidence of syphilitic with mercurial peculiarities has made it much more difficult for learners to obtain clear perceptions respecting them than would otherwise have been the case. The neglect to discriminate between the two has led to innumerable errors. Into this error both Professor Parrot and the author of this paper have, I hold, fallen, and hence the doubts expressed by the latter.

Dr. Quinet, Brussels: There is no doubt that M. Parrot and Mr. Hutchinson hold different views as regards the influence of hereditary syphilis on the temporary teeth. It is proved by the facts of surgery that this diathesis, which so often kills the child in its mother's womb, and leaves its traces on the thymus gland, on the lungs, and on the liver, must also necessarily leave its mark upon the temporary teeth. Animals also sometimes present in their teeth the marks of blind erosion, as in the oturia gulata and the oturia stellaris in the Museum of the Royal College of Surgeons. Lastly, I recognise a general diathesis—rachitis scrofula—which has the same influence on the dental organs, an influence slow and prolonged in its action, and leaving its traces in the entire organs, and especially characterised by uniformity.

Professor Parrot, Paris: I shall answer the argument of M. Magitot, who rejects the syphilitic origin of dental atrophy, and adopts the "convulsive" theory, by three remarks. Firstly, I would call attention to the *systematic* nature of the dental change, certain teeth being affected, and others never. Thus all the teeth of the first dentition may be affected, and among those of the second dentition, the incisors, the canines, and the first molars; while the second and third molars never suffer, and the premolars but rarely, as I have never seen an example. Why should certain teeth have such an immunity? Convulsions take place at all times of infantile life, so all the teeth ought to be affected. Do convulsions or eclampsia take place in intra-uterine life? We do not know anything on this matter, but it is not very probable. But it is during intra-uterine life that the development of the caps of dentine of the first dentition takes place, and indeed also of several of those of the second dentition. It would therefore follow that during this time there must be numerous convulsions. And, lastly, the extent of the affected dentine and enamel cannot be regarded as agreeing with the relatively short duration of the convulsive attacks.

M. Magitot, Paris: Le premier orateur qui a pris la parole après ma communication, M. Moon, attribue les diverses espèces d'érosions à l'action du mercure. C'est une opinion que je ne saurais comprendre. Si nous connaissons bien les effets du mercure, c'est sur les gencives et non sur les dents qu'il porte ses actions et la gengivite est capable dans certains cas de produire sur les dents une certaine altération. C'est là une lésion tout autre et qui n'a rien de commun avec celle-ci. Il nous semble donc qu'il y a ici de la part de M. Moon une confusion entre l'érosion proprement dite qui est congénitale et tératologique et certaines altérations pathologiques de la couronne qui se montrent au niveau même du bord gengival enflammé. Mon excellent ami

M. Ch. Tomes défend encore aujourd'hui, je le vois, son ancienne opinion qui rattacherait l'érosion aux affections diverses de l'enfance. J'ai insisté assez longuement sur cette explication pour n'y point revenir : les conditions essentielles pour qu'une érosion puisse se produire, c'est *l'invasion brusque*, suivie de cessation aussi brusque qui caractérise l'affection, cause possible d'érosion ; c'est ce caractère qui amène l'interruption subite entre la région restée saine de la couronne et l'échancrure ou le sillon. C'est encore la nature même de la maladie intercurrente qui doit être à la fois assez grave pour amener une perturbation ou une suppression dans la formation des tissus, et assez générale pour causer un tel trouble de la nutrition. Or, ces conditions, nous les remontrons au plus haut degré dans l'éclampsie dont le mécanisme, bien qu'encore indéterminé, répond certainement à un état du système nerveux ; état toujours grave, et dont les crises convulsives si brusques et si fugaces sont la manifestation extérieure. Cette objection s'adresse également à M. Dally dont l'opinion se rapproche de celle de M. Tomes. En ce qui concerne M. Coleman qui est le partisan des idées de M. Hutchinson ma réponse se confondra avec celle que j'opposerai au chef de la doctrine, à M. Hutchinson lui-même. Or, le savant médecin de l'hôpital de Londres ne reconnaît pas dans les spécimens des lésions que je présente les altérations caractéristiques, suivant lui, de la syphilis héréditaire, celles qui accompagnent la kératite interstitielle, affection qui serait essentiellement syphilitique. Pour M. Hutchinson la seule forme caractéristique de la syphilis, c'est l'échancrure du bord libre. Il nous paraît cependant impossible de se refuser à admettre que les variétés quelconques d'érosion sont de nature identique et que dès lors si la syphilis héréditaire peut produire certaines formes, elle en produirait aussi bien certaines autres, la seul différence portant sur l'époque d'invasion des accidents et le niveau contemporain de l'érosion. Or je produis ici un nombre considérable de faits d'érosion de toutes les formes, celles de M. Hutchinson, aussi bien que les autres et dans leurs rapports étroits avec l'éclampsie. Si M. Hutchinson persiste à regarder comme syphilitique la disposition spéciale en échancrure, je lui demanderai d'établir deux conditions : la première c'est de prouver que les sujets qui en sont porteurs n'ont pas présenté dans leur enfance et à l'époque contemporaine aucune crise éclamptique ; la seconde c'est de démontrer que les mêmes sujets sont bien et dûment des syphilitiques héréditaires. Or M. Hutchinson n'a pas et ne peut avoir sur beaucoup de ses malades aucun renseignement précis sur la question de l'éclampsie. Ces sont des individus de la consultation externe sans famille sans parent pouvant donner des renseignements sur leur première enfance. J'ai certainement constaté dans plusieurs cas que M. Hutchinson a bien voulu mettre sous mes yeux la co-existence de l'altération dentaire et de la kératite interstitielle, mais de là à la communauté étiologique, il nous semble qu'il y a encore loin. Enfin il est une autre question à juger, celle de la nature syphilitique de la kératite parenchymateuse elle-même, que certains auteurs considérent comme une manifestation scrofuleuse. Or l'identité des deux diathèses n'est peut-être pas suffisamment démontrée.

Ces diverses observations s'adressent naturellement à M. Parrot à qui je dois, cependant, une réponse sur plusieurs points particuliers :

M. Parrot me demande comment j'explique que les dents temporaires soient si rarement atteintes d'érosion et que certaines dents permanentes (deuxième-prémolaire, deuxième et troisième molaires) ne le sont jamais.

Le première explication est fort simple : Les dents temporaires n'accomplissent pas leur évolution complète avant la naissance, et le tableau que nous avons montré indique pour chaque dent la hauteur de son chapeau de dentine à cette date. Or, que l'intervention morbide survienne dans les premières semaines de la vie, l'érosion occupera sur la couronne des dents temporaires un point

déjà très éloigné du bord libre, et c'est aussi que les dents de lait ne présentent jamais d'érosions en coup d'ongle mais bien des sillons toujours très élevés.

Il est certain toutefois que certaines dents temporaires présentent manifestement des traces d'altérations analogues, si non identiques à l'érosion, et c'est pour ces cas que M. Blache demande si je crois à une influence morbide intra-utérine. Apparement, toute lésion des dents temporaires implique une cause absolument maternelle. Mais qu'elle est cette cause? Ce'st encore, dira-t-on, l'éclampsie? Je n'oserais ici l'affirmer bien que j'ai cru une moment saisir un fait de genre chez une mère morte en couches à huit mois d'un enfant porteur d'une érosion aux canines temporaires. Mais à qu'il est facile de comprendre c'est qu'à cette période fœtale, les influences perturbatrices de l'évolution intra-folliculaire d'une dent sont encore plus facile, que plus tard et que le retentissement de la mère sur le produit soit très admissable.

Nous avons dit d'ailleurs dans le cours de notre communication que beaucoup de cas d'érosion de dents temporaire présentés par M. Parrot nous avaient semblé être des altérations cadavériques. Maintenant, si certaines dents permanentes ne sont jamais frappée d'érosion, c'est ainsi que nous l'avons fait remarquer, que l'état de leur développement ne s'y prête pas. Dans un cas d'érosion en sillon de deux premières prémolaires nous avons retrouvé l'éclampsie à trois ans et demi, époque contemporaire de l'état de développement de la couronne. Enfin, les deux dernières molaires apparaissant encore plus tard, aux dates que nous avons établies, ne peuvent subir l'influence de l'éclampsie, affection dont l'apparition ne dépasse guère la quatrième ou cinquième année. M. Parrot insiste avec raison sur la nécessité, dans cette recherche des causes de l'érosion dentaire, de systematiser les faits qui s'y rattachent. C'est précisement à quoi tendent tous mes efforts : seulement tandisque M. Parrot systématise au point de vue de la syphilis, je systématise au point de vue de l'éclampsie, et c'est en raison de ce principe que je renonce à comprendre comment la syphilis héréditaire, dans son action lente et continue, peut produire, avec cette netteté d'un sillon ou d'une échancrure, l'altération que nous avons décrite.

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## *Les Bossus et la Méthode de M. Sayre.*

Dr. A. M. Da Cunha Bellem, Lisbon.

Messieurs et Très-Honorables Confrères,—Lorsque nous avons été à la sixième session du Congrès International des Sciences Médicales, réuni à Amsterdam en 1879, nous avons eu l'avantage d'y entendre l'exposition faite par M. le professeur Sayre, de New York, à l'égard de sa méthode pour le traitement des sujets atteints du mal de Pott et de scoliose, et d'y voir l'application du bandage plâtré à un jeune bossu, qui portait depuis long temps et sans aucun résultat un appareil orthopédique. La simplicité de la théorie, ainsi que celle de la méthode même nous a séduit, et nous nous sommes empressé, dès notre retour à Lisbonne, de faire venir de Londres un trépied, d'après le modèle du professeur américain, pour faire des essais sur sa méthode.

Nous avons eu l'honneur d'être le premier et le seul jusqu'à ce moment qui avons fait en Portugal l'application du bandage plâtré dans le but de maintenir le redressement da la colonne vertébrale, obtenu par la suspension, selon les renseignements du très-illustre praticien du Bellevue-Hôpital.

Nos observations n'ont pas été nombreuses, mais elles suffisent pour nous mettre à même d'avoir des idées arrêtées sur les avantages de la méthode de M. Sayre, tour à tour vantée outre mesure, et blâmée d'une façon impitoyable.

Avant de rapporter devant cette respectable assemblée ce que nous avons recueilli de notre expérience à cet égard, il nous faut avouer que nous n'avons reconnu ni les torts que l'on s'est plu à attribuer à la méthode ni les merveilleux résultats que l'inventeur en a prônés.

D'abord on blâma le chirurgien américain de ne pas avoir été original dans les ressources qu'il met à profit pour le redressement de la colonne vertébrale, et d'avoir caché les noms de ceux qui l'ont devancé en des démarches analogues à celles sur lesquelles reposent la théorie et la pratique de la méthode.

Nous ne voulons pas aborder cette question de priorité, mais nous trouvons bien remarquable que ceux-là mêmes qui blâment la méthode, qui lui font l'accusation formelle d'être erronée, réclament avec autant d'empressement pour leurs compatriotes la priorité de l'erreur.

L'idée de la suspension des bossus, le redressement des courbures de la colonne vertébrale par l'extension et par la contre-extension est d'ailleurs tellement simple, tellement intuitive, qu'il ne faut pas nous étonner de ce qu'elle se soit présentée à plusieurs reprises à tous ceux qui cherchent le moyen de guérir une difformité aussi affligeante que celle qui frappe les bossus.

Eloigner les épaules des hanches, voilà l'idéal de tous ceux qui tiennent à guérir ou du moins à améliorer les courbures du rachis, qui racourcissent l'espace entre les omoplates et les os du bassin.

Que les efforts de ce redressement soient faits, le patient couché ou debout, à la position horizontale ou à la position verticale, tiré des deux bouts de son corps, d'une façon ou d'une autre, le soutenant par la tête ou par les bras, afin que le poids même du tronc et des membres inférieurs fasse la contre-extension, ou le faisant s'appuier sur le point saillant de sa gibbosité, de manière à se procurer le redressement de la colonne vertébrale par l'action des deux leviers représentés par les deux parties du corps, depuis la saillie du rachis jusqu'au synciput, et jusqu'aux talons, ce sont là des détails très-secondaires de n'importe quelle méthode de traitement des difformités de l'épine.

Les négres d'une des possessions de l'Afrique portugaise suspendent par les bras les jeunes bossus et leur envelopent ensuite le thorax d'un entourage résineux.

Pourrait-on blâmer M. Sayre d'avoir emprunté l'idée fondamentale de sa méthode aux manœuvres brutales des tribus africaines soumises à la domination du Portugal ?

Pourrait-on même reprocher à M. Cocking d'y avoir puisé l'idée de son gilet de feutre, imprégné de résine, dont M. Busch, de Bonn, a fait usage, en remplaçant les bandages plâtrés ?

Qu'est-ce que cela prouve ? Que le principe sur lequel repose le traitement des courbures de la colonne vertébrale par l'extension et la contre-extension, ainsi que celui de l'immobilisation du rachis, le redressement, plus ou moins complet obtenu, sont des principes intuitifs, qui se sont présentés à l'esprit des savants, et à l'esprit des ignorants, à Bouvier, à Gillebert d'Hercourt, à Shaw, à Glisson, à Nucks aussi bien qu aux négres de l'Afrique portugaise, à M. Sayre, ainsi qu'à ses devanciers.

Quoiqu'il en soit, la méthode du chirurgien de New York a fait du bruit dans le monde médical, elle a fixé l'attention de plusieurs chirurgiens de l'Europe et elle y a fait ses preuves, les opinions se partageant à l'égard de ses avantages.

Pour ce qui nous concerne, nous nous maintenons au juste milieu entre les détracteurs et les prôneurs de la méthode, notre expérience nous apprennant que l'on échoue autant que l'on réussit.

Les vues particulières, que M. Sayre a sur le mal du Pott, sur le processus pathologique d'après lesquel la courbure de la colonne advient, sont peut-être trop exclusives, le traumatisme pouvant être invoqué assez souvent, mais pas

toujours, les diathèses ayant quelque fois une influence rien que secondaire, pour en avoir la principale dans plusieurs cas.

S'il y a des courbures, le point de départ gisant à la carie d'une vertèbre, qui fait l'angle saillant avec la vertèbre inférieure et les autres, en dessus et en dessous, restant à peu près régulières ou tout-à-fait régulières, il y en a d'autres, où la série des vertèbres supérieures et celle des vertèbres inférieures, au point anguleux qui fait la saillie de la gibbosité, sont elles-mêmes déformées dans leur rapports réciproques, de sorte à former non une ligne à peu près droite de corps superposés, mais la ligne courbe des étéments d'une voûte, la difformité s'élargissant pour ainsi dire sur et sous son point de départ.

Il est évident que les cartilages intervertébraux, si ce n'est pas les corps des vertèbres mêmes, ont souffert un aplatissement à leur partie antérieure, en conséquence du poids qu'elles supportent, et dont le centre de gravité va tomber en dehors de la base de sustentation par effet de la courbure même.

Si la suspension peut amener, tant peu que ce soit, le redressement de l'angle formé par les corps des deux vertèbres où git la difformité, elle peut bien mieux redresser les courbures supérieure et inférieure, qui ne sont que le résultat de la fatigue éprouvée par la colonne, lorsqu'elle soutient la tête d'une façon anormale, et hors des conditions d'équilibre.

Or, lorsqu'on fait la suspension du malade suivant le diamètre occipito-mentonnier, le rachis étant tout-à-fait soulagé, cède lui-même, aux lois de la pesanteur, obéit à la contre-extension faite d'elle même par le poids du tronc et des jambes, et se redresse à merveille.

C'est ce résultat qui représente, selon nous, le plus grand avantage de la méthode de M. Sayre, le redressement de l'angle lui-même devant être proscript à l'état aigu du mal de Pott, ou, à la période de non-réparation ; devenant à peu prés impratiquable ou du moins n'offrant, que de très-faibles chances de réussité, à la période de consolidation absolue, et le chirurgien n'ayant affaire que par exception à des malades dont l'état soit intermédiare entre la réparation incomplète et la consolidation tout-à-fait complète.

Nous avons vu assez souvent échouer la tentative de redressement de l'angle formé par les deux vertèbres malades, tandis que celui de la courbure secondaire des vertèbres supérieures et inférieures réussit parfaitement. Il est d'ailleurs très-facile à comprendre que si l'on a affaire à deux os ankylosés à la suite d'un processus inflammatoire dont ils ont été le siège, on ne parviendra à vaincre leur position vicieuse qu'en risquant de rompre les adhérences déjà formées, d'amener la luxation des vertèbres en conséquence des violents efforts qu'il faudrait employer, de parvenir même à la rupture de la moelle.

Néanmoins, si on parvient à surprendre la maladie à l'état intermédiare entre la période aigüe et l'ankylose formée, on peut s'attendre à voir, par effet de la suspension, le redressement de la position vicieuse des deux vertèbres qui font saillie et, en tous les cas, on redressera la difformité qui résulte de la position des autres vertèbres suivant un arc de cercle.

Toutes les autres ressources orthopédiques, depuis les béquilles jusqu'aux lits mécaniques, échouant toujours pour ce qui concerne le redressement de la colonne, la méthode de M. Sayre, c'est-à-dire, l'application de l'extension et de la contre-extension par la suspension occipito-mentonnière et par le poids du corps, est très-rationnelle, selon M. Dally. Le détracteur le plus acharné de la méthode américaine se plaît à l'avouer.

L'immobilisation du rachis par le bandage plâtré est le complément de la méthode de M. Sayre, et elle est aussi la partie la plus rudement blâmée des manœuvres mises en jeu pour parvenir au redressement.

Mais tandis que d'un côté on reproche à la méthode de ne trouver, ni sur le bassin, ni aux épaules, des points absolument fixes pour amener l'immobi-

lisation absolue de la colonne vertébrale, d'un autre côté, on lui reproche de trop immobiliser, de ne pas laisser libres, les mouvements des vertèbres saines, qui doivent par leur activité suppléer au défaut des mouvements des vertèbres malades de se procurer l'ankylose, ce qui est une erreur.

A quoi donc devrons-nous nous en tenir en fait d'immobilisation ?

La critique des uns répond à la critique des autres.

Il n'y a point d'immobilisation absolue. Donc, les dangers de l'immobilisation absolue ne sont qu'imaginaires.

Les résultats, obtenus par l'extension et la contre-extension, ne pourraient se mantenir qu'à l'aide d'une immobilisation, quand même incomplète et imparfaite. Donc, il faut mettre à profit cette immobilisation obtenue par le bandage plâtré.

Mais la cuirasse dure et légère que l'on obtient par les rouleaux plâtrés n'agit pas seulement par l'immobilisation qu'elle amène, cette immobilisation étant imparfaite, comme on vient de le dire. Son rôle principal est de maintenir le résultat de la suspension, c'est-à-dire, l'éloignement autant que possible entre les os du bassin et ceux des épaules, et, en conséquence, le redressement de la colonne obtenu par la suspension même.

Pour cela il suffit qu'elle s'appuie sur les os du bassin, malgré leur mobilité et que les aisselles viennent s'appuyer sur son bord supérieur. La cuirasse ne peut pas descendre, la saillie des os du bassin l'empêchant de se porter en bas, et les omoplates ne peuvent non plus vaincre la résistence qu'elle oppose à leur descente, les deux bouts du tronc ne peuvent plus se rapprocher, les épaules et les hanches resteront éloignées, autant qu'au moment de la suspension.

On a surpris alors les aisselles éloignées des os iliaques, le rachis distendu, et le redressement plus ou moins complet de la colonne obtenu ; on maintient ces résultats en prolongeant la suspension. Et si celle-ci rend des services, comme M. Dally le reconnaît lui-même, le bandage plâtré ne fait que devenir permanents ces avantages que l'on ne refuse pas de reconnaître à la suspension.

Si l'immobilisation parfaite et complète n'est qu'une pure illusion, si les vertèbres, dans leur cuirasse, peuvent se mouvoir, plus ou moins, au gré des mouvements du bassin et de ceux des épaules, comme chez tous les bossus, le centre d'action des mouvements du rachis se déplace, ou en dessus ou en dessous le point saillant, ou n'empêchera pas ces mouvements quand même on parviendrait à les amoindrir.

Mais s'il en est ainsi, la prescription des exercices gymnastiques, y compris la suspension journalière, est une puérilité inconcevable.

A quoi peut-il faire tort ce mouvement limité des vertèbres ? A quoi peut-il nuire, lorsqu'on blâme l'immobilisation absolue ? Comment pense-t-on obtenir la guérison ? Par l'ankylose ? Dieu nous en défende. Donc, ce ne sera que par la réparation des cartilages intervertébraux, ou des corps des vertèbres mêmes. Eh bien ! Le point du rachis où siège la lésion osseuse ne peut jamais devenir le centre de mouvement ; et en conséquence si l'on parvient à rompre les adhérences déjà formées, ou si elles ne sont pas encore formées, le redressement de la saillie peut s'obtenir, les stalactytes osseuses entre les deux vertèbres en angle peuvent aisément s'établir, tandis que toutes les autres vertèbres, en dessus et en dessous de la gibbosité, libres de se mouvoir, débarrassées du poids de la tête, peuvent suffire à leur réparation, et contribuer en conséquence au redressement de la colonne.

D'ailleurs, M. Dally, partant de ce fait que toute déformation chronique du rachis offre, comme indication première, une élongation de la portion de ligaments vertébraux raccourcie par adaptation à une attitude vicieuse, fait lui-même l'éloge de la suspension et de la contention par le bandage plâtré.

Pour ce qui concerne la suspension, M. Dally fait remarquer qu'il suffira

souvent d'allonger ces ligaments pour favoriser le redressement ; et la suspension y contribue puissamment.

A l'égard de l'application du bandage plâtré, lorsqu'on reconnaît qu'elle ne peut amener l'immobilisation complète, et en conséquence le danger de l'ankylose, on sera forcé d'admettre qu'elle favorise cette alongation des ligaments vertébraux raccourcis par adaptation à une attitude vicieuse.

Nous avons donc accepté la suspension préalable, le bandage plâtré et des exercices journaliers de suspension après l'application du bandage, et nous ne nous sommes nullement repentis de ces procédés.

Seulement nous avons reconnu l'inutilité de la suspension axillaire, qui rehausse les épaules sans rien apporter de profitable au redressement, surtout lorsque la gibbosité siège à la partie supérieure des vertèbres dorsales. D'ailleurs si l'on cherche à faire la suspension cervico-axillaire, comme les bras des malades sont tenus de se lever au-dessus de la tête, les mains saisissant la corde en cette position, il arrive que l'angle des aisselles s'efface tout-à-fait, et que les lacs de la suspension axillaire deviennent absolument inutiles.

La suspension mento-occipitale, lorsqu'elle est pratiquée avec tous les ménagements, suffit, et elle est la seule profitable, pour obtenir le redressement de la colonne.

Mais nous ne croyons pas qu'il soit nullement indifférent, de saisir un malade dont la colonne est courbée depuis quelque temps, de lui faire subir un redressement forcé en vertu de la suspension, et de maintenir tout de suite ce redressement par l'application du bandage plâtré. Il y a des relations vicieuses établies à la longue, parmi les organes de l'économie en conséquence de la courbure anormale de la colonne ; et il faut ménager ces rapports et ne pas vouloir, tout d'un coup, les contraindre à se transformer sous l'influence du redressement subit du rachis. Nous croyons même que la mort survenue à la suite des vomissements persistants, après l'application du bandage de M. Sayre, chez un malade, dont ce fâcheux résultat fut rapporté par M. Alfred Willett et arrivé au St. Bartholomew's Hospital, a été peut-être la conséquence de ce redressement violent et subit qui amena la dilatation excessive de l'estomac, cet organe ayant acquis l'habitude vicieuse d'être plié sur lui-même selon l'arrangement anormal des organes du thorax et de l'abdomen, déterminé par la courbure du rachis.

La prudence nous conseilla donc, et avec le plus heureux succès, de faire faire aux malades plusieurs exercices de suspension méthodique et journalière avant l'application du bandage plâtré.

Pendant une semaine à peu près nous ne faisions que suspendre nos malades par le menton et par l'occiput, d'abord rien que pendant quatre ou cinq minutes, ensuite successivement pendant dix, quinze, vingt minutes, jusqu'à ce que le patient pût se maintenir suspendu, sans être nullement gêné durant une demi-heure.

En agissant de la sorte nous avons l'avantage d'habituer tout doucement les organes à la position qu'on va leur faire prendre par l'effet du bandage, nous corrigeons peu à peu les relations vicieuses que les dits organes ont contractées, nous les ramenons sans violence et sans contrainte à la normalité qu'ils avaient perdue. En outre, nous parvenons à familiariser les malades avec les exercices de la suspension, dont ils s'effarouchent, de prime abord, et nous obtenons ainsi qu'ils puissent se maintenir dans la position convenable pendant l'application des rouleaux plâtrés, ce qui est un avantage qui n'est pas à dédaigner.

En même temps que les malades, surtout les jeunes gens, deviennent de cette façon, bien plus dociles, qu'ils se prêtent bien mieux à se laisser suspendre, qu'ils s'habituent à l'auto-suspension, en tirant eux-mêmes la corde et en levant leurs bras au-dessus de la tête, on obtient des garanties très-sérieuses contre la rupture

des adhérences déjà formées, contre les leuxations incomplètes des vertèbres, voire même contre la rupture de la moelle.

Les malades préparés d'avance avec ces exercices graduels et préalables, nous leur appliquons l'appareil de M. Sayre, selon les renseignements précis de ce professeur, et d'après ce que nous avons appris nous-mêmes en le voyant faire l'application d'un appareil à un jeune malade, à Amsterdam.

Il va sans dire que nous repoussons absolument l'anesthésie, par n'importe quel agent. On ne pourrait, par de telles démarches, beaucoup trop imprudentes, que s'exposer à étendre par trop la colonne vertébrale et dépasser le but ou se risquer de produire la syncope respiratoire et cardiaque, comme M. le professeur Boyer l'a fait remarquer.

Et que pourrait-on obtenir de bon par l'anesthésie des patients? La souplesse des ligaments vertébraux? La flexibilité des éléments qui forment la colonne vertébrale?

On obtient tout cela par les exercices méthodiques mis en usage une semaine d'avance,

S'agit-il d'une opération douloureuse? Point du tout.

Donc, à quoi bon l'anesthésie?

Pour obtenir la docilité de l'enfant ou pour vaincre la peur de l'adolescent?

Nous en venons à bout bien autrement par les exercices préalables, et il n'y à point de malade qui, au bout d'une semaine tout au plus, ne se soit familiarisé avec le trépied, avec les courroies qui lui tiennent la tête par l'occiput et par le menton, avec la corde qu'il tire à merveille. Et tout cet attirail qui l'effarouche au premier jour, dont l'aspect seul le fait fondre en larmes devient pour lui à bref délai un jeu qui l'amuse, auquel il s'adonne du meilleur cœur.

L'exemple donné par un autre jeune malade, instruit déjà aux exercices de l'auto-suspension, et la familiarité avec le médecin traitant, sont les moyens les plus sûrs de porter la conviction à l'esprit des jeunes gens, et de vaincre toute leur résistence à s'y soumettre.

Du reste les jeunes gens sont très-dociles aux exercices de la suspension, quelques jours passés; et la mise du bandage plâtré ne les effraie nullement. Il faut néanmoins les caresser et leur distraire l'esprit, pendant qu'ils sont tenus d'être couchés sur un matelas dur et uni, en attendant que la cuirasse vienne à desécher, pour se rendre assez solide.

Aussitôt qu'on les met debout, comme leur conditions d'équilibre se sont transformées, ils éprouvent quelquefois de la gêne à se maintenir en position verticale, de la difficulté pour marcher; et cela arrive surtout chez les jeunes malades, qui avaient contracté l'habitude d'appuyer une de leur mains sur leur genou pour pouvoir marcher, ou bien mieux chez ceux qui s'appuyaient de leur deux mains, ce qui n'est pas assez vulgaire d'ailleurs.

La cuirasse les empêche de porter la main sur le genou et ils s'épouvantent en se voyant debout et sans l'appui habituel, de telle façon qu'ils croient même qu'ils vont tomber en avant, et ils ne veulent point quitter ceux qui les sontiennent, de peur de se faire du mal dans la chute.

Le bandage gêne aussi, tant peu que ce soit, le mouvement des cuisses, et il presse sur les hanches de tout le poids provenant de l'appui des aisselles sur le soubord supérieur.

Mais dans peu de jours les malades s'habituent aisément à toutes ces petites contraintes, et, une semaine passée, tout au plus, on les voit courir, s'amuser, sans gêne et sans difficulté dans leurs mouvements, ayant tout-à-fait oublié leur ancienne habitude de chercher un point d'appui sur le genou.

Dans un seul cas nous avons reconnu que le bandage plâtré nuisait à la dilatation de l'estomac après l'ingestion des aliments malgré la précaution du

*deriver-pad*, bien que nous ayons l'usage de le mettre sur le sommet de l'estomac de tous nos jeunes malades.

Mais il s'agissait d'une jeune fille d'une constitution très-chétive, qui avait souffert des fièvres d'Afrique, et qui portait en conséquence la physchonie de l'estomac, s'accompagnant d'hypertrophie du foie et de la rate.

Cela non obstant, l'habitude a vaincu la contrainte première et une semaine après, la jeune malade pouvait faire l'ingestion des aliments sans gêne, et les digestions se faisaient régulièrement comme elles s'étaient toujours faites.

Pour ce qui concerne la respiration et la circulation, elles deviennent plus libres et plus régulières après l'usage de la cuirasse plâtrée, et ce n'est pas là un des moindres bienfaits de la méthode de M. Sayre.

Les jeunes gens se trouvent tellement bien à l'abri de cet corset dur et résistant, qu'ils le réclament, comme un soulagement reconnu, lorsqu'on vient à les en débarrasser pendant quelques jours pour leur donner des soins de propreté.

Voyons maintenant les désavantages inhérents à l'usage de la cuirasse.

D'abord, le bandage plâtré ne peut se maintenir en place, du moins dans les climats chauds, que pendant un mois tout au plus,

Nous avons essayé de le maintenir pendant un plus long délai, et nous nous en sommes repenti. L'accumulation de la sueur, la poussière du plâtre même ont produit des démangeaisons très-désagréables chez les jeunes malades, qui commençaient de maigrir et de devenir tristes et impatients à cause de ce désagrément. Ensuite les conséquences qui dérivent de la malpropreté du corps se prononçaient de plus en plus, une odeur fétide et insupportable même sortant de dessous l'appareil, ce qui était une atmosphère nuisible qui venait entourer les malades.

Leur odorat, leur gôut même en souffraient ; ils perdaient l'appétit, leurs bonnes couleurs s'effaçaient, ainsi que leur joie ; ils vivaient plongés dans la tristesse, cherchant en vain à se gratter, se dégoûtant de tout, sous l'influence d'un martyre perpétuel que leur procuraient les démangeaisons et la puanteur dont ils étaient entourés.

Nous avons donc reconnu le besoin de remplacer le bandage plâtré à de courts intervalles, d'un mois à peu près.

L'excoriation du point saillant de la colonne devient inévitable aussi, lorsqu'on n'a pas le soin d'y obvenir par le remplacement très-fréquent du bandage plâtré. Malgré la précaution prise d'éviter le contact du bandage sur la saillie vertébrale, en y formant une petite cage à l'aide de deux rouleaux de bandage placés à côté l'un de l'autre, de telle façon qu'ils fassent une saillie plus grande encore que celle de l'angle des vertèbres mêmes, on ne parvient pas à obvenir tout-à-fait au frottement des tours du bandage qui passent sur ce point saillant, ni à celui du gilet de fils de laine, quoiqu'il n'ait ni contures, ni boutons, ni plis ; et l'excoriation survient, cette excoriation étant d'autant plus difficile à guérir que les malades sont en général des gens chez lesquels le lymphatisme, voire même l'escrofulisme prédominent d'une façon assez remarquable.

Lorsqu'on s'obstine à maintenir le bandage, l'excoriation peut aller jusqu'à l'ulcération, et, alors, aux fâcheuses conséquences de la malpropreté, de l'accumulation des produits de la perspiration cutanée, des incrustations de la poussière du plâtre, on peut ajouter celles d'une suppuration, déguisée sous le bandage, sans qu'on y puisse porter les soins d'un pansement régulièr.

Si l'excoriation vient à paraître, et bien mieux encore si l'ulcération éclate, on est tenu de soulever l'appareil, et d'en débarrasser le jeune malade pendant plusieurs jours de suite, pour les applications convenables. C'est donc un temps perdu que celui qu'on croit épargner en maintenant trop longtemps la cuirasse plâtrée sans la faire substituer. Mais nous sommes parvenu à éviter

le désagrément de l'excoriation, en plaçant sur la saillie du rachis une pièce de sparadrap carrée, et taillée en croix de Malte, afin qu'elle puisse s'adapter parfaitement sur l'angle saillant de la colonne. Néanmoins et malgré même cette précaution il nous a fallu toujours remplacer le bandage à de très-courts intervalles, d'un mois tout ou plus.

Et il y avait là encore un autre avantage. Les bords de la cuirasse viennent à s'user par le frottement, quelle que soit la dureté que le plâtre ait prise, elle se ramollit ; destinée à maintenir l'écartement entre les hanches et les épaules, elle s'élargit d'elle-même, ou en conséquence de ce que le jeune malade vient à maigrir ; et alors elle ne serre pas assez bien le tronc pour assurer le maintien du redressement obtenu ; ou, par contre, elle peut devenir trop serrée jusqu'à produire de la gêne, si le malade devient plus gras.

La substitution fréquente du bandage y obvie à merveille.

Nous avons essayé de faire glisser du glycérolé phénique sous l'appareil, le portant aussi profondément que possible à l'aide d'une plume d'oie, dans le double but de corriger l'odeur désagréable et de guérir l'excoriation, mais nous n'avons obtenu rien d'assez avantageux de cette pratique, le remplacement de l'appareil étant la seule démarche qui nous ait fourni de véritables avantages.

Chez nous l'usage est admis d'envoyer aux bains de mer tous les jeunes gens chétifs, souffrants de lymphatisme, de scrofulisme, d'anémie, et il faut que le médecin traitant fasse des concessions à cette habitude populaire.

Il ne faut pas discuter ici ce que l'on peut obtenir de bon de l'usage des bains de mer comme ressource thérapeutique. Il suffit de constater le fait de l'habitude inveterée chez les familles de nos jeunes malades ; et quand même on viendrait à reconnaître que le bain de mer ne vaut guère que comme ressource hygiènique, que comme un moyen de faire respirer aux jeunes malades l'atmosphère saine des plages surchargées de brome et d'iode qui se dégagent des eaux, il y a raison pour avoir des raccommodements avec ce culte à peu près superstitieux pour la vertu des bains de mer.

Or, l'appareil plâtré entourant le tronc des jeunes malades, leur empêche l'usage de n'importe quels bains, puisqu'il devient impossible d'essuyer la peau, puisque les bandages mouillés pouvent prédisposer aux bronchites, aux pleurésies, aux pneumonies.

Mais lorsqu'on a obtenu quelque résultat de l'usage de l'appareil, c'est dommage d'en débarrasser les malades, pour leur permettre de se baigner, car si l'on n'a pas encore obtenu la consolation, si les cartilages intervertébraux ne sont pas devenus encore assez solides et assez résistants pour supporter le poids de la tête, la courbure revient sous peu.

Nous nous sommes tiré de notre mieux de cet embarras en commençant le traitemeut pendant les mois d'octobre ou de novembre après que les malades avaient pris leurs bains, car en agissant de la sorte nous avons devant nous dix mois pour amener la guérison de la gibbosité.

Nous avons dit que les jeunes malades étaient très-dociles à l'usage du bandage plâtré. Il n'en est pas de même pour ce qui concerne l'usage de la tige d'acier ou de fer se levant derrière l'appareil, se courbant au-dessus de la tête pour la maintenir en suspension occipito-mentonnière perpétuelle.

Lorsque la gibbosité se présente à la partie supérieure de la région dorsale, lorsque les vertèbres cervicales ont pris une position anormale, lorsque la tête est enfoncée entre les épaules, le bandage plâtré ne suffisant plus pour maintenir le redressement obtenu par la suspension, il faut avoir recours à la tige d'acier qui joue le rôle d'une colonne vertébrale supplementaire destinée à débarrasser les vertébres de la besogne de supporter le poids de la tête.

Mais les jeunes malades sont on ne peut plus indociles et rebelles à l'emploi de ce moyen. Ils se plaignent de bonne heure, ils deviennent tristes, ils ne

veulent plus bouger, ils ne jouent plus, et on est forcé de supprimer cette partie de l'appareil qui d'ailleurs est indispensable lorsqu'il s'agit des courbures siégeant très-haut ou donnant à la colonne vertébrale la forme d'une S.

La méthode de M. Sayre nous est donc restée impuissante dans ces cas, et nous n'y sommes parvenu qu'à redresser un petit peu l'atitude vicieuse du thorax, sans que nous ayons pu redresser la courbure même, ou vaincre la saillie des vertèbres dorsales, pas plus qu'à faire mettre en ligne les corps des vertèbres cervicales se présentant en concavité tournée en arrière.

La méthode de M. Sayre améliore tous les malades souffrant du mal de Pott; néanmoins elle ne les guérit pas en tous les cas, le redressement parfait de la colonne vertébrale ne pouvant s'obtenir chez plusieurs malades, pas même à l'aide de la suspension régulière exercée tous les jours.

A la période de consolidation des vertèbres il devient impossible de rompre les adhérences déjà formées, l'ankylose même, à n'importe quelle période ; elle est impuissante pour agir sur les difformités du rachis siégeant très-haut, à la region dorsale, ou à plus forte raison, à la région cervicale.

Cependent avec l'application de la cuirasse plâtrée selon cette méthode, on parvient assez souvent, presque toujours même, à transformer les bossus difformes en des bossus passablement élégants, car si la saillie reste, la courbure s'efface et la colonne devient, de la sorte, redressée en dessus et en dessous l'angle des vertébres, et en conséquence les pauvres malades peuvent maintenir la position verticale et ils n'ont plus besoin de s'appuyer de leur main sur leur genou.

Il résulte de nos observations que l'usage du bandage plâtré pendant six mois ne donne pas toujours des garanties suffisantes de ce que la colonne redressée ne vienne à reprendre sa courbure vicieuse.

Cela se comprend aisément. La saillie est le résultat du processus morbide qui a eu lieu au corps d'une vertébre; si l'ankylose est formée et si l'on ne parvient à rompre cette ankylose, l'amélioration n'a lieu qu'en conséquence du redressement des autres vertèbres, siégeant en dessus et en dessous le mal. On ne peut donc s'attendre à une consolidation semblable à celle que l'on se procure à la guérison d'une fracture osseuse. Ce serait ankyloser toute la colonne vertébrale. Ces vertèbres ayant pris une position vicieuse en conséquence de l'applatissement des cartilages intervertébraux ou du corps des vertébres mêmes, on ne peut pas s'attendre à les voir assez solides pour résister au poids de la tête sans que la nutrition y ait amené de nouveaux éléments, et sans que le long repos de toute fatigue de soutenir le crâne ne leur permette de se restaurer en effet de ce travail de nutrition.

Or, ce n'est pas là un travail qui puisse s'accomplir en des semaines, en des mois même, si la créature est chétive, si ses os sont mous et très-pauvres en phosphate calcaire. C'est ainsi que lorsqu'on supprime trop de bonne heure l'usage de l'appareil, la tête pesant tout de suite sur les vertébres mal affermies, les force à se courber tout comme auparavant; et il faut qu'elles soient assez solides pour ne pas céder au poids et pour ne pas reprendre l'attitude vicieuse qui leur est donnée par le besoin de chercher des conditions d'équilibre en compensation du deséquilibre qui leur vient de la saillie de la vertèbre où siègea la carie.

Si, par contre, les adhérences vicieuses sont rompues, ou s'il n'y a pas encore d'adhérences formées, il faut s'attendre à ce qu'elles se forment, à ce que l'ankylose s'établisse parmi les vertèbres saillantes, et dans ces cas mêmes, qui sont les moins vulgaires, l'ankylose ne se fait pas aussi rapidement que pourrait se faire la consolidation des deux bouts d'un os ayant souffert une fracture.

Des jeunes gens que nous avons débarrassés de leurs bandages six mois après les avoir portés nous les avons vus, se courber encore, redevenir, à peu près, à la position primitive et chercher le point d'appui sur leurs genoux.

Mais à la longue les vertèbres deviennent plus solides et elles sont à même de supporter le poids de la tête et la verticalité du tronc.

L'usage du sirop au lacto-phosphate de chaux de Dusart et de l'huile de foie de morue contribue puissament à la réussite.

Pour ce qui concerne les cas de scoliose, nous ne pouvons nous prononcer d'une façon très-positive, les observations nous faisant défaut, sur les avantages de la méthode de M. Sayre à leur égard ; mais nous croyons que lorsqu'il existe un développement inégal des côtes de chaque côté du thorax, la suspension ne peut y porter remède, et la compression circulaire du bandage entourant le tronc est insuffisante pour corriger cette inégalité de développement ; et en conséquence nous croyons bien moindres les avantages de la méthode de M. Sayre dans les cas de scoliose que dans ceux de courbure anormale provenant du mal de Pott.

Nous nous sommes imaginé de produire la suspension permanente en maintenant les jeunes malades, pendant toute la journée, sur une sorte de vélocipède, qu'ils feraient mouvoir de leurs mains et de leurs pieds, se tenant appuyés sur leur séant, presque dans la position verticale. De l'arrière de ce vélocipède se lèverait une tige de fer courbée en haut pour soutenir les courroies destinées à faire la suspension tout comme ou la fait au trépied. Et le soir on ferait coucher les jeunes gens soumis à ces manœuvres sur des lits appropriés où l'on pourrait maintenir l'extension et la contre-extension par des lacs élastiques.

Mais, pour en venir à bout il faudrait monter un établissement destiné à soigner les jeunes bossus, et y avoir des institutrices assez intelligentes et assez dévouées pour soutenir le moral des malades, pour les amuser toujours, pour les instruire tout doucement, pour leur égayer l'esprit, pour leur faire subir sans peine et sans contrainte le désagrément d'une position constante et d'une suspension permanente.

Les bossus ne sont pas assez nombreux dans notre pays, pour que l'on puisse se hasarder à une telle entreprise ; et les pratiques de l'orthopédie vulgaire y dominent encore tellement qu'on s'attendrait en vain à voir les familles se séparer de leurs enfants pour les faire entrer en pension à l'établissement où l'on essayerait une nouvelle méthode.

En conclusion : La méthode prônée par M. Sayre, qu'elle soit de son invention, ou qu'elle soit copiée d'anciennes méthodes, est digne d'être prise en considération, lorsqu'il s'agit de guérir ou tout au moins d'améliorer les malades souffrant de courbure vicieuse de la colonne vertébrale.

L'orthopédie classique y échoue dans la plupart des cas ; la méthode du professeur américain, quand même elle n'ait pas un plus grand succès, a l'avantage de rendre la respiration et la circulation plus libres.

Les inconvénients dont on l'accuse sont très-faciles à corriger, et de cette façon la méthode devient une ressource précieuse.

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## *Spinal Curvature in Children, with Special Reference to Sayre's Method.*

Mr. C. H. Golding-Bird, London.

While the mechanical method of treating spinal disease introduced by Sayre is applicable both to adults and children, it is in these latter more especially, I think, that the value of it is seen.

Of all physical means of restraining the movement of the trunk in children,

none surpasses the plaster jacket, while of all it is the least irksome—less so even than bed itself.

Of the four deforming diseases of the spine—excepting that from cicatrices or empyæma—three are found only in children—viz., caries, rachitis, "general curvature;" the fourth, mollities osseum or osteomalacia, does not concern us now, being an affection of adult life.

A rational treatment is arrived at only by a study of the etiology and pathology of these diseases; and though our knowledge on these points is imperfect, yet we know enough to be able to formulate some general therapeutic rules.

In past years too much attention has been given to the deformity of the spine, too little to its causes and accompaniments; while this fundamental fact must be admitted at the outset, that there is nothing special in these diseases when affecting the spinal column: they are the same as are found in other parts of the osseous system, and they essentially demand the same lines of treatment.

Therapeutically the diseases of the spinal column in children belong to one of two classes:—

*a.* The inflammatory, or caries—the same disease as is often seen in the cuboid bones of the body.

*b.* The non-inflammatory, or (i.) "general curvature," a weakening of ligaments and a yielding of bones, the pathology of which is not clearly understood; and (ii.) rachitis, which is a perverted osseous development, the bones being preternaturally weak.

Mechanical treatment, however perfect, is but half the surgeon's duty; due regard must of course be paid to medicaments and hygienic conditions, but these do not belong to my subject.

Inflammatory disease in the bones, or caries, wherever situated, requires that the bones affected should be immovable, free from pressure, in a state of "physiological" rest, and that pus should have ready exit if formed.

Some, but not all, of these requirements rest in bed will afford; but all, without exception, are obtained by the plaster of Paris jacket, with the additional advantage to the child of being able to take exercise in the open air.

The jacket referred to, as you all are aware, consists of muslin bandages steeped in plaster of Paris, moistened, and then applied closely over a skin-fitting merino shirt from the armpits to one inch below the anterior iliac spines.

The patient must be in a position, however, which shall ensure (*a.*) the chest being in a state of full inspiration—*i.e.*, the ribs raised; (*b.*) the inflamed vertebræ free from superincumbent weight.

Such conditions are best obtained either in "Sayre's swing" or by holding up the child under the arms whilst the bandages are applied. Some surgeons prefer a horizontal position, but the maximum benefit is not thus attained.

The various pads of wool employed to prevent chafing must be used according to the surgeon's experience.

If well and closely applied, the jacket is borne by very young children; I have used it in the infant under a year old. We all know the difference between an ill-fitting or good-fitting article of dress: when moulded to the form of the body it ceases to be felt, and only becomes uncomfortable and troublesome when from any cause it begins to press unduly on one part or the other.

It is the same here. Children cannot wear a loose jacket that shifts, however little; and this, in inexperienced hands, is often an unsuspected source of failure and annoyance. We all know how irksome were the old spinal supports of steel and leather to wear, however lightly made: they never formed an integral part of the patient's body, like the plaster jacket; and hence their dead weight, though perhaps not in excess of that of the jacket, told upon the wearer.

The jacket should be worn as long as it is comfortable; and obviously the rate of growth of the child is the chief cause for a change. In children two to three months is an average time for one jacket to last, but this may be extended in patients of ten years or upwards. The presence of a sinus necessitates a window in the jacket; but with an unopen "cold" abscess, no change beyond a protection of cotton wool under the jacket is to be made. I have seen an ilio-psoas abscess the size of the patient's head disappear without discharging while using the jacket in a girl of about nine years of age.

If the disease is in the cervical or upper dorsal regions, then the "jurymast" is to be employed if the child is old enough to walk; if not, or if for the time too ill to do so, then rest in bed, with sandbags, is undoubtedly preferable.

Also, if acutely ill, whether the disease be in the neck or back, rest in bed, according to ordinary surgical rules, must be first adopted. Sayre's method only becomes applicable when the acute symptoms have subsided.

To express *statistically* results of the jacket in children with carious spines is impossible—all the concurrent conditions cannot be reckoned;—but I can truly say that I have never known a case fail in showing results, the outcome of the "physiological rest" of the jacket, which were not incomparably better than any obtainable by any other mechanical means. All my results with the jurymast have been most satisfactory.

*Non-inflammatory disease of the spine.*—The full value of Sayrism in "general curvature" cannot be appreciated in the case of children only. The period of puberty and of young adult life furnish the most illustrative cases of all degrees.

Strictly, in childhood some gain may be hoped for as a rule in all non-inflammatory cases, inasmuch as they are of necessity, with one exception, in an early stage.

The worst non-inflammatory deformities in children are from rickets. This disease, if it affects the vertebræ, has ample time by, say, six years of age, to cripple as much as an old-standing case of general curvature later on in life; and such cases, with marked deformity, a "hump," from protrusion of the angles of the ribs backward and lateral curvature, can be benefited but slightly by any treatment.

Rickets seen early may be at once treated by the application of the plaster bandage, and a favourable prognosis may be given.

General curvature in children requires one golden rule to be followed out—"Never over-support."

Disappointment is met with, and Sayrism is abused, by the surgeon at once putting *all* his cases of general spinal curvature into jackets.

As in flat-foot we modify the support to the requirements of each case, so as not to take all the work off the adductor muscles of the ankle, so must the support be regulated in general curvature of the spine. Indeed, it is not uncommon to find curvature and flat-foot concurrent in the same patient, not, I believe, as cause and effect, but both as the expression of one constitutional condition.

In caries the keynote of treatment is "rest;" in general curvature or in rickets it is "restoration." The former cannot be done without mechanical appliance—*i.e.*, the jacket—unless the patient is to keep his bed. The latter is done primarily, *not* with the jacket, but with the extension of the tripod. The jacket is added, not according to the age of the patient, but the age and degree of the disease.

In cases of early lateral curvature, when the outgrowing blade-bone and elevation of the shoulder are the prominent symptoms—*i.e.*, where lumbar deformity has not become marked, and where rotation is not obvious, the jacket is not needed, such patients are cured by the use of the tripod exercise alone. The earliest age at which children show signs of lateral curvature, say six to seven years, is

none too soon for them to understand and employ this calisthenic exercise. I have employed it at that age with perfect success, and many times also at later periods with the same result.

The deformity of rickets, occurring as it does in infancy often, must have the support of the jacket, however slightly the spine may be affected.

The exercise, I need here only say, is that introduced by Professor Sayre. It differs from that of the hand swing or parallel bars, inasmuch as the patient exercises from the head and hands, and not from the latter only. The restoration of the spine to its normal line thus twice daily, and the regular work given to the dorsal muscles are the immediate causes of cure.

In cases fitted for treatment by the exercise, three to six months should suffice generally for cure. One of my cases, æt. 10, was cured under two months. In those cases in which the symptoms of lateral curvature, or indeed of rickets, are more severe than those enumerated already, where the lumbar curve and rib-rotation are both marked, or finally, where the disease has been a year or more in progress, then the support of the jacket must supplement the use of the exercise, and the concurrent use of both will attain a result unattainable by the separate use of either.

Even in extreme deformity in children, where no permanent spinal improvement is expected, this treatment of opening up the chest relieves the thoracic and abdominal viscera from undue pressure ; and thus, though indirectly, permanent gain to the general health is obtained. This I have often verified.

Whether applied for inflammatory or non-inflammatory disease in children, the jacket should be entire. The use of the cut-up felt is for many reasons not to be recommended. The mode of application of the jacket in general curvature is the same as that in caries.

In Sayre's method there is a great tonal gain. When Sayrism fails to give relief, there is no instrumental means that will carry the cure further. The right thing is to tell the child's parents so ; to order some reasonable support—in the way of stays—to keep up the good attained, and then to let the case be no longer an invalid in his surgeon's hands, but start life in the best way that his deformity will allow.

I repeat, though we cannot always promise a cure, I believe we can recognise, after a trial, the condition of incurability.

I am thankful to record my experience of Sayrism in the lateral curvature of children as always successful—never failing to give substantial relief, even where the case was past cure. These cases "past cure" will, I think, mostly be found to be rickety in origin,—not that the rickety condition is incurable, but since it begins to deform early, it has time, if not soon treated, to bring about terrible deformity even in the child.

Many of the complaints of failure in Sayre's treatment made to our journals show too clearly that the surgeon regarding Sayrism as a miraculous rather than a rational aid to a natural process of repair, has expected too much, and has only failed because he attempted to cure the incurable.

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## *On the Treatment of Spinal Curvature, with Special Reference to Sayre's Method.*

Mr. Henry F. Baker, London.

In the consideration of the treatment of spinal curvature with special references to Sayre's method, it will be necessary to divide the subject into several parts.

1.—The suitability of this form of treatment in cases of angular curvature.

2.—At what stage in the treatment of the curvature it should be made use of.

3.—Its suitability in cases of lateral curvature.

4.—Whether the whole method of treatment should be carried out, or only a part.

In the first place, with regard to angular curvature—is it the best form of treatment to suspend the patient, and to encase his body in the plaster jacket?

If the disease of the vertebræ is diagnosed by the surgeon before deformity takes place, I suppose that even Dr. Sayre would hardly recommend suspension, as it could obviously be of no service. If, however, the vertebræ have begun to fall together and cause the characteristic deformity, is it advisable, or, indeed, justifiable, to attempt at once to rectify the accident and bring them apart again?

This mode of treating caries of the spine by extension has been in use from the earliest ages, although not in the exact way suggested by Dr. Sayre, and it has been generally discontinued and condemned by modern surgeons, for the simple reason that in the acute stage of the disease, before anchylosis takes place, it is attended with danger, and also that in the more advanced cases it is of very questionable advantage. This is well shown in the case mentioned by Mr. Alexander Shaw in his article on this subject in Holmes' "System of Surgery."

In the great majority of these cases of angular curvature in which more or less anchylosis has taken place, suspending the patient, although harmless, is perfectly useless, and the increase in the patient's height, which undoubtedly takes place, is due, not to the straightening of the diseased curve, but to the extension of the compensatory curves, and also to the stretching of the healthy intervertebral cartilages, and its practice can only engender false hopes in the minds of the friends of the patient. If these views then be correct, extension, whether by suspension or by other means, should never be practised in cases of caries of the spine.

With reference to the use of the plaster jacket in cases of angular curvature, all surgeons, including Dr. Sayre, I believe, hold the opinion that the one essential point to attain in the treatment of this disease, before anchylosis takes place, is *rest*. If this desirable condition of rest can be accomplished by the plaster jacket, while the patient walks about or follows his usual employment, and, at the same time, if it does not interfere with the healthy development of the chest by compression, I think every one must agree that this is the thing that has been so long wished for—this is of all forms of treatment the best.

If, however, the structure of the spinal column be considered, as well as its relations to adjacent parts—viz., the chest and abdomen, and the duties these are constantly called upon to perform, it appears incredible that a bandage can be placed around them so as to obtain the required rest for the spine; and, indeed, according to my experience, it does not do so.

Again, if this bandage is kept on, as has been advised, "from one to two or three months," it must of necessity prevent the proper development of the chest, especially in weakly and sickly children, and in the case of adults it is difficult to understand how the pressure caused by it is less hurtful than that of ordinary stays, the ill effects of which are sufficiently recognised.

The idea, then, that *rest* can be obtained for the diseased vertebræ by means of the plaster jacket, or, indeed, any other contrivance, at the same time that the patient is allowed to walk about, is, in my opinion, an error. I venture to think that the only way to obtain rest is to keep the whole body in a state of recumbency.

In those cases in which anchylosis has begun to take place, although sus-

pending the patient can be of no use, I have seen cases in which the plaster jacket has given satisfactory support, and, without doubt, has helped to counteract the weakness that is always felt by patients for some time, even after the diseased vertebræ are anchylosed together. As this is the case, there are probably some cases amongst the very poor, and also in out-of-the-way districts, where its use may be indicated on account of its cheapness and the ease with which it can be applied. As it appears to me, however, that it cannot be kept on for any length of time without causing injurious pressure by preventing expansion of the chest, especially in the case of children, I consider that other forms of support, made either of steel or felt, which can be removed at will and enlarged as the child grows, are far better.

In the consideration of this subject in relation to lateral curvature of the spine, it appears strange that the same form of treatment that is advised for the cure of caries of the vertebræ should have been so confidently recommended for the treatment of lateral curvature, as the two affections are so entirely different in their pathology and progress. Angular curvature is always the result of one cause—viz., caries of the vertebræ, so that it is rational to treat all cases in a similar manner; on the other hand, lateral curvature is due to a variety of causes, and it appears somewhat irrational to suggest one form of treatment for all cases, and for all the different stages through which the deformity passes. For instance, in a great number of cases, in which a lateral deviation of the spinal column is observed, the cause will be found to be a tilting of the pelvis, the result of an inequality in the length of the lower limbs. If this deformity is observed soon after it takes place, the treatment is self-evident, and no spinal support of any kind is needed, and the encasing of the body in plaster or anything else must be useless and hurtful. In all cases of lateral curvature of the spine, the cause must be discovered before we can hope to carry out a successful course of treatment.

In cases of simple curvature of the spine without disease, extension by means of suspension or otherwise is useful, but in those cases in which a support for the spine is absolutely necessary, the plaster jacket is not so efficient as a properly adjusted steel support, by means of which the pressure can be regulated at any time by the surgeon attending the case.

I should not have ventured to express my opinion so positively in opposition to what I am aware is held at the present time by so many able surgeons, were it not the result of a considerable practical experience of these cases during a residence of five years at the Royal Orthopædic Hospital, upwards of 700 cases of different curvatures of the spine having come under my notice during that time. I may also mention the fact that I have myself applied the plaster jacket forty or fifty times, in cases both of angular and lateral curvature, during the period in which experiments were made with it at the Orthopædic Hospital.

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## *Note on Some of the Abuses of the Jacket Treatment of Spinal Disease.*

Mr. Walter Pye, London.

Although the time at my disposal will compel me to keep very closely to the subject I have in hand, I must in the first place guard myself against any suspicion of undervaluing the very great step which has recently been taken in treatment of the various forms of spinal lesion, and especially in the cases of

carious necrosis of the vertebræ, usually known as Pott's disease. And further, it is only right to acknowledge in the fullest possible way that it is due to Dr. Sayre's eloquent and forcible advocacy of the "jacket," especially of the plaster of Paris jacket, that, in this country at any rate, the general adoption of this method of treatment is due.

It cannot, at least, be said of us English surgeons that we were prejudiced against, or slow to adopt, a method of treatment, new or revised, because it came from the other side of the Atlantic, when we reflect how universal, in the course of four years only, the plaster of Paris jacket treatment has become throughout the country, and how on certain days in some of our children's hospitals we may see the floors of a ward strewn with small patients of various ages, laid out to dry in their shells, like so many half-finished mummies!

On the contrary, it appears to me that we have been, and are, rather too ready to box up a spine in a jacket as the only panacea for all the ailments and diseases it is heir to.

It is for this reason that I have been tempted to very briefly call the attention of this meeting to some points which in certain cases contra-indicate its employment, and to cases in which its use has been, I believe, not only useless, but actively harmful. I propose also to call attention to the principles which should guide us in constructing a spinal jacket, some of which, so far as my experience goes, are frequently violated, not without danger to the patient.

In the first place, then, I will speak of the discrimination of those cases which are proper for the application of a stiff jacket (whatever material may be used), and of those which are unfit; and in the second, of the modes of application of the jackets, and the risks attending this application if it be improperly or carelessly performed.

It would be mere waste of time and almost an impertinence to my audience if I were to do more than allude to what is, however, in my experience, the commonest error committed in selecting cases for jackets—namely, in mistaking merely rickety spines for cases of commencing serious disease. In some cases, no doubt, the diagnosis is obscure, but it may always be cleared up by watching for a short time. In most, the contrast between the careful rigidity of the true disease and the helpless flexibility and general slackness of the whole spinal column in the rickety child, is so strong, that even if tenderness along the spine be present, it is difficult to account for the error being so often committed.

However, it *is* made, and the consequences are serious enough sometimes. The yielding legs and pelvic bones, already overtaxed by the weight of the tumid belly and large heavy head, are made to bear no slight additional burden, with the result of increased permanent deformity, and very likely a permanent yielding or slackness of the vertebral ligaments and muscles.

The application of a rigid supporting jacket to cases of simple lateral curvature again appears to me to be not infrequent. Yet surely, if it should ever be done, its use should be confined strictly to those cases in which the deformity is so great, that the general health of the patient suffers from the altered anatomical relations and diminished capacity of the abdominal and chest cavities, and where, too, all chance of bracing up the spine by exercise and other means of improving its nutrition have failed.

In these extreme cases of simple curvature I believe a felt or plaster jacket to be better than any apparatus designed by an instrument-maker, but in these only. For while a chance remains of improvement of the muscular and ligamental nutrition, it can only be harmful to bar the door of hope in this direction by preventing all possibility of movement or exercise. Putting aside these two classes, about which I do not suppose there can be much difference of opinion

as to the unwisdom of encasing, I come to a class about which the sense of such a meeting as this may well be taken.

In cases of true Pott's disease, detected at or about its commencement, and before any noteworthy deformity has occurred, should a stiff and immovable support be at once applied or no? Probably the answer will differ with the age of the patient. It must be recollected that we are only here discussing the treatment of the illnesses of childhood; still there is distance enough between a child of nine months and one of say nine years to allow of a different answer being given in the two cases.

Probably the answer for cases which commence after the sixth or seventh year of life will be, that the sooner the support is applied the better, both for the patient's present comfort and for the probability of its ultimate recovery with the least possible deformity.

But what should be the answer in very weakly children, or in those of very tender age? Three lines of treatment may be adopted.

1.—The absolute horizontal position, prone or supine, with occasional counter-irritation, and endeavours to mitigate as far as possible the evils of a prolonged confinement in one position.

2.—A spinal support, presumably some form of jacket combined with the horizontal position.

3.—The jacket, without any confinement of position, or restriction of movement.

Of these three lines, for infants, I believe the last to be the worst. It is tempting, of course, to indulge in the hope of being able to see a patient through the whole course of an illness, such as spinal caries, avoiding the manifold ills of confinement to bed. But can any supporting jacket, put on such young and frail children, be expected to secure even tolerable immobility, and yet not be hurtful in the way of confining the chest and abdominal cavities, and of being by its weight a distressing cause of fatigue, if the patient is allowed to move about?

When we bear in mind that spinal disease (with most others) is not a mere local condition, but is the expression of a generally vicious nutrition; and remember, too, the enormous importance of the due development in infancy of the cavities of the trunk, for the future maintenance of even tolerable health, we may well doubt if it will not be best to tread the old paths in which our surgical fathers walked, and in the early stages of Pott's disease in infants and very young children, simply let the confinement be to the bed or couch, but to secure that the recumbency be absolute and long continued.

For the same reason, *i.e.*, the danger of arrest of development, I am inclined to think the second plan—confinement both to bed and jacket—to be unwise too, though here the fatigue of the apparatus is avoided to a great extent.

These suggestions apply only, as I have said, to *commencing* disease in very young children. Undoubtedly in them, as in other cases of Pott's disease, we may shorten the period of recumbency very greatly by the application of the jacket as soon as consolidation has begun to be established.

I come to another class of cases in which I believe the jacket, if applied at all, should be so with the greatest possible care; and its application followed by careful watching. These cases are the ones in which there exists any noteworthy amount of bronchitis, or in which there is evidence of tubercular deposition in the lungs, active or quiescent; or in which there is cardiac disease.

I can call to mind, and I know my experience is not singular, at least five or six cases in which exceedingly alarming and urgent respiratory difficulty has followed the putting on of the jacket; and two, in which death actually occurred.

In some of these cases, of course, the distress or disaster may have been caused by the jacket having been put on badly, a point I will refer to directly. This was not the case, however, in all, or most of them; and we should, I am very sure, bear in mind in similar cases, that even if there be no apparent respiratory distress before the jacket is applied, still if there be lung or heart disease present, the child may very possibly be without any reserve breathing capacity at all, and that a very slight embarrassment of respiratory movement may produce dangerous dyspnœa.

The last class of cases I will mention in which the jacket often I think does harm, and in which its use must be attended with great caution, are those which are complicated by any high degree of paralysis. Apart from the common sense difficulty of keeping the jacket and parts beneath it clean and sweet when there is dribbling of urine or incontinence of fæces, these cases are very liable to the extremely rapid formation of sores, somewhat akin to the acute decubitus of Charcot; and as these are very insidious in their origin, and often at first nearly painless, great destruction of tissue may take place before the jacket is removed on account of the offensive discharge, and the mischief is discovered.

With these I end the list of cases in which I believe that the employment of a jacket, be it of plaster of Paris or felt, or other material, is either directly harmful, or is only to be decided on and its effects watched with care.

It remains for me now to notice one or two of the more important ways in which the jacket is harmful, not because it should not have been put on, but because it was put on in the wrong way. I will not waste your time by discussing questions of mere detail in which errors are due either to want of common sense, or want of practice, but will consider those cases only, where the principle of the treatment appears to me to be wrongly conceived.

In the first place, plaster of Paris jackets are very often far too heavy. I have seen some on quite young children weighing as much as five and six pounds. No argument is needed to point out that the weight of the structure very seriously diminishes its utility. And it is, moreover, quite unnecessary. If care be taken that the jacket is really fitting, and that the bandages lie in the direction of intersecting loops or figures of 8, a very slight thickness will serve as an abundantly strong support. A more important question to settle, however, is that of the employment of the swing and pulley, and I should be glad to elicit some expression of opinion on this subject. I myself hold that in children the use of the swing is bad in practice, and that, moreover, it is founded on erroneous surgical principles.

The question resolves itself into one of the justifiability of forcible extension of the spine. I know that it is held out as one of the great merits of the jacket treatment that by it the angular curvature can be cured, and it is not at all unreasonable to suppose that complete suspension may sometimes succeed in breaking down adhesions between consolidating vertebræ, and in untwisting the rotated axis of the spine. But is this an end we are surgically justified in trying for? Surely if we consider what consolidation means, and what the destruction has been which has thus happily ended, we may rest satisfied with so applying the support, that there can be no increase of the deformity, and that the settling together of the vertebræ may be as thorough as possible.

Again, in children, the swing has always appeared to me to fail in one very important point—namely, in securing that during the whole time that the jacket is being put on, the walls of the chest be in a condition of extreme inspiration. I believe that in those cases in which this point has been neglected the respiratory trouble often occurs. The swing, with stirrups under the armpits, seems to me, through the yieldingness of a child's shoulder girdle, to pull up the

shoulders and displace the scapulæ, so that this inspiratory position is not attained. The swing also is an object of great terror and awe to most children, and although this point would not be worth considering if there were no alternative, still it may be borne in mind. If the idea of forcible extension be abandoned, the points to be aimed at in considering the position of the child at the time of the application of the jacket are—*firstly*, that the body shall be as far as possible in a straight line—all departures from this, which are due merely to weakness or slackness of ligaments, being remedied; *secondly*, that the chest shall be in a condition of extreme inspiration. These conditions can obviously be most easily fulfilled by the use of a single inclined plane, on which the patient may lie with his arms extended over his head, and there grasping something, say a rod. In this position a roller plaster of Paris bandage cannot be well applied, but if mucilage and plaster be used, and the bandage cut in strips in the way advocated by Mr. Walker, it will be found a very comfortable way of putting on a jacket, and it is, I believe, certainly the best position in which to apply any of the resinous felt jackets, now often employed.

For the ordinary roller plaster of Paris bandage I believe the best method is simply to hold the child up by the arms close to the shoulder, allowing so much weight to be borne by the feet as will prevent any undue extension of the spine. In this way the proper position of the chest walls and body is secured.

Of Mr. Davy's method of extension by swinging in a hammock in a prone position, I am sorry to have had no experience. It seems, however, hardly suitable to young children.

Lastly, it is difficult to insist too strongly on the importance of the jacket clasping the pelvic bones below, and following accurately whatever curve the spine may take. It is very common to see jackets looking, and being, in fact, just like so many tubs with their ends knocked out encircling the patient. Jackets of this kind can never fix the spine—indeed, they never can fit properly at all—and working up and down as they do with every change of the patient's position, they are a very fruitful source of sores.

I am very conscious that I have laid myself open to the charge of unduly insisting on petty details. My defence is, that they are all suggested by faults I have seen committed, or have committed myself. Moreover, the details of the surgery of adults may perhaps in children be regarded as having a greater importance when we recollect that we have in them to deal with a balance in which the question of life or death, of recovery or permanent hurt, are weighed in a far more sensitive way, and which is far more readily affected for good or ill, than is the case with grown-up people. "De minimis non curat lex" may be good law, but "De minimis non curat chirurgicus" is bad surgery in whatever sense we may prefer to translate it.

## DISCUSSION.

Mr. A. E. Barker, London: The purpose of these remarks is as follows:—To give an entire allegiance to the principles of treatment of caries of the spine formulated by Professor Sayre; to express further the belief that the method of treatment designed by the same surgeon is the best of any yet devised for carrying out those principles; to submit that when the application of the jacket has been followed by no benefit, it is owing to the fact that the details of application developed and insisted on by Professor Sayre have not been carefully carried out. Two or three conclusions have, however, forced themselves

upon me as the result of experience, which are at variance with those expressed in some of the papers read to-day:—(1.) That the corset is not suited to caries in very small children. (2.) That an actual improvement in the actual angle by suspension is not to be hoped for, or indeed aimed at, in view of dangers observed. (3.) That there are cases quite consolidated where the corset is often applied unnecessarily.

Dr. Martin Oxley, Liverpool: I consider Sayre's treatment is most satisfactory, and having had a large experience at the Liverpool Infirmary for Children, where every case of Pott's disease is put up in a plaster jacket, I have much pleasure in stating that I consider it to be the only treatment for this disease. When I first began to put on the jacket, I have no doubt that I frequently over-extended my patients, and it may be possible that I did harm, not good. For this I must say, with all due deference, Dr. Sayre is in some measure to blame, owing to his having demonstrated the extent of curve before and after extension. The points in the treatment which require careful attention are as follows:—All prominences ought to be covered with a pad of cotton-wool in the form of a ring. We must not over-suspend, as the object of the suspension is simply to take the weight off the diseased vertebræ, not to straighten the deformity, although, as a matter of fact, we do straighten the spinal column as a whole; for the patient, instead of bending forward, as is usual, and leaning the hand on the knees, or on some object near, such as the back of a chair or table, has the whole body above the disease supported by the articular processes and the pully. In conclusion, I would say that so far I have not seen any case of disease of the spine that I would not treat with a plaster jacket. To my mind, the most important point in the treatment is the long-continued rest which a jacket, worn for half a year or more, gives to the diseased vertebræ. On this account I would strongly insist upon keeping the jacket whole, not slitting it up the front, as is too often done; for when a jacket is cut, we must, in order to make it a support, bind it firmly on the body, compressing the chest, and doing away with the space in front left by the removal of the inner pad. Patients may be kept perfectly clean for months by changing the Sayre's singlet every week, or oftener, in the following manner. Before applying the plaster jacket, put on two of Sayre's singlets instead of one; when the change is necessary, tack a clean singlet to the lower edge of the singlet next the skin, then, by drawing the latter (the soiled one) up over the head, the clean one easily follows and takes its place.

Mr. Edmund Owen, London: The position which a not inconsiderable experience with the plaster of Paris treatment in the out-patient department of the Children's Hospital has led me to assume may at first sight appear anomalous; for though with certain modifications I have carried it out for some years, and though I hope still to employ it, yet I do not consider it to be the best method of dealing with cases of spinal caries. I welcomed the Sayre treatment because I saw that it would afford us the means of dealing with spinal caries on the same principles as those which influence us in the case of caries of the tarsus—viz., of ensuring absolute physiological rest; also I read in the plaster scheme the death-knell of the expensive, heavy, and often useless mechanical supports. Certainly, since Professor Sayre's demonstrations my own cases of spinal disease have had a much happier time; but I think that the introduction of the method has suffered from the too great enthusiasm with which it was surrounded. One was led to suppose that there were to be no more angular deformities, and that those which had occurred were to be straightened out, "unless nature had already thrown out ossific matter, and adhesions were

beginning to form; that then they were not to be broken up by too severe extension." ("No, no," from Professor Sayre.) Those were the Professor's own words, and surely they implied that a straightening out of many angles might be expected. I have given up all suspension, and I am now relieved from that anxiety which must attend the suspension of a child with an ulcerated spine. I am glad to hear gentlemen confessing that such accidents were within their knowledge. Had I a child of my own the subject of commencing spinal caries, I would not put him in a plaster of Paris jacket, but I would keep him flat and absolutely quiet in bed for months or years, until the disease was quiescent; only then would I encase him in a firm support, and have him moved about. I gratefully accept Dr. Sayre's method (without suspension) for my hospital out-patients, because it affords them the nearest approach to physiological rest which their domestic surroundings permit.

M. Dally, Paris: According to my experience, the suspension acts in a very limited manner, and only when the patients are not entirely suspended. If they are raised from the ground, it is the muscles of the neck that are elongated, and not those of the back proper. If patients are not entirely suspended, and if they are directed not to make any muscular resistance, no doubt then some elongation can be obtained by the suspension. In cases of rotatory deformity, in cases of anchylosis between the vertebræ, and in the costo-vertebral articulations, suspension cannot effect elongation. In the latter cases it is only by proper training that any modification in the shape of the spinal column can be effected. In the early stage of vertebral caries, vertical extension may certainly be of great service by removing the weight of the upper part of the body. But when the torsion is confirmed, how can one reasonably hope that vertical extension can bring about a cure. It is impossible both on mechanical and physiological grounds. If stretching *were* possible in cases of caries, death would result, and in a few cases has occurred. Fortunately suspension is limited by the muscles of the neck. I am pleased that extension is not practised so much as formerly, and that the best part of Sayre's treatment is being retained.

Mr. Bernard Roth, London: I cordially agree with everything that has been said in favour of Dr. Sayre's plaster jacket for the treatment of spinal caries, and I have never had any sores or other discomforts resulting from it in the forty or fifty private patients I have thus treated. In almost every case, without exception, the patient has assured me how very much more comfortable he or she felt the moment the tripod was left with the jacket on. I beg to thank Dr. Oxley most heartily for the very useful hints he gave us yesterday about changing the vests under the jacket, which completely removes all objection to Sayre's jacket on the score of cleanliness. With reference to lateral curvature of the spine, I am sure Dr. Sayre has been led too far by his noble enthusiasm, and that his teaching will do much harm. It is very difficult, from what has been both written and spoken by Dr. Sayre, to understand to which stages of lateral curvature he refers when he speaks of some brilliant cures by his method applied to lateral curvature. I constantly examine cases of lateral curvature which at the first glance appear very badly deformed, with scapulæ very unsymmetrically placed—one hip unduly prominent; yet I have found on careful examination that it is quite possible to restore the apparently deformed spine back into a perfectly normal shape, and for the hip to retain this position for a few seconds; the fact being that there was no real osseous deformity, only a relaxed vicious condition of the bones, ligaments, and muscles. These cases are claimed as *cures* by Sayre's treatment, but there never was any deformity

present. I challenge Dr. Sayre or any of his disciples to exhibit at a Medical Society here a case of lateral curvature with real osseous deformity of the vertebræ and ribs, even to a very moderate amount—but it must be there—and then to exhibit the case again within six or twelve months as being cured by his treatment. Daily suspension by the head in the tripod does not strengthen or exercise the muscles, as maintained by Dr. Sayre; it is against common physiological sense. If you wished to strengthen a weak and flabby biceps muscle, surely you would not put it in a plaster bandage with the elbow extended to its full extent, and then hang the patient repeatedly daily by the hand of the imprisoned limb. I have now seen several patients with osseous lateral curvature who have been daily suspended by the head for months, and if Dr. Sayre's theory were true, at all events the neck muscles should have been strengthened, as they are most severely acted upon by the extension from the weight of the body; but nearly all of them poked their head as much as ever they did, I was informed by the friends. If you really wish to strengthen the muscles of the spine by exercise, you must put the patient in such a position that these muscles are called into play. Let the patient lie on his stomach (prone) on the ground, and while the pelvis is held, request him to raise the head and upper part of the trunk a few inches from the ground without the aid of his arms, and the whole mass of the erectores spinæ are bound to come into action. Sayre's jacket is only of use in lateral curvature in cases of extreme osseous deformity in supporting and giving relief to the patient, and in preventing the deformity becoming worse, also in cases of paralytic lateral curvature; short of this I am convinced it only does harm, like the ordinary steel spinal supports with crutches, which I am glad to see are being more and more condemned by most general surgeons, except those specially interested in them.

Mr. KEETLEY, London: I have suspended patients chiefly for the application of plaster cases, but partly for those of poro-plastic jackets, and to a lesser extent for mere purposes of measurement, over one thousand times, and with no worse result than an occasional tendency to faint on the part of adults. And I have not met with a case of fainting for nearly a year, nor do I expect to again, now that experience has brought greater expedition and a quicker perception of the signs of approaching trouble. Cleanliness may be secured with the use of the plaster jacket by wearing two linen or cotton handkerchiefs beneath the jersey next the skin, one anteriorly, the other posteriorly. These are changed by stitching the dirty one on to the clean one which is to take its place, and then using the former to drag the latter in. This is a similar method to that by which it appears Dr. Oxley changes his inner jersey. The handkerchiefs take up no perceptible room, are too thin to make any uncomfortable wrinkle, exercise no elastic pressure on the mammæ, are smooth, and are always readily obtained. The plaster case is just as removable and reapplicable as any instrument, but—and this is a great advantage—only when the surgeon wishes it. To change the plaster case from a fixed to a movable one, it should be neatly sawed up the middle in front, and then a *triple lace* applied. Holes are bored along each side of the opening, and a separate cord laced up and down so as to make a row of loops on each side. These loops are permanent. The jacket is then reapplied, and a third movable lace is laced in and out the loops of the two fixed laces. Sores and excoriations are best avoided by kneading the case to make it fit accurately all prominences. The plaster jacket can be made at once light and strong by building it up on the principle applied by Fairbairn and Stephenson to the construction of the Menai tubular bridge, and of hollow girders in general. The felt jacket, though intrinsically inferior to the plaster one, is very useful, because it is convenient and economical of time.

Dr. DIVER, Kenley: I have had some opportunities for forming an opinion upon Sayre's method, though not approaching the large experience of previous speakers. But I have seen enough to lead me to form a distinctly favourable opinion of the process. I have used it in cases where the patient was by it increased in height, made distinctly comfortable, and every one concerned with the patient more happy. Sayre's treatment is not only better but much less costly than the old spinal support plan. For as many pence as these instruments cost pounds a better result can be brought about. With the steel instruments the history of the patient is, that after a few days' wear of them a screw becomes loose or lost, and the apparatus has to be taken off and sent to the machinist for repair and await his convenience to put right. Then in a week or two perhaps a steel support gives way, and another period of time is lost while the apparatus again goes to the maker. So expense and loss of time are brought about. I would ask Professor Sayre one or two questions which were suggested by difficulties in carrying out the treatment. One, whether it is possible to adopt a method of moistening the roller without on the one hand soaking the roller unduly, and on the other of losing much of the plaster by its falling from the bandage? Also what method is best in evenly distributing the plaster over the bandage? Also the best way of opening the apparatus without the dust of unwinding? Whether Dr. Sayre will say within what age his plan is likely to be useful? Also whether there are cases in which his treatment is unadvisable if not inadmissible? In short, if he will help us in eliminating unsuitable cases from the employment of his method? It cannot be that Dr. Sayre's treatment is commonly injurious, as some have said, for it is impossible for men who have had experience of hundreds of cases to speak with almost unrestricted praise of a method, if that method, when properly used, be harmful.

Mr. J. H. MORGAN, London: Nearly all previous speakers have referred to the danger which may attach to suspension, and the necessity of modifying the swing apparatus of Sayre. Why suspend these patients? the speaker ventures to ask. Is the position of the various elements of the spine when the body of a patient is suspended in that condition of physiological rest which it is the object of the plaster of Paris bandage to maintain? Surely the muscles and ligaments whose function it is to keep the various elements of the spine in such a relationship as will support the superjacent weight are in anything but a state of rest when the body is suspended in this way. The only conditions under which all the elements of the spine can be at rest are when the body is recumbent. If, then, the spine is fixed by the plaster of Paris bandage—for the use of which in such cases all credit is to be given to Dr. Sayre—the spine is immovably maintained in a position of rest, and weight is removed from it and transferred to the bandage.

Professor SAYRE, New York: My whole mission this year is to correct, if possible, some of the errors which many distinguished gentlemen have made in my treatment. I thought that my directions were so simple and plain that they could not be misunderstood; but I find this is not the case, as I see that even so distinguished a man as Professor von Langenbeck has used extension (in my judgment) to a rather dangerous extent, and even recommends the use of an anæsthetic during the suspension. With all due deference to the opinion of so distinguished a gentleman, I must strongly protest against the use of any anæsthetic while the suspension is being made; and I beg any gentleman, if he does use chloroform or any other anæsthetic, not to call the treatment by my name, as I do not approve of its use, and am unwilling to be held respon-

sible for its employment. I make these remarks thus strongly because I notice in various medical journals a report of the proceedings of the German Surgical Society, in which Professor von Langenbeck reported a fatal case occurring in his clinic, and some of these journals have headed the article with leaded type as "*Fatal Result from the Application of Sayre's Jacket.*" Now the fatal result was not from the application of the jacket, but from rupturing of an abscess by the suspension of the patient, the suspension being made under chloroform, in direct violation of my teachings; and therefore I do not think my method of treatment should be held responsible for such a result. In my work on "Spinal Curvature" (Smith, Elder, & Co: London, 1877), pp. 21, 22, will be found the following definite instructions. "Before narrating the cases illustrative of the principles of treatment here advocated, I wish to give, or rather repeat, a word of caution, fearing that I may not have enforced it with sufficient distinctness already. It is this: *Do not attempt the impossible*—do not try to straighten curved spines, the result of caries, that have become partially or completely consolidated. If nature has already thrown out ossific matter and adhesions are beginning to take place, *do not break them up* by too severe extension, but simply extend the patient very slowly, so that the contracted muscles alone will yield, *until the patient says he feels comfortable, and never extend the patient beyond that point.* If it is a child who cannot talk, watch his countenance, and as soon as the expression of *pain* is changed to one of *pleasure, there stop*, and *secure* your patient by the plaster bandages, keeping him in that position until the plaster has set; he will then retain this sense of comfort so long as the bandage is properly adjusted." There, gentlemen, I don't know how to write any more distinctly than that, and yet I have been compelled to cross the Atlantic this year in order to impress this principle of treatment upon the professional mind—namely, giving your patient perfect comfort and freedom from all pain. This is to be done by making extension just sufficient to take off all pressure from the inflamed parts, and then retaining the parts in the position by the plaster bandage. If the patient is under the influence of an anæsthetic, it is impossible for him to tell you when you have extended him to the exact place desired, and no one else can; it should therefore never be used in the suspension of a case suffering from Pott's disease under any possible circumstances. There is no pain whatever given by the *proper* application of the suspension, and therefore no anæsthetic is required to relieve it. On the contrary, the proper application of extension gives immediate relief to the patient, and this is the invariable testimony of the many hundred cases in which I have employed it. I prefer to make the extension in the vertical position by suspension because it is more easily managed, and while in this position I can apply the plaster bandages better than when the patient is in the recumbent position. But suspension is not absolutely necessary to carry out my principles of treatment, for extension can be applied in the horizontal posture, as advised by Mr. Walker, or in the hammock, as advised by Mr. Davy; but neither of these methods is so convenient or so easily managed for both surgeon and patient as the extension by means of the pulleys with head and axillary straps. The objection to Mr. Davy's plan has been very well pointed out by Dr. Oxley of Liverpool in the July number of the *Liverpool Medical Journal*—viz., the danger of over-distension by curving the body too much backward; and he therefore suggests cutting holes through the hammock for the legs to protrude. I have never had any difficulty in applying suspension, even in the youngest cases. They often cry while being undressed for the first time, and having the skin-fitting shirt, dinner pad, &c., properly adjusted, particularly if they have already been in the hands of other surgeons, and have had instruments applied, as they think some new torture is to be inflicted; but so soon

s the head and axillary straps are properly adjusted and the extension properly nade, they cease crying almost on the instant, and give a deep, full, diaphragnatic respiration in place of the short, grunting, catching breathing they had before the extension was applied. This was very markedly the case in the ittle patient which Mr. Davy saw in my office, and which he has described as uffering such torture from the suspension, whereas the instant the extension ad reached a certain point the child ceased crying, and her face was entirely elieved from all expression of pain. But Mr. Davy's feelings were so touched y the previous crying of the child that he turned from it to examine some ictures in the office, and thus lost the important moment of observation, when he "countenance changed from pain to pleasure," and he has thus innocently reresented the tortures and pain of suspension, not having observed the instant reief to the pain when the suspension had reached the proper point. In a very few ninutes the jacket was applied by my son, and the child, almost immediately fter being laid on the air-bed for the plaster to "set," fell into a sound sleep, uch as the mother said it had not enjoyed for months. I have seen a number f cases go to sleep while the jacket was being applied, and with children as a ule they go to sleep while the jacket is hardening or getting "set." The only reatment to be adopted in cases where the jacket is inadmissible—as in very mall children or some cases of abscess—is the horizontal position; but even when the horizontal position is assumed, it must in many cases be supplemented y extension, to overcome the tendency to reflex muscular contraction induced by he inflamed vertebræ. I have seen many cases that have been confined to the orizontal position for years, and yet the disease was still progressing, and in nost of them there was marked deformity; and I have seen complete recovery with consolidation in a great number of cases in less than a year, and with ery slight, and in some cases no, deformity whatever, with extension and the laster jacket; so that the prophecy of Mr. Furneaux Jordan, that "the days of he humpback were ended," is not, after all, so visionary. If we diagnosticate he case early, and treat it *before* the deformity is commenced, we can in he majority of cases effect a cure in from eighteen months to two years, and with very little or no deformity perceptible. If, however, the deformity is llowed to occur, it will remain permanent; and although you will hasten the onsolidation, and thus effect a cure more rapidly and with more comfort to he patient by the method I have suggested than by any other mode of treatnent, still the deformity will remain just in proportion to the extent of the estructive process that has taken place in the bodies of the vertebræ before the reatment was commenced. It has been asked at what age should the treatment e commenced. I answer, at any age. If a person of seventy breaks a limb, ou certainly try to repair it the same as in a younger person; and the same ule holds here. In children too young to walk it is unnecessary, as they can e carried outdoors in the wire cuirass; but as soon as they can walk, the laster jacket is better, as it enables them to take free exercise in the open ir, which improves their digestion and powers of nutrition, and this is the ssential element of cure. When the disease is in the cervical or upper orsal vertebræ, the "jurymast," or head-rest, is absolutely essential, and I am satisfied that the suggestion of Dr. Samuel Grey of Philadelphia, hat it should be used when the disease is even lower down, is a good ne, as it relieves the parts from the pressure of the superincumbent weight f the head. I have applied it in many cases of two years, and in one f sixty, and one—an old Irishwoman at the "Home of Incurables" in New York, paralysed in lower extremities, and confined for nearly three years to bed—by making gentle extension from the feet, while Dr. Jones, the resident physician of the Home, sustained her under the axillæ, she almost

immediately said that she could feel in her toes. This settled the question that sensation was interfered with by pressure, and she was suspended while sitting in the bed, and a jacket and head-rest applied. In three weeks she was so much improved as to be able to stand, when a new jacket was applied, which was much better fitting, as she was erect during the application; and in one month she was able to walk with a stick, in six months walk without any assistance, and is now perfectly well. Objections have been made to the want of cleanliness. This is easily obviated by the suggestion of Dr. Oxley of Liverpool, by using two knitted jackets, the under one being pulled out when necessary, after stitching a clean one to it, which is thus drawn into position. There has been a mistake in putting Pott's disease and lateral curvature together in the same discussion. One is a disease producing deformity the result of inflammatory disintegration, the other is a distortion the result of unequal muscular contraction. Mr. Adams says, "It is as bad for lateral curvature as it is good for Pott's disease." This I am confident is a mistake. Lateral curvature, being the result of debility, requires gymnastic exercise to develop the muscles, and self-suspension, as suggested by Dr. Brig Lee of Philadelphia, is the best. This, with "Massage" electricity and general toning up of the system, is all that is required, and the jacket is unnecessary. But in advanced cases the jacket is requisite to retain the benefit obtained by self-suspension. When the jacket is thus applied, the patient is to continue the self-suspension twice a day, until the body is so straightened out as to receive no support from the casing, and then the projecting hump is to be pulled off from the shirt, and the triangular portion thus cut away is covered over with a fresh roller of plaster bandage after the patient has suspended herself, thus securing again the body in the improved position. This process is to be renewed as often as necessary, until the patient is improved as far as she is capable of being, and then the jacket is made into a corset, and worn as an ordinary dress corset; and being made of plaster bandage *on the body of the patient*, is more accurate in its fit than any other apparatus that can be made. I have now treated eighty-seven cases of lateral curvature by this method since January 1, 1877, thirty-nine of which are cured, and forty-eight are still under treatment. Several of these cases are perfectly cured, and are straight, as will be seen by these photographs, taken before and after the treatment. All of them present better results than I have ever been able to obtain by any other method.

A large number of photographs to prove the accuracy of his statements was then exhibited.

The Surgical Vice-President, Mr. Holmes, London, at the request of the President, summed up the debate. He said that the following appeared to him to be the main conclusions arrived at:—(1.) Nobody seems seriously to contest the priority of Dr. Sayre as the introducer of the method. What traces there may be of somewhat similar treatment in the hands of former surgeons are not more than the resemblances always met with between our predecessors' ideas and those of our own day. (2.) The discussion had been obviously imperfect in respect to other spinal curvatures than that from caries. In fact, it would have been better to restrict the discussion from the first to angular curvature. (3.) The debate had not enabled us strictly to define the class of caries in which the treatment might be considered as more especially indicated. Most of those who recommend it agree that the earlier it is employed the better; but we are still unable to say whether and how far symptoms of decided spinal irritation or inflammation are to be taken as contra-indicating it. (4.) Only a small minority of the speakers reject the method entirely, and believe that confinement to bed is preferable. The opinion of the

great majority seems to be, that in at any rate a very large proportion of cases of spinal disease, this method offers very large advantages, both local and general. (5.) It results from this discussion that no form of extension, whether by suspension or otherwise, is to be regarded as a necessary feature of the treatment. The jacket can be applied either in the suspended, the erect, or the horizontal position. (6.) There appears no evidence that any actual straightening of the spine has ever been produced. Dr. Sayre has strongly dissuaded the attempt to straighten the spine when there is any reason to think that the bones are at all consolidated together; and although he said that if applied before any deformity existed, his method would prevent its occurrence, he seemed quite uncertain as to the possibility of redressing even slight and commencing deformities. (7.) As to the question whether other plastic materials would not do as well as plaster of Paris, this discussion seems to show that there is no reason why such should not be the case, though Dr. Sayre and most of the speakers seem to prefer the plaster. (8.) An important point had been brought out as to the possibility of changing the inside shirt without removing the plaster case. This is not only convenient for cleanliness' sake, but may in some cases be a matter of serious practical importance. (9.) That there are drawbacks to the method in the shape of ulcers, abscesses, &c., seems not only possible, but inevitable. The extent and nature of such drawbacks should be clearly stated. They form no radical objection to the treatment. (10.) The average length of time required for cure is a matter of very great importance. It will probably be found to be much less than in the treatment by rest in bed. (11.) Finally, the general opinion seems to be that this is a real and a great advance in practical surgery.

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## *On the Nature of the So-called Scarlet Fever after Operations.*

Mr. Howard Marsh, London.

In 1864, Sir James Paget gave a clinical lecture on scarlet fever after operations, in which he related the case of a boy, who, on the day after he had undergone lithotomy, had an eruption exactly like that of scarlet fever, and a month later a considerable hæmorrhage from the kidney, followed in two days by sore throat and a return of the rash, and subsequent desquamation.

After stating that in the previous year, when scarlet fever was very prevalent, he had seen six cases following operations in private practice, Sir James Paget expressed the opinion that there is something in the consequences of operations which makes patients peculiarly susceptible to the influence of the scarlet fever poison, and added, that in such cases the disease undergoes certain modifications: the incubation period is shortened; the rash is irregular; in some instances there is no sore throat; and in some no desquamation; while in others sore throat and desquamation alike are absent. Since this lecture was published the subject has been carefully studied, and the opinion of Sir James Paget has been so fully confirmed, that this peculiar liability is established to the satisfaction of a large number of skilled observers.

It may, however, be well on the present occasion to state concisely the evidence on which this conclusion rests. For this purpose I will turn to the Hospital for Sick Children, for in its records convenient illustrations can be found of all the points that I shall have to mention.

1.—In the original building, consisting of two large dwelling-houses, the

wards, containing both surgical and medical cases, were on the first and second floors, while immediately above there was a fever ward, containing six or eight beds.

In order to prevent contagion stringent regulations were in force. Double doors were placed on the lobby at the entrance of the fever ward; the fever nurse had rooms on the same floor, and all intercourse between the fever and the general wards was as far as possible cut off. There was, however, only one staircase for all the wards, which was unavoidably used for the conveyance of the patients and their linen, diet, &c., and by the medical officers, and other officials. The fever ward was rarely empty, and often contained four or six cases of scarlet fever or its sequelæ. In these circumstances a state of things existed equivalent to a series of epidemics of scarlet fever; while the occasional inadvertent admission of a case into the general wards or the outbreak of the disease among the medical patients told in the same direction. Under these conditions the appearance immediately after operations of what all who saw the cases regarded as scarlet fever was frequently observed. Of forty-three cases of lithotomy by Mr. Thomas Smith, seven—that is, nearly 17 per cent.—were attacked, and so were many in whom other operations were performed.

But since the building of the New Hospital, the removal of the scarlet fever ward to an isolated block, and the separation of the medical and surgical cases, the disease in question has become very rare.

2.—Although children attacked with the disease under consideration were placed in the fever ward, in which the poison of scarlet fever must have existed in a concentrated form, they suffered no further infection.

3.—In some instances the features of scarlet fever were clearly marked, and the disease was of so severe a type that it proved fatal, either primarily or through its sequelæ. In all Mr. Smith's cases the rash was present, while in three there was desquamation, and in three severe albuminuria.

4.—The fact that the question has been considered by what I may term a jury of experts, composed of Sir James Paget, all the staff of the Children's Hospital (with whose opinion I am acquainted), Mr. Stirling (who has written a very able paper in the St. George's Hospital Reports), Dr. Goodhart and Mr. Paley of the Evelina Hospital, M. Trélat, and others, and that all have expressed their opinion that the disease is scarlet fever, goes far, I think, to carry conviction with it.

And here I would, in passing, observe that a belief in the peculiar liability of surgical patients to scarlet fever does not in any way clash with the opinion that eruptions of a non-scarlatinal origin occur after operations. Such rashes, whether produced by some form of blood-poisoning or by drugs, are undoubtedly met with, and have been already carefully observed and described. But I shall pass them by, for they lie beyond the range of the present communication.

This peculiar liability to scarlet fever has been manifested after every variety of operation, from the most trivial, such as tenotomy, the aspiration of an abscess, or the removal of a small sequestrum, to such as lithotomy, resection of the large joints, or amputation. Some observers have conceived the liability to be greater after operations on the genito-urinary organs, but facts are, I think, wanting to confirm this view. Assuming, then, that this peculiar liability may be regarded as established, the question now arises, Can this fact be explained? The one constant element in the problem is the existence of a recent wound. Let us, therefore, follow the subject by examining the action of the wound, first, in its purely local character, and, secondly, in reference to any influence it may exert on the general condition of the patient.

1.—It has been suggested that the materies morbi is conveyed by the hand

or instruments of the operator—that the process is one of true inoculation. Such an hypothesis is, at first sight, plausible; inoculation is a method by which poisons are readily introduced, and one which conduces to their rapid action. But the inoculation theory seems to derive no confirmation from the facts of the case. Operating surgeons are so rarely brought into contact with scarlet fever that it may fairly be held they rarely convey it to their patients. And in the Children's Hospital it seems much more likely that contagion was derived from cases occurring either in the same or a neighbouring ward. Even in an instance which occurred some years ago, in which the operator, whose own children had scarlet fever at the time, probably infected the patient, there is no ground for believing that the poison was conveyed by inoculation. It may reasonably be held that it was transmitted by the surgeon, just as it might be carried by a nurse from one child to another in the ordinary way. But though there is no direct inoculation, does not the wound, we may ask, serve as the portal through which the materies morbi enters, for the surface of a recent wound offers conditions very favourable to absorption? The answer seems to be that infection through the wound, at least in many of the cases, is improbable, first, because scarlet fever has been developed in instances which were treated with strict antiseptic precautions—that is, by the free application to the wound of carbolic acid—an agent which is believed to destroy the scarlet fever poison; and, secondly, because the wound has remained healthy, which, as Mr. Stirling remarks, would hardly be the case were it the passage by which the poison found an ingress.

Thus, as to the manner in which the poison is introduced, we may, I think, acquit the wound of any direct or local agency. And yet the fact remains that the presence of a recent wound and the appearance of scarlet fever are intimately associated; and we are led to infer that in consequence of an operation the condition of the patient is in some way changed, so that, if he is exposed to contagion at or near the time of an operation, not only is he in more than the usual degree liable to be affected, but the incubation period is cut short, and the disease almost immediately appears; while, if he has been in contact with the poison some time before, though he has hitherto escaped, he is now apt to be forthwith attacked. That contagion occurring at or near the time of an operation is followed by an abnormally rapid outbreak is a generally acknowledged fact, clearly confirmed by many of the cases at the Hospital for Sick Children, in which the source of infection could be distinctly traced, and in which the incubation period was not more than twenty-four hours.

As to the instances in which the outbreak immediately after an operation appears to result from a long previous infection, some have supposed them to be cases of prolonged incubation. But this seems a very doubtful hypothesis. Though our information as to the incubation of the exanthemata is far from complete, all that is known on the subject points to the conclusion that each has a period that is liable to but little variation, and that the period for scarlet fever is rarely, if ever, more than seven days. Many cases, however, have occurred in which the interval between the infection and outbreak appears to have been so extended, that, if we accept this limit of seven days as even approximatively correct, we cannot with any show of probability regard them as examples of prolonged incubation. They may, I think, be more satisfactorily explained on the theory that the patient had carried the poison in a latent form about him, and that it was only when an operation had (in whatever manner) altered his condition that he became amenable to an attack. If we may hold this view, the cases of remote (infection) concur with cases of recent infection in illustrating the same fact, that, in the words of Sir James Paget, there is something in the consequences of an operation which renders a patient peculiarly

liable to the influence of the scarlet fever poison. No formal proof need be offered of the vitality of the poison of scarlet fever. But how, it may be asked, does it exist, or what is its habitat during those considerable intervals that are sometimes observed between its reception and its outbreak? Is it lodged only in the clothes, or on the external parts of the body of the patient? or having been inhaled, or possibly absorbed from the surface, is it retained in situations where it is in close proximity to the circulation? The latter view would afford an explanation of the sudden way in which the disease (though imbibed long before) makes its appearance after an operation; for the materies morbi is already in the same intimate association with the blood current as it would be were it inhaled or absorbed by the patient at the very time at which the operation is performed.

As to the nature of the change produced by an operation I am unable to express any definite opinion. The subject is one that leads us into a field of inquiry in which at present very little has been discovered; for it has reference not merely to the phenomena and results of diseases, but to their primary and essential nature. We know the syphilitic poison by its results, its eruptions, nodes, and ulcers; and we know that its course and the features which it assumes are influenced by the constitutional tendencies of the patient: that gout and tuberculosis, for example, stamp it with different characters. And we have acquired similar experience in respect to other diseases. We know that some patients are so prone to measles or scarlet fever that they are repeatedly attacked, while others enjoy a complete immunity, however much they may be exposed to infection. And Sir William Jenner has pointed out that some families are peculiarly liable to diphtheria, and others to typhoid fever. Here, however, we are merely observing phenomena, and it is the same with regard to scarlet fever. We see that operations increase the liability of patients to an attack, but of the cause of this liability we are still ignorant.

Whatever the nature of the change may be, the probability is that it acts through the nervous system. The effect produced on the ordinary process of nutrition by nervous exhaustion following prolonged fatigue, loss of sleep, or severe mental distress is well known; and the same condition has often been seen to predispose individuals to cholera, fever, diphtheria, &c.; and Sir James Paget has recorded an instance in which a severe fright was followed in two days by a copious syphilitic eruption.

It is, I think, to some such change—of the nature of nervous depression—that this peculiar liability to scarlet fever after operations may most reasonably be ascribed.

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## *Ueber die Natur des sogenannten chirurgischen Scharlach's.*

Dr. Riedinger, Würzburg.

Mit Rücksicht auf die vorgeschrittene Zeit und die soeben gehörten Mittheilungen des Herrn Collegen Marsh werden Sie mir erlauben, dass ich mich so kurz als möglich fasse, und Ihnen meine Erfahrungen über den vorliegenden Punct mittheile. Es ist eine merkwürdige Thatsache, dass bis jetzt nur von wenigen Seiten auf die Complication zwischen Scharlach und Wunden aufmerksam gemacht wurde. Englische Aerzte waren die ersten, welche denselben erwähnten. Paget sah in kurzer Zeit zehn Mal Scharlach nach Operationen; Murchison und May machten dieselbe Beobachtung; ebenso Harrison und Wilks; doch hielten die beiden Letzten das nach Operationen auftretende Exanthem nicht für wirklichen Scharlach, sondern für eine, dem Scharlach

ähnliche Affection. Auch französische Schriftsteller berichten über Exantheme nach Wunden. Verneuil sah solche wiederholt bei septischen Processen auftreten; dieselben hatten eine ungemein schlechte Prognose, alle Kranken gingen zu Grunde. Martinet beobachtete ein solches bei einem an Osteomyelitis femoris leidenden Knaben. Der Ausschlag hatte Aehnlichkeit mit Urticaria, zeigte grosse, rothe, erhabene Flecken, welche bald rund waren, bald nur das Segment eines Kreises darstellten. Auch Legroux sah solche Exantheme bei Puerperalkranken, ohne dass jedoch die Prognose eine absolut lethale war. Verneuil, Raynaud und Dieulafoy erwähnen ausserdem noch das Auftreten von Urticaria nach Wunden. Dieulafoy allein sah sie 14 Mal unter 27 Punctionen von Lebercysten. Aehnliche, aber immer ohne Fieber verlaufende Affectionen finden sich bekanntlich auch nach Genuss gewisser Speisen.

Ein Haupterforderniss für unsern Gegenstand ist jedoch, dass wir genau zwischen diesen Formen unterscheiden und nur dasjenige Exanthem als chirurgischen Scharlach bezeichnen, welches die characteristischen Symptome der Scar latina bietet. Wenn es nun auch wirklich richtig ist, dass die Scarlatina einen sehr wechselvollen Symptomencomplex bieten kann,—Exanthem, Fieber, Angina, Abschuppung, Incubation ect. unterliegen den stärksten Schwankungen, so ist doch das Gesammtbild ein so characteristisches, dass man wohl im Stande ist, eine Differentialdiagnose zu stellen. Von L. Thomas und andern sind solche abnorm verlaufende Scharlachfälle gesammelt worden; doch will ich auf diese Puncte nicht eingehen; nur das eine soll hier berührt werden, dass man wohl nicht selten ein Erisypel mit Scharlach verwechselt hat. Auch über den ersten Fall, den ich zu beobachten Gelegenheit hatte, habe ich mich im Anfange getäuscht; ich will diesen hier kurz erwähnen. Es handelte sich um ein sechzehnjähriges Mädchen, bei welchem ich am fünften September 1877 auf der chirurgischen Klinik zu Würzburg ein grosses Lipom, welches in der Lendengegend sass, extirpirt hatte; die Kranke befand sich am Tage der Operation recht wohl, aber schon Tags darauf hatte sie starke Kopfschmerzen, eine Morgentemperatur von 40° und eine Abendtemperatur von 41·2°; der Puls erreichte die Höhe von 120. In Folge dessen wurde der Verband gewechselt und es zeigte sich eine starke Röthe von der Wunde ausgehend und auf den Nacken übergreifend, welche ich für ein Erisypel hielt; die Wunde war dabei nicht besonders alterirt. Die Schwellung im Hals, später Eiweiss im Urin und die characteristische Abschuppung fast am ganzen Körper stellten jedoch den Scharlach ausser Zweifel. Ein Mal auf diesen Zusammenhang aufmerksam geworden, war ich in der Beurtheilung der Röthe, welche von Wunden ausging, vorsichtiger geworden und schon im vorigen Jahre konnte ich in No. 9 des *Central-Blattes für Chirurgie* zehn Fälle aus meiner Praxis zusammenstellen, zu denen ich heute noch 5 weitere hinzufügen kann. Ich verweise auf meine diesbezügliche Mittheilung und füge hier ausdrücklich bei, dass in allen Fällen die Diagnose von andern Collegen bestätigt wurde. Auch von vielen Seiten höre ich jetzt, dass man ähnliche Erfahrungen gemacht hat. In vielen Fällen schloss sich der Scharlach fast direct an die Wunde an; in andern kam der Scharlach später zum Vorschein; der höchste Termin war 14 Tage nach der Verwundung. Interessant ist der Umstand, dass diese Complication mit Ausnahme von drei Fällen, *erwachsene* Individuen betraf und dass darunter allein vier Collegen sich befanden, die jedenfalls vorher schon vielfach mit Scharlachkranken in Berührung gekommen waren. In der Familie des einen hatten bei einer Epidemie alle Geschwister desselben, mit Ausnahme von ihm, den Scharlach durchgemacht. Im Grossen und Ganzen kann man sagen, dass der Scharlach immer ein schwerer war. Drei Collegen hatten sich septische Phlegmonen durch Verwundungen bei

Sectionen zugezogen. In einem Falle handelte es sich um die Section eines an Typhus Verstorbenen.

Wir stehen übrigens in der Chirurgie mit dieser Complication, nicht allein; auch in der Geburtshülfe hat man die gleiche Erfahrung gemacht. Olshausen hat vielfach Scharlach direkt nach Entbindungen gesehen und wahrscheinlich sind die Fälle von Legroux hierher zu rechnen. Fünf Fälle von den obenerwähnten waren antiseptisch behandelt worden. In vielen Fällen war oft weit und breit kein Scharlach vorhanden, und ein ganz besserer, hervorzuhebender Punct ist der, dass in zwei Fällen andere Personen in der nächsten Umgebung angesteckt wurden.

Die Hauptfrage, die sich an diese Thatsachen unmittelbar anknüpft, ist selbstverständlich die, ob die Kranken schon vor der Verletzung inficirt waren, oder ob dies erst nachträglich geschah. Man kann dies natürlich nicht mit voller Präcision für alle Fälle entscheiden. Für die meisten der vorliegenden Fälle dürfen wir den Scharlach, als von der Wunde abhängig, bezeichnen; jedenfalls würde die Ansicht, dass die Kranken vor der Operation schon befallen worden sind, gewiss nicht für alle Fälle passen, da die Incubationszeit dann für manche eine allzulange wäre; um somehr als wir wissen, dass durch Murchison festgestellt wurde, dass der Scharlach zu den Infectionskrankheiten mit kürzerer Incubationszeit gerechnet werden muss und sieben Tage die Höhe ist. Richardson und Trousseau berichten über sieben und achtstündige Incubation, und Gerhardt beobachtete ebenfalls ganz kurze Incubationszeit. Murchison sah in 75 Fällen fünfzehn Mal in den ersten 24 Stunden nach der Infection Scharlach auftreten. Der zweite Punct, der hier nicht ohne Erwähnung bleiben darf, ist der von wo aus das Scharlachgift in den Körper gelangt. Es liegt die Annahme, dass dies auf dem Wege der Wunde geschehen kann, gewiss nahe und wir müssen in dieser Ansicht um so mehr bestärkt werden, als, wie oben erwähnt, das Exanthem seinen Anfang von der Wunde nahm, und in deren Umgebung am stärksten ausgeprägt war. So gut dies bei einem Erisypsel der Fall ist, mit dem ja doch Scharlach, Diphtheritis nahe verwandtschaftliche Beziehungen haben, eben so gut kann dies auch bei unserer vorliegenden Krankheit der Fall sein. Weitere Beobachtungen, die in dieser Beziehung sicher jetzt in reichlicherem Maasse gemacht werden, als früher, dürften wohl schon in der allernächsten Zeit mehr Licht in die Frage bringen.

## DISCUSSION.

Mr. Holmes, London: I have myself seen a good many cases of rash occurring after operations, although I have not preserved any numerical or other record of my cases. The result of this experience, as well as the reading of the literature on the subject, together with the interesting observations of Mr. Marsh, Herr Riedinger, and M. Trélat, leads me to the firm conclusion that persons after surgical operations are much more liable to scarlet fever than others, as stated by Sir J. Paget. This is the ordinary disease, and is in all probability contracted in the ordinary manner, though, as M. Trélat seems to think, it is possible that in some instances the inoculation may affect the wound itself. However that may be, the disease has the same essential features as scarlet fever, and, as some of Dr. Riedinger's cases showed, it may propagate scarlet fever to others. But I am equally convinced that many of the cases which are classified as "surgical scarlet fever" are not scarlet fever at all, but some of them the rash of pyæmia, some, possibly (as Dr. Riedinger has hinted), erysipelas, others anomalous roseolous rashes, without any symptom of scarlet fever, and some of these quite destitute of danger, or indeed of any importance

whatever. On the nature, symptoms, and causation of these hitherto unclassified cases, further experience is necessary in order to enable us to speak positively.

M. TRÉLAT, Paris: related particulars of three cases of scarlatina in children, which supervened after slight operations. He had not observed anything peculiar in the disease, and believed it to be nothing but an intercurrent affection, due to direct contagion.

Dr. GOODHART, London: I am quite in accord with what appears to be the now preponderating opinion that the majority of scarlatina-like rashes after operations are veritable scarlatina. I also think with Mr. Marsh that occasionally rashes closely simulate scarlatina, as the belladonna and other rashes may sometimes do, and which are not scarlatina. But these cases are so difficult to distinguish, that for practical purposes it is proper, unless there be unusually clear indications to the contrary, to isolate all patients affected on the appearance of the rash, for the benefit of surrounding patients. But I think we must all be more interested in the question how the peculiarities of the diseases are best explained. To this point I shall address myself; and, as most suggestive, the points which occur to me are chiefly these—(1.) That though such cases are chiefly found after operations (seventeen out of twenty-five of the cases collected by Mr. Paley and myself from the Evelina Hospital for Children were so), still that a fair proportion—one-third in our series—occur in other surgical cases, many of them not having even wounds. (2.) That *medical* cases are by no means exempt, and that cases of phthisis and such like, which have certain obvious relations with surgical cases, are more likely to be attacked than others. This has been so at the Evelina Hospital, and similarly in one of the wards at Guy's Hospital a year ago, a child with scarlatina was admitted by accident, and the only adult case which suffered was a bad case of phthisis. Now what have these cases in common? They are all of them cases of local inflammation, and it is this which I venture to suggest is the important factor. I might support this view if it were necessary by recalling to your minds the experiments which of late years have been made showing that inflammatory foci are good soils for the cultivation of fevers; but with those before me from France and Germany who have made the study of the natural history of fevers so peculiarly their own, it is unnecessary. I would rather appeal to two diseases which I think support me. Pharyngeal diphtheria is allowed to be particularly liable to take root in the subject of chronic tonsillitis and faucial catarrh; and, secondly, I would point to the disease we in England know as ulcerative endocarditis, but which is called diphtheritic endocarditis abroad. In the great majority of cases this is found, not as a primary disease of the valves, but as secondary to their chronic inflammation. It would almost appear as if germs of all kinds swoop upon foci of chronic inflammation like vultures upon a corpse. It seems to me possible that some such occurrence as this takes place in children after operations—takes place in persons the subject of any local inflammation; and thus those who have not previously procured immunity by passing through the disease are peculiarly liable to be attacked. In conformity with this, though no doubt admitting of other explanation, is the fact observed by Mr. Paley and myself, and subsequently by Mr. Stirling at St. George's, that children with wounds dressed upon the antiseptic system did not escape. Thirteen out of seventeen wound cases were dressed under strictly antiseptic precautions, and yet they suffered. Now, I think it is proved by a large body of evidence that such dressings are sufficient to eradicate certain diseases, as erysipelas, and from this I infer that they enter by the wound, and that the scarlatina poison does not. It will, I know, be objected to such a view, that many of the cases, though cases

of operation, have been subjected to so slight an operation as to be virtually exempt from its risk. Tenotomies, for instance, have not unfrequently been followed by scarlatina. I may appear to be somewhat straining my arguments, but it seems to be sufficient to meet this by saying that local inflammations, if they do produce a good soil, are perfectly well able to cultivate the disease, be they small or great. But no doubt severity within certain limits (for some very putrid inflammations appear to be destructive to germs) is favourable to this production, particularly if they are associated with much fever. I would also add that, from what I have seen in peritoneal exudations, there are few fluids so good for cultivation of bacteria as extravasated blood. The modifications which this interesting affection undergoes are also perfectly explicable, or, if not explicable, are rendered probable from analogy upon some such hypothesis.

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## *Ueber Empyem-Operation bei Kindern.*

Prof. C. Gerhardt, Würzburg.

Die Schicksale der Empyem-Operation waren wechselvolle. Sie wurde bald als lebensrettend gepriesen, bald als erfolglos verworfen. Schon in der hippocratischen Zeit zu hohem Grade von Vollkommenheit gebracht, (Ausspülung, Offenhalten der Wunde) wurde sie später häufig von der Masse verworfen, immer wieder von einzelnen hervorragenden Forschern in die Höhe erhoben. Eine Zeit lang schien ihr Schicksal mit dem der physikalischen Diagnostik verknüpft zu sein. Laennec und Skoda haben sich beide mit grossem Interesse dieser Operation zugewandt, später durch wenig günstige Erfolge entmuthigt, sie wieder verlassen. Ein wichtiger Fortschritt wurde durch die strenge Scheidung der Indicationen gewonnen: Punction mit Luftabschluss beim Prüfen, freie Eröffnung des Pleurasackes bei eitrigen Ergüssen. Gleichzeitig gewann durch die Probepunction mit Pravàz'scher Spritze die Diagnose des Empyems ungemein an Sicherheit. Unter einen neuen und glücklichen Stern trat der Brustschnitt mit dem Aufleuchten der antiseptischen Methode. Aber auch diesmal wurden die ersten Hoffnungen nicht erfüllt. Brustschnitt und tägliche Carbolausspülung der Pleurahöhle gaben wenig günstigere Resultate als Unterlassen jeder Operation (Statistik von Ewald). Nach diesem Scheitern der ersten Erwartungen bildete sich eine Anzahl weiterer Methoden aus, die in ihren Wegen weit auseinandergehen.

Ihre Berechtigung ist zum Theil nach der Auffassung einer Vorfrage zu beurtheilen, nämlich ob das Empyem einfach als ein, nach innen von den Rippen und nach aussen von den Lungen gelegener, Abscess anzusehen sei. Wenn das Empyem Nichts anderes wäre, wie ein gewöhnlicher Abscess, so müssten breite Eröffnung unter antiseptischen Cautelen, Ausspülung mit Carbollösung und Sorge für genügenden Abfluss ebenso rasche und sichere Heilung erzielen wie bei jedem anderen Abscess. Heilungen auf natürlichem Wege, ohne Operation, dürften nur äusserst selten vorkommen. Nun zeigt aber die Erfahrung, das kleinere eitrige Pleuraexsudate sehr oft, grosse noch hie und da durch einfache Resorbtion zur Heilung kommen, dass die Resultate des erwähnten Verfahrens in weit höherem Maasse die Gefahr der Carbolintoxication mit sich bringen, als dies bei anderen Abscessen der Fall ist, kurz, dass die Resorbtionsverhältnisse an der Pleura mit ihren offenstehenden Lymphgefässmündungen ganz andere sind, als an gewöhnlicher Abscesswand. Gelegentlich der Spontanheilungen sei hier noch jene schon von Hippocrates erwähnte, von Traube eingehend gewürdigte Beendigungsweise eitriger Exsudate betont, welche durch Aushusten des Eiters ohne Lufteintritt in den Pleurasack zur Heilung führt. Sie ist, wie ich glaube,

häufiger, als man gewöhnlich annimmt, kommt namentlich den Empyemen nach Pneumonie so oft zu, dass ich sogar bei diesen mit dem operativen Eingriffe etwas länger zu warten, mich berechtigt halte. Ein zweiter, gerade für diese Dinge wichtiger Gesichtspunkt ist aus den Ausdehnungsverhältnissen der Lunge abzuleiten. Einerseits ist das Pleuraexsudat um so leichter resorbtionsfähig, je geringer die Einschränkung der Ausdehnungsfähigkeit der Lunge, anderseits ist die dauernde Schädigung der physiologischen Leistung und anatomischen Structur um so geringer, je weniger die erzielte Heilung zugleich Einbusse an Ausdehnung und Ausdehnbarkeit der Lunge bedingt. Darauf hat aber die angewandte Operationsmethode bedeutenden Einfluss und man wird wohl sagen dürfen, dass diejenigen Methoden, die grundsätzlich das Einsinken der Seite begünstigen, mehr den Abscess als die Lunge berücksichtigen.

Die Methoden, welche gegenwärtig am meisten in Betracht kommen, dürften sein: 1. Freie Eröffnung der Pleurahöhle unter antiseptischen Cautelen, Drainirung, Abfluss unter antiseptischem Verbande. Die Oeffnung wird nach König's Methode in ihrem Bestande gesichert durch Resection einer Rippe. Nur bei zersetzter Beschaffenheit des Exsudates, Ausspülung und zwar nicht mit Carbollösung, sondern mit einer minder giftigen, desinficirenden Flüssigkeit, z. B. Chlorzinklösung. 2. Punktion des Pleurasackes, Ausspülung durch die Troicartröhre unter Luftabschluss mit einer indifferenten, z. B. Kochsalzlösung oder desinficirenden Flüssigkeit, wie Salicylwasser. Wiederholung des Verfahrens bei Bedarf. 3. Langsame Ausspülung. Ableitung des Exsudates durch die liegenbleibende Troicartröhre unter Carbol- oder Salicyllösung. Oeftere Erneuerung dieser Flüssigkeit. Eintreiben der Flüssigkeit mehrmals täglich durch Erheben des gewöhnlich tiefer stehenden Gefässes. Welche dieser Methoden den unbedingten Vorzug verdient, möchte heutzutage schwer zu sagen sein. Der eigentlich chirurgische Eingriff, wie ihn König entwickelt hat, verfügt über die zahlreichsten und besten Resultate, die bis jetzt aufzuweisen sind. Grössere Statistiken liegen weder vor über den direkten Erfolg, noch auch über etwaige Häufigkeit von Nachkrankheiten. Die zweite Verfahrungsweise ist nach manchen misslungenen Versuchen Früherer, erst durch Bälz zur brauchbaren geworden. Der Eingriff ist geringer, die Resultate sind weniger entscheidend, auch nicht so günstig; ausgedehnte Erfahrungen liegen noch nicht vor. Noch mehr gilt Letzteres von der dritten Methode, die von Playfair, wenn auch nicht ganz in dieser Weise angegeben, von Bäumler neuerdings ausgebildet wurde.

Fragen Sie, was hat dies Alles für das Kindesalter zu bedeuten? so lässt sich die Antwort in wenige Sätze zusammenfassen: Die Kräfte des Kindes werden früher erschöpft, Pleuritis purulenta ist für das Kind eine durchschnittlich acutere, gefährlichere Erkrankung. Die Indication zur Operation tritt daher früher ein. Die Prognose ist günstiger, weil man es mit Individuen zu thun hat von lebhafter Reproduction, ungeschädigten, inneren Organen, biegsamer Thoraxwand. Die Heilungsdauer nimmt kürzere Zeit in Anspruch, das Zurückbleiben von Fisteln und bedeutenden Missstaltungen ist weniger zu fürchten. Während bei Erwachsenen oft nur Abkürzung langen Siechthums, wesentliche Besserung in ernsten Leiden erzielt wird, tritt gerade im Kindesalter der lebensrettende Charakter der Operation bestimmter hervor.

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## *Die chirurgische Behandlung des Empyems.*

Dr. Adolf Baginsky, Berlin.

1.—Die pleuritischen Exudate des kindlichen Alters sind weit häufiger eitrigen Charakters, als diejenigen der Erwachsenen.

2.—Die Diagnose des Empyems wird nicht sowohl durch die physikalische Diagnostik und durch die Fiebertemperaturen, als vielmehr neben beiden durch die antiseptisch vorgenommene Probepunction festzustellen sein.

3.—Nach Feststellung der Diagnose durch die Probepunction ist insbesondere bei jüngeren Kindern (1–3 Jahren) nicht sogleich chirurgisch einzugreifen. Man beobachtet ziemlich häufig einen Durchbruch des Eiters durch die Lunge, (Entleerung reichlicher Eitermengen mit dem Husten) unbeschadet der späteren vollständigen Restitution sowohl der Lunge, als der Pleura.

4.—Die Indication zum chirurgischen Eingriff geben *a*) andauerndes Fieber, *b*) andauernder quälender Hustenreiz, *c*) Appetitlosigkeit mit Abmagerung.

5.—Die Punction (antiseptisch) mit einfachem, mit Condom zum Zweck des Luftabschlusses versehenen Troicart, ohne nachfolgende Ausspülung, genügt in einzelnen Fällen zur definitiven Heilung des Empyems. Diese Erfahrung weist darauf hin, insbesondere bei jüngeren Kindern (1–3 Jahren), die Punction jedes Mal vorerst zu versuchen, und bei Wiedererneuerung des Eiters dieselbe zu wiederholen. Die Punction erfolgt an einer möglichst tiefen Stelle des Thorax in der hinteren Axillarlinie. Es darf nicht der gesammte eitrige Inhalt des Thorax sofort entleert werden, sondern nur so lange darf man den Eiter fliessen lassen, als er im Bogen und unter stärkerem intrathoracischem Druck fliesst. Die Punctionsöffnung ist sorgfältig luftdicht mittelst Heftpflasters zu schliessen.

6.—Wenn nach 2–3maliger Punction das Fieber andauert, das Empyem sich wieder erneuert, die Kräfte des Kranken und seine Ernährung herunterkommen, ist zur Incision zu schreiten. Die Operation geschieht unter antiseptischen Cautelen. Langsam, mit präparirenden Schnitten vorgehend, wird die Pleurahöhle eröffnet. Die Wunde wird an einer möglichst tiefen Stelle, parallel zum Verlaufe der Rippen, entsprechend einem Intercostalraume angelegt, so zwar, dass der innere Winkel in der hintern Axillarlinie beginnt. Der Eiter wird zunächst durch freies Fliessenlassen unter Carbolspray entleert. Die Thoraxhöhle wird sodann mit einer 3 % Lösung von Acid. salicylicum sorgfältig ausgespült, bis die Lösung rein abfliesst. (—Carbolsäure ist bei der Ausspülung in der Befürchtung einer Carbolsäureintoxication zu vermeiden.) In die Wunde wird ein desinficirtes, aus einem mittelstarken Gummirohr bestehendes Drainrohr eingelegt. Dasselbe wird mit Faden und Heftpflaster an der Thoraxwand befestigt. Die Wunde mittelst antiseptischen Verbandes sorgfältigst bedeckt.

Die Resection einer Rippe kann bei Kindern in der grössten Anzahl der Fälle vermieden werden.

Ausspülungen des Thorax erfolgen nach der Operation und, wenn neue Fiebertemperaturen ohne anderweitige Complicationen, welche dieselben etwa bedingen können, auftreten, und wenn diesselben auf eine neue, auch physikalisch nachweisbare Ansammlung von Eiter hinweisen.

Die Erneuerung des Verbandes erfolgt sobald eine Durchfeuchtung desselben mit Eiter sich kund giebt.

## DISCUSSION.

The PRESIDENT: With a view to making the discussion as practical as possible, I would suggest the following points as especially worthy of consideration :—(1.) The indications for thoracentesis in acute purulent pleurisy. (2.) The indications for the repetition of the puncture, and the comparative advantages and risks of frequently repeated punctures or of a permanent opening. (3.) The question of drainage; the best situation for the counter-opening (if any); and the further question of drainage tubes, or of means for keeping the opening patent. (4.) The employment of carbolised or other injections as a means either of restraining the discharge or of counteracting decomposition.

Dr. A. JACOBI, New York: In the course of a single year I have observed three cases of empyema in young children which required but a single aspiration. Complete recovery took place, though the quantity of pus in one case amounted to three or four hundred grammes. The flexibility of the young ribs is such that, even in those cases in which the lungs will not expand again to their former condition, the sinking in of the thorax is considerable enough to promote recovery. In adults I have resorted to the resection of large pieces of rib to accomplish the same end.

Professor RANKE, Munich: I think the opening of an empyema into a bronchial tube is of rare occurrence. The reason of my scarcely ever seeing it may, however, be that I never allow a case of empyema to run its course unchecked. It is my habit to explore every case of very considerable effusion into the pleural cavity which has lasted for several weeks with an ordinary morphine syringe. If the fluid extracted be pus, it is my habit to operate at once, without waiting for an aggravation of the symptoms; and I obtain excellent results by this method. I will endeavour to give my experience on some of the points Dr. West has mentioned. First, in regard to the question whether a free incision should be made, or only a small opening with a trocar. I always make an incision, through which I can easily introduce a tube to drain off the matter. As to washing out, in my first cases I invariably practised it, using either a one per cent. solution of carbolic acid, or a two per cent. solution of boracic acid; but latterly I have found that washing out is not necessary, and the cases do almost better without it. I have therefore given it up. I perform the operation under the carbolic spray, and apply strictly antiseptic dressings to the wound for about four weeks; after that time I treat the wound as an open one. Frequent tapping instead of drainage I have never tried, and am therefore not prepared to advise it. A counter-opening I have never made, because I never found one necessary. It has often surprised me to notice how the lung, which at first had been entirely compressed by the purulent effusion, will steadily expand again, the drainage tube having to be shortened gradually as the cavity grows smaller. Severe cases of empyema, however, often take a long time to heal—sometimes ten or twelve months. The deformity of the spine need by no means be great after a successful cure. In fact, I could show some of my cases in which the spine shows scarcely any deviation from the normal state. It is wonderful how completely a *restitutio ad integrum* can be brought about in a growing child.

Dr. ROBERT LEE, London: In the surgical treatment of empyema it is probable that in any particular case there will be little difference of opinion—that is to say, the necessity of making a permanent opening and inserting the drainage tube will be decided by the nature of the symptoms. The opinion has certainly been expressed by one who has had opportunities of watching cases of empyema during the period of convalescence, both after aspiration and the insertion of the drainage tube, that better results were obtained by repeating the aspiration than by the latter method. The question of chief interest seems to be the management of the tube, which, as generally used, is clearly opposed to the physical necessities of respiration. When it is remembered that the insertion of a tube through which air can pass freely into the thoracic cavity in the movements of inspiration must deprive the lung of the force of atmospheric pressure, on which expansion depends, the disadvantages of this mode of treatment are obvious. In the direction of meeting this difficulty and in observance of the physical requirements of these cases there is still much to be done, so that, if possible, the

advantages of aspiration may be combined with those of the free opening and the drainage tube—that is to say, a method by which a constant and gentle pressure can be exerted on the lung tissue. That the theoretical considerations of this subject are supported by practical observation I have been able to prove in one case of a child of four years of age, who wore an elastic cup over the opening of the tube, which cup the child replaced when it became detached, as it did at intervals of between three and four hours, apparently finding some comfort from the atmospheric conditions thus obtained in the thoracic cavity. I mention this view of the question of the treatment of empyema as the one which seems deserving of consideration in the surgical treatment of the disease.

Mr. F. Richardson Cross, Bristol: Early removal of pleuritic effusion is necessary to encourage re-expansion of the lung. If aspiration shows pus, and a second aspiration is not followed by recovery, free incision in the ninth or eighth interspace to allow continuous drainage through a tube is indicated. If this is done under Lister's antiseptic method, the pus will be replaced by serum, the wound will soon heal, and perfect recovery of chest shape and lung power will result. Three cases treated on this method, females aged $2\frac{3}{4}$, 15, and 18 years (the only ones the author has had an opportunity of treating), were quoted. The wounds had healed in six weeks, seven weeks, and three weeks respectively. Two months afterwards one chest had returned to its perfect state, the most favourable case was lost sight of, and the third and most chronic showed fair breathing and was improving.

Mr. R. W. Parker, London: As the question of treatment must very much depend on the mechanical condition of the chest, it would be well to divide empyemas into two chief classes, as found (1.) in children, and (2.) in adults. For we have to remember that whatever method of treatment is adopted, we cannot expect a favourable result unless the conditions regulating chest movement assist us. Thus an empyema cavity cannot be emptied unless the lung can re-expand or the chest wall fall in. In children these conditions obtain more commonly than in adults, and hence the disease is much less serious. In old people, whose chest walls are very rigid, empyema is a serious disease, and often all but incurable. I believe that aspiration—two or three times repeated if need be—is the best treatment in childhood, and ought always to be adopted before other measures are tried. No doubt the next best mode of cure is the expectoration of pus through the lung; but bearing in mind how this is brought about, it is hardly safe to postpone treatment until it takes place spontaneously, and unfortunately there are no mechanical means by which it may be hastened. I have seen many cases in which the pus has been evacuated in this manner, and I think it occurs, to some extent, more frequently than authors seem to admit. When aspiration repeated two or three times has failed (or sooner if the collection is large or if the patient seems to be losing ground), a free incision into the lowest and most dependent position of the empyema cavity, with antiseptic precautions, is called for. In some cases, antiseptic precautions notwithstanding, decomposition takes place and the discharges may become exceedingly fetid. For such cases washing out the cavity is then necessary. A solution of quinine (5 grs. – ℥i.) will be found the most serviceable. If the patient be a small child, it may be placed in a bath up to his neck; in this way the abscess cavity may be most thoroughly washed out. In adults I also advocate aspiration before other treatment is tried. But if the cavity is large and the chest walls rigid, I believe that great advantage is to be obtained by injecting filtered and carbolised air into the pleural cavity; for while this helps to displace the fluid, it also lessens the dragging sensation often complained of after tapping;

and by supporting the delicate vessels in the pyogenic membrane, it lessens subsequent exudation, and thus the risk of reaccumulation.

Professor GERHARDT, in reply: My final remarks refer chiefly to the points to which our President directed attention. The successful cure of empyema by expectoration through the lungs (after the manner mentioned by Traube) is much less common in children than in adults, but it certainly does sometimes occur in early age. Antiseptic washing out appears to me only necessary when decomposition of the pleural contents occurs at an early date. In like manner, the need of a counter-opening is only felt in special cases, as, for instance, when great narrowing of the first opening occurs, or a fistula leading to a small cavity becomes established. I am very glad that in the course of the discussion on the mechanism of respiration the difficulty of re-expansion of the lung by atmospheric pressure was dwelt upon. For the present, I believe that the method of treatment by incision promises the best results, but I cannot think that this will permanently be the case.

The Surgical Vice-President, Mr. HOLMES, then summed up the debate. After rendering the best thanks of the Section to Professor Gerhardt for his admirable treatment of the question, he said that the queries propounded by Dr. West seemed to him to cover the whole field of the debate. To most of these questions very clear answers had been given by Dr. Gerhardt, and, as far as he could discover, the following conclusions on them had been accepted by the majority of those present. It seemed to be admitted that simple aspiration of the pus is often successful, and that it is, on the whole, the most successful method of treatment, simple cases requiring no permanent opening into the pleura. The practice of different surgeons seems to differ as to the frequency with which the puncture should be repeated, but most seem in favour of not repeating it unless the symptoms are obviously increasing. That the pus may be spontaneously expectorated seems admitted on all hands, and also that this is not so common as to form any real indication—*i.e.*, that we ought never to take this into account surgically, but operate as the symptoms seem to require. Some of the speakers (*e.g.*, Dr. Ranke) seem more in favour of a permanent opening by means of a canula, the mouth of which is kept always "antiseptic." Permanent opening by incision seems chiefly indicated when there is a portion of the pleura in which there is an encysted collection, and the lung cannot rise into the vacuum formed by the aspirator. In such cases it seems also right to wash out the cavity, either with weak carbolic lotion, with boracic acid, or (as recommended by Mr. Parker) with purified air. In these cases the cavity probably gets obliterated by adhesions, and as these adhesions contract and cicatrise, they probably draw the lung towards the ribs, as well as the reverse. Counter-openings and the retention of drainage tubes in the chest seem rarely necessary, and then mainly in the case of encysted collections. If it be necessary to make a counter-opening, it seems that this should be at no great distance from the other, and not too near the spine. The prognosis and treatment of the resulting deformity was hardly touched on in the debate. In fine, the whole discussion seemed to prove the gratifying fact that the treatment of empyema in early life had become much more successful than formerly.

## *Observations on the Pathology and Treatment of Genu Valgum.*

Dr. William MacEwen, Glasgow.

Flat-foot has been put down as a characteristic of genu valgum, whereas in the great majority of instances the foot is highly arched. Indeed, in all cases where the person is able to walk much, the foot is found arched. In children, who can walk very little, the foot is flat; but as these children grow older and stronger, the genu valgum continuing, the arch of the foot develops and increases. All such persons wear the outside of the heel of the boot, the inside being little touched. The high instep in genu valgum results from the great muscular action required to maintain the equilibrium of the patient while walking. When such a patient walks with nude feet, one perceives that the toes seems to grasp the floor at each step.

Genu valgum cannot be accounted for by a single pathological character. The internal curve of the lower third of the femur is the most common factor in its formation, being met with in 120 out of 166 limbs affected with genu valgum. There is likewise an abnormal elongation of the internal condyle in most cases, but this is to a much slighter extent than was formerly supposed, and it is very seldom sufficient to account for the whole of the deformity. In about 10 per cent. of the cases the head of the tibia is set askew on the tibial diaphysis. In aggravated cases of this kind there is generally a prominent portion of bone resembling a spine projecting from the inner side of the tibia about the position of the insertion of the internal lateral ligament. Where such exists, the limb is rendered more symmetrical by its removal.

*The age at which osteotomy for genu valgum ought to be performed.*—The question is often asked, at what age ought the operation to be performed for the cure of genu valgum? It is impossible to fix a hard and fast line at any age during the period of growth, and say that on one side of this line operations for genu valgum ought to be performed, while on the other they ought not. It depends on the state of the bones, and each case ought to be considered on its own merits. The question may be answered by stating that no operation for genu valgum ought to be performed during the period of *ramollissement*. Attention to this would at once prevent the performance of operations on patients who might recover by good hygiene and simple remedies, and at the same time it would do away with the likelihood of any return of the incurvation after the performance of the operation. I have very seldom performed operations for the relief of genu valgum under eight years of age, but I have refused to operate on much older patients who bore traces of an acute and recent attack of rickets, and whose bones were still soft.

Some surgeons are stated to operate for genu valgum at two and three years of age. Personally I deprecate such a course, believing that many such deformities occurring at that age disappear by attention to the bodily health along with a little care on the part of the parents. Therefore many such children are subjected to an operation which might be done without. Again, the bones in such young patients are soft and easily yield, and the deformity, though remedied for the time by the operation, would be apt to return, especially after an attack of measles, scarlet fever, or other disease incidental to childhood. Lastly, when the bones are so soft as "to permit the chisel to be pushed through easily by the hand alone," as some surgeons say they do, it must be apparent that under these conditions the operation could easily be avoided, and some simple splint or manipulation substituted.

*The results of supra-condyloid osteotomy*, as performed by me for genu

valgum, have been excellent. The advantages of the operation and the results may be summed up as follows :—

A deformity of a very aggravated kind has been removed ; the height of the patient has been increased, often by several inches ; the patients have been able to walk easier, much better, and a great deal farther than they could prior to the operation. As a consequence they improved in health and strength, and in many instances they have been enabled to occupy situations from which they had previously been debarred. It would be out of place to give cases in detail, especially as they are numbered by hundreds, but by way of illustration the following may be quoted :—

A lad, eighteen years of age, was affected with genu valgum to such an extent that a horizontal line drawn from the internal malleoli measured 18 inches, while a vertical line running from the horizontal one to the apex of the knees measured 15 inches. He could neither walk nor stand for more than five minutes at a time, and was totally unfitted for work. After supra-condyloid osteotomy his limbs were straight; he could walk and leap with ease, the movements of the knee-joints being perfect. He was several inches higher than he was prior to the operation. A year afterwards he several times walked eighteen miles at a stretch without fatigue. He is now engaged as a mechanical engineer, in regular employment, besides walking six miles daily while going and coming from his work. A man twenty-five years of age, affected with double genu valgum (15 inches between the malleoli), had to give up his work, some time previous to his admission into my ward, on account of his inability to walk to and from his work and the difficulty of standing at his employment. After supra-condyloid osteotomy his limbs were straight, his knees were freely movable, his stature increased, and he was capable of doing his work with ease. A year afterwards he walked in four consecutive days over one hundred miles without undue fatigue. A third, affected with marked genu valgum, could scarcely go with the help of crutches from his own door to the shop at which he wrought, situated about thirty yards distant. There he had a sedentary occupation. After operation, besides having straight, freely movable limbs and being increased in height, he was able to walk well without any support. He shifted his residence, walking then four miles daily to his work. At one time he walked ten miles at a stretch without fatigue.

Do the femora continue to grow after supra-condyloid osteotomy? Yes, they do, just as though there had been no operation performed. There are hundreds of cases where the increase has been distinctly marked. For instance, one boy aged eight years, affected with genu valgum, measured four feet in height; after supra-condyloid osteotomy he measured four feet three inches. Four years after, he was measured, and found to be four feet ten inches, the femora being proportionately increased.

Up to April 6, 1881, there have been 767 limbs osteotomised by me, and in these 1149 osteotomies were performed. Out of these there were three deaths from causes independent of the operation (pneumonia, diphtheria, tubercular meningitis), and published in detail elsewhere ("Osteotomy," p. 165). Of these 767 limbs which were operated on, 521 were for genu valgum, the supra-condyloid method being employed.

This operation has been performed many times in Scotland, England, and Ireland, and very favourable accounts have been received of it from Germany, Italy, and America.

## *On Genu Valgum and its Varieties.*

Dr. W. J. Little, London.

By genu valgum (in-knee) should be understood a distortion of the *knee-joint*, and should be distinguished from a deformity similar in appearance which mainly depends upon curvature and changes in form of the femur and tibia.

Hence many writers have treated of the distortion under two forms—(1.) the *statical*, in which the influence of gravity has been paramount in its production; and (2.) the *rachitic*, in which softening of bones has been the primary disturbing cause. It is, however, obvious that when rachitic softening has taken place, and the sufferer endeavours to assume the erect posture and to effect locomotion in that posture, statical influence will come into operation as well in the rachitic form as in that denominated statical *par excellence*.

It is desired in this paper to draw fully the attention of surgeons to the fact that genu valgum is an alteration in the form and relation of parts of the knee-joint which is apt to accompany several—indeed the majority—of disordered states to which the knee is liable, either in their early or in their later stages.

The author lays it down as an axiom that any disorder or disease of the knee structures, active and passive, through which either the relations of parts or the equilibrating forces are disturbed, the condition or distortion termed genu valgum will arise unless interfered with by art.

Hence it may be said that there are almost as many clinical varieties of genu valgum as there are knee affections; and further, that the distortion may arise even in a perfectly healthy knee, when, owing to disorder in one limb, from accidental injury or from paralysis, for example, the sound one is overladen and overworked.

In-knee may exist at birth; it may originate in the one-year-old fast-growing infant from the want of mother's milk, from improper and from too watery a diet without rachitis; it may depend upon unequivocal rachitis, upon infantile paralysis, and spasm. It arises (without rachitis) less frequently in the later years of childhood, unless the child has been debilitated by measles, hooping-cough, or scarlet fever, and has been permitted during convalescence to resort too soon to standing or prolonged exertion. On the approach of puberty in both sexes, during another fast-growing period, say from the twelfth to the sixteenth year or later, a liability to the distortion again sets in.

At any period before adult age the occurrence of white swelling (strumous synovitis) is apt to present, besides contraction of the joint in the flexed position, manifest inward inclination of the knee, with corresponding eversion of the leg (genu contractum et valgoideum). With advancing years, the rheumatic knee, especially in subjects who were regarded as strumous in their youth, besides becoming contracted in the flexed position, is apt to assume a distinctly valgoid direction, with marked pain in the neighbourhood of the internal lateral ligament and internal condyle, especially when attempts to use the limb are made, and the tendency to distortion is not checked by art.

The author has seen several cases of considerable genu valgum in tall, robust adolescents, and adults affected with polysarcia, inordinate height and weight having apparently contributed in unequal proportions to the distortion.

This frequent liability to genu valgum under so many conditions does not spring, as often asserted, from the natural form and relation of the component parts of the limb, and especially of the articulating surfaces of the knee-joint; for in the normal state, in a well-knit knee, the active and passive structures, the

moving and resisting powers, are so well balanced that the most perfect symmetry, and a large reserve of capability for use beyond the average use exists. It can no more be admitted of genu valgum than of congenital club-foot, as has been stated by some surgeons, that every child born into the world has a certain degree of, or a tendency to, both those affections. All that can be admitted is, that when disorder or disease of the knee or foot takes place and distortion ensues, the form of the distortion will be determined in a certain direction, rather than in other directions, by the natural anatomical relations and functions. In the causation of genu valgum the natural greater size of the internal condyle, the naturally adducted position of the femur in relation to the trunk and to the tibia, the asserted naturally less developed condition of the external articular surface of the tibia, the known greater physiological range of adduction of the tibia over adduction in some positions of the knee, cannot be regarded as primary causes, and should not lead the surgeon to the employment of a particular operation, say, for example, that of detaching the internal condyle from its surrounding relations as a means of cure.

The author equally denies the primary influence of contraction of any muscle, *e.g.*, the biceps femoris, in producing the distortion, and denies the necessity for tenotomy, except in rare and extreme cases.

He places the two more loudly proclaimed causes of genu valgum—viz., enlargement of the internal condyle and contraction of biceps, in the rank of consequences of in-knee, and as secondary obstacles to restoration.

The most important secondary obstacle is that of diminished development of the external condyle, and of the corresponding external articulating surface of the tibia, both of which are caused by the weight of the body during standing and walking being from the earliest stage of genu valgum unduly borne by those parts of the articulation. As time advances, the distortion remaining uncured, the continued compression to which these parts remain subjected occasions a continuous increased disproportion between the size of these compressed parts, and the internal articulating parts of the femur and tibia. Indeed, the internal parts of the femur and tibia from ceasing to bear a due weight, and being instead exposed with the internal ligament to undue stretching and dragging, exhibit undue growth.

From the operation of these causes it is constantly observed in the two-year old infant, or older, affected with statical or rather "non-rachitic" genu valgum, that there exists a gap between the external condyle and corresponding part of the tibia, when the knee is gently straightened by the hand of the surgeon, into which, but for the integuments, fascia, and ligaments, the tip of his finger could be deeply introduced. When the bones of a severe genu valgum in an anatomical cabinet are disarticulated, and are placed as nearly perpendicular as may be, in their natural relation, the gap just alluded to is represented by a triangular space between the external articulating surfaces, the depth of the base of which may measure from one quarter of an inch to one inch according to the severity of the case. This coign of disadvantage constitutes the grave anatomical fact which the surgeon in the treatment of the majority of cases, especially in the non-rachitic ones, has to keep in view.

In the bones of rachitic cases this gap is less marked, probably because the bones in the softened stage had moulded themselves under the pressure of gravity towards one another, and because the curvatures in the shafts induced by gravity have often more to do with the valgoid form of the limb than the essential parts of the articulation. In fact, the author would deny to rachitic leg deformity with in-knee any other term than *spurious* genu valgum, and

would consider *true* genu valgum to be that statical form in which the joint and its active and passive structures are alone affected.

Leaving out here all mention of adjuvant, dietetic, and hygienic influences, and the narration of all the known symptoms of in-knee, the author may state that a continued experience of over forty years has shown him that to cure in-knee two things are necessary—to keep the knee extended during the treatment by an *inflexible* apparatus, and to employ a similar contrivance to produce or to maintain, as the case may be, adduction of the leg. The opposite surfaces of the external articulating portions of the femur and tibia being held apart, nature fills up the gap between them; probably she ceases meanwhile to add to the inner side of the joint. The choice of apparatus rests with the surgeon. The author has already described elsewhere that which he has commonly employed.

We may here remark that genu valgum may be arrested and cured by being maintained in a bent position, as C. Hüter has stated. Better still would be to apprentice the patient to a tailor, in the work of which the patient needs to sit *à la Turque*. We have found advantage in causing children disposed to knock-knee to sit in that manner, and do much of their ciphering and reading in that position.

Infants even who cannot walk evince no pain or inconvenience from the treatment; in them the apparatus may also be removed for a couple of hours night and morning, it being permitted to them to spend their day upon the floor. The rule of treatment should be, "*arte non vi*" and "*nullum retrorsum.*" He would remind the surgeon of an axiom of John Bell, applicable to our present subject, to the effect that "force is only successful where it is not needed." If the utmost gentleness be used, nature will do her work well with the utmost docility. All bandages and straps require to be applied with a comparative looseness that may seem ridiculous to the inexperienced. Infantile cases are commonly remediable within two or three months, not needing afterwards retentive apparatus. Children of two or three years and upwards, able to walk, may wear the apparatus day and night, and walk about as much as they like in the day time, with reasonable rest, the apparatus being removed morning and evening for a *few minutes* only for cleanliness, and to enable the nurse twice daily to bend the knee-joint *once*. In all cases the patient may follow his nursery, school, or business vocation; as soon as it is found that the knees evince no decided tendency to return to the valgus form directly the controlling apparatus is removed, a gradually extended permission to habitually use the parts in full mobility may be given. It is rarely necessary in any but the severest, neglected, or unrelieved cases of non-rachitic genu valgum, even in the adolescent up to the age of sixteen or seventeen, to confine the patient to the couch or bed, to resort to tenotomy, or to forcible examination, or forcible straightening under anæsthesia.

When such rapid forcible measures are employed, much is apparently gained at the moment, but apparatus is much less easily borne after such treatment than without its use, and in the long-run the tortoise is apt to outrun the hare. If force be absolutely necessary, the author prefers the more modern form of applying force—supra-condyloid osteotomy, especially as carried out by MacEwen of Glasgow; for when this operation is properly performed, and the limb is secured in a straight position, a gap is formed on the external margin of the femur at the place of division, and the external condyle is lowered to a corresponding amount. By the consequent change of relation of the leg, the tibia is adducted and the genu valgum disappears. The gap in the femur artificially made is substituted, as it were, for the analogous gap felt in the straightened

genu valgum in the young by the surgeon's hand when the laxity of the joint permits the manœuvre.

After MacEwen's operation, nature fills up the triangular gap in the femur by callus, just as nature, by a return to natural growth where wanted in the femur and tibia, fills up the gap between the external condyle and the opposing articulating surface, when the surgeon, with the aid of mechanical apparatus, without osteotomy, has succeeded in holding those structures asunder, so as to enable her to act.

Mechanical treatment which can be carried out by the mother and nurse, when properly instructed and watched by the surgeon, is the proper remedy for all cases of genu valgum in early childhood, whether or no rachitic. The greater number of young adolescent cases of genu valgum, when taken in time, are also quickly recoverable without osteotomy. When, however, they have been long neglected, when they have lost all elasticity of structures, and the impairment of size of the external condyle and opposite articulating surface of tibia is considerable, when the patella has abandoned its natural groove between the condyles, MacEwen's operation is indispensable.

Osteotomy is then the complement to mechanical treatment, and it will strike the reader who has followed the author's description of the physical obstacle to cure afforded by the deterioration of the outer half of the articulating knee surfaces, that correct instrumental treatment and supra-condyloid osteotomy work towards cure in the same groove as regards "indication" of treatment.

In the rachitic cases termed genu valgum, but in which considerable curvature of thigh and leg bones has more to do with the distortion than any changes in the knee-joint, after the age of seven or eight years, when instrumental treatment can effect little improvement in the *curvatures*, multiple osteotomy can alone be relied on. It should, however, be remembered that a moderate amount of permanent curvature of thigh and leg bones is not incompatible with effective use in after-life.

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## *On the Pathology and Treatment of Genu Valgum.*

Mr. B. E. Brodhurst, London.

Genu valgum, or knock-knee, is a protrusion of the inner condyle of the femur inwards, through relaxation and yielding of the internal lateral ligament of the knee-joint. It is a constitutional rather than a local defect, and it is for the most part accompanied by a relaxed condition of the ligamentous system.

Genu valgum is, for the most part, preceded by flat-foot, and it may be said to be produced by it; for, as in flat-foot, the ligaments on the inner side of the ankle and those in the sole of the foot have yielded and become lengthened, so that the arch of the foot is more or less obliterated, and the foot itself everted, the inner malleolus is made to project inwards, and the shaft of the tibia is consequently inclined outwards. But, because the tibia is inclined outwards, the shaft of the femur is necessarily inclined inwards, and the superincumbent weight is no longer transmitted through the long axis of the femur and of the tibia to the arch of the foot, but it is directed unduly to the inner side of the limb, and is borne on the inner margin of the foot. Thus, in consequence of

the position of the foot and of the tibia and the femur, the internal lateral ligament of the knee-joint yields and becomes elongated, and the condyle protrudes. Then the condyles assume an oblique position in the articulation, the inner occupying a lower plane than the outer condyle.

The examination of numerous femora shows that there is generally some difference in length of the two condyles, and that the inner is somewhat longer than the outer. But it is also found that in the finest formed and strongest femora there is scarcely an appreciable difference in the length of the two condyles. Such a femur is in the Hunterian Museum, No. 320. This specimen is 20 inches in length, and its condyles occupy the same plane. On the other hand, a rickety femur may present an inner condyle one inch longer than the outer. This may be observed in a crooked and stunted femur, No. 2872$^c$, also in the Museum. In this instance the outer condyle is flattened, and the inner is elongated and nipple-shaped. These specimens may be seen varying in every degree between the most perfect bone and the most rickety. A moderately healthy bone shows a difference probably of $\frac{3}{10}$ths of an inch.

It is not to be understood that because the inner condyle is $\frac{3}{10}$ths of an inch longer than the outer there is therefore genu valgum, for such is not the case; but rickets obtains very widely throughout the population, and this is one of the signs of it.

When genu valgum is first observed, the foot rests flat on the ground, the arch has disappeared, and the inner edge of the foot is in contact with the ground. But if now the weight of the body is removed, the child being raised from the ground or placed horizontally, the knee will resume its normal shape and the protrusion will disappear, in the same manner as, under similar circumstances, flat-foot and scoliosis disappear.

At length deformity becomes permanent and obliquity of the femoral condyles increases. There is no new formation, but pressure is somewhat increased on the outer condyle, and this becomes atrophic, while it is diminished on the side of the inner condyle. And at the same time the biceps tendon becomes tense in proportion to the obliquity and the use of the limb; and, later, the external lateral ligament becomes tightly stretched, like a cord beneath the skin —rigid and unyielding, and the ilio-tibial band is drawn into a sharp line projecting on the limb.

Such, then, being the relative position of the bones and the soft structures, the question to be solved is, How shall the limb be restored so that a right line may pass through the long axis of the femur and the tibia and impinge on the dorsum of the foot?

Genu valgum is not often merely a local defect. It may, however, be induced by accident. Then it is seen without flat-foot. For the most part, however, the ankle drops and the foot is flattened and everted, as may be observed in figs. 13 and 14.

In the treatment of genu valgum, it is essential to restore the arch of the foot. Unless this is done, the knee deformity will return.

There are especially three modes of treatment which have been adopted, and to which I will call attention—namely, forcible straightening of the valgous knee, osteotomy, and tenotomy.

I will not refer to the mechanical treatment, for time does not permit of it, but in considering this question it ought not to be ignored.

Forcible straightening (*redressement brusque*) has been applied both to children and to adults. Eighteen years of age is fixed by M. Delore as the limit at which this mode of treatment may be practised. He directs that the limb is to be placed on the outer side, when, the foot being raised from the table,

manual force is to be applied to the inner side of the knee in a series of jerks. The limb is straightened in from five minutes to half an hour, and to effect this as many as eight assistants may be necessary. The epiphyses of the femur and the tibia are separated by this violence, and the external lateral and the crucial ligaments are ruptured, the external popliteal nerve may be torn through, the periosteum is lacerated, and suppurating periostis has followed; violent inflammation of the joint has been induced, and in a large number of cases a useless limb has resulted.

Rizzoli of Bologna endeavoured to straighten these limbs by instrumental means, force being applied suddenly; and Bardeleben employed an apparatus consisting of two cylinders enclosing the limb, which were applied during flexion. The limb was then forcibly extended and thus straightened.

I am compelled to conclude that such treatment is not surgery, and that, notwithstanding famous names, it is not likely to find followers at the present day.

Osteotomy has been practised by some in every case of genu valgum, even

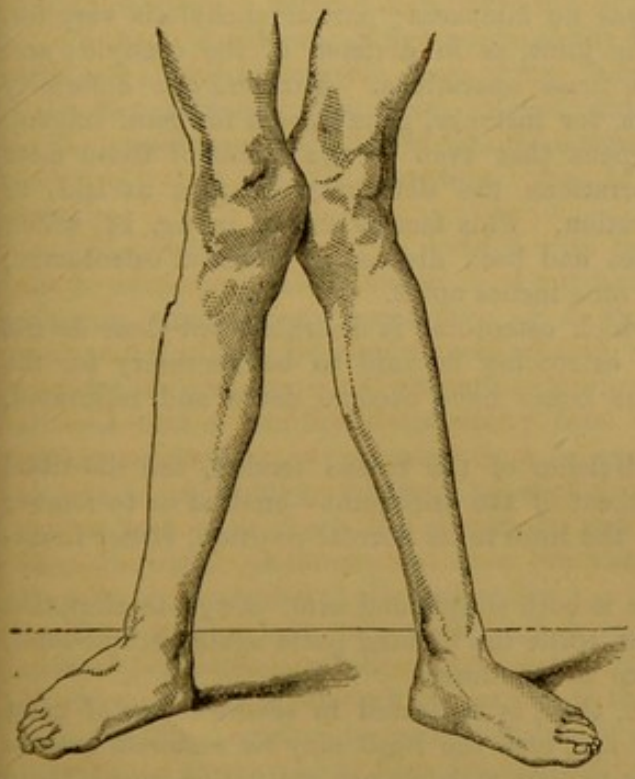

Fig. 13.

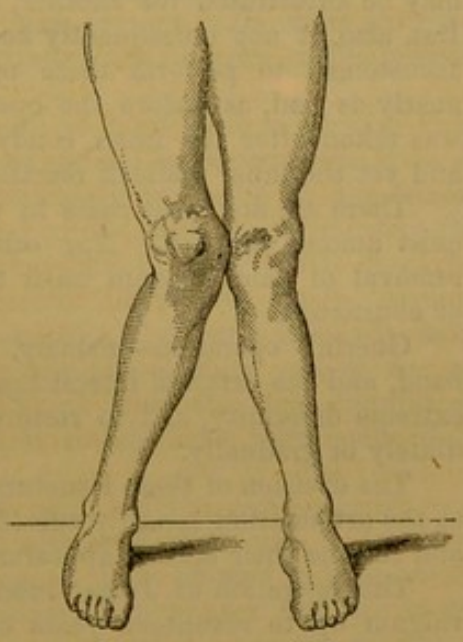

Fig. 14.

in the earliest years. Thus, some operate at the third year; others not earlier than the sixth year; while again there are those who prefer to operate at the ninth or the tenth year. At this age, and after, it is thought to be right to perform any number of osteotomies. Thus the femur, the tibia, and the fibula may be cut through, and perhaps in more than one place; or a wedge of bone may be removed from the shaft, and the bone then forcibly straightened, or the inner condyle may be divided. Thus, one author says: "Having operated on 557 limbs belonging to 330 patients affected with various osseous deformities requiring osteotomy, a few observations on the results may not be without interest. Of the 330 patients, 220 were affected with genu valgum, presenting 367 limbs for treatment. The remaining 110 patients were made up of bow-

leg, anterior and other tibial curves, and anchylosis of the hip and knee—in all, 190 limbs. Though 557 limbs were operated on, the number of osteotomies is not thereby indicated, as in many instances more than one operation was performed on one limb. This happened in those cases of genu valgum which were complicated by other curves, such as anterior tibial. In such the tibia and fibula were divided besides the femur, making three osteotomies in one limb. In tibial curves of old standing, the tibia and fibula had at times to be divided in two places each, making in this way four osteotomies in one limb. Bow-leg, when aggravated, often required the tibia and fibula to be divided in two places, and the femur in one, making in this way five osteotomies in one limb, or ten in each patient. So there have been 835 osteotomies performed on these 557 limbs. Both limbs were operated on at once; and, with few exceptions, all the osteotomies necessary for the correction of a deformity, or the deformities, were proceeded with at one sitting, as many as ten having been performed at one time." *

But, lightly as these osteotomies are spoken of, they are by no means harmless; for the growth of the limb is thereby impaired; lameness frequently is induced where previously there was no lameness; partial anchylosis very frequently follows penetration of the joint, as in division of the condyle; and pyæmia occasionally results from these operations. Further, one deformity may be substituted for another, as, for instance, genu varum for genu valgum. But, also, it not unfrequently happens that even in the hands of those most accustomed to perform these operations the deformity remains as bad, or nearly as bad, as before the operation. This fact is shown in fig. 14, which was taken after the inner condyles had been divided by a skilful osteotomist, and yet the inner malleoli remain nine inches apart.

There are doubtless cases in which osteotomy is desirable, but these do not exist among children. Nor can osteotomy be said to be necessary for the removal of genu valgum until the bones have become dense and indurated, or eburnated.

Guérin's operation—namely, division of the biceps tendon, the ilio-tibial band, and the external lateral ligament of the knee-joint—enables us to remove extreme deformity, and to restore the limb to its normal position, either immediately or gradually.

The division of these structures is both simple and safe. Any lateral motion at the articulation speedily subsides, while the divided parts again become solid, and resume their normal appearance and texture.

This operation of Jules Guérin, then, is indicated in severe forms of genu valgum up to seventeen years of age, and the limb may be restored both in shape and function. This involves neither violence nor mutilation, nor is there danger of pyæmia or of anchylosis.

It is, however, only in a small number of cases that it is necessary to have recourse to Guérin's operation in its entirety. Usually it is sufficient to divide the biceps tendon alone. I have never known any difficulty in restoring the normal shape of the limb without further division than of the biceps tendon only until fourteen years of age.

When genu valgum is complicated with femoral and tibial curves, these also will need attention. It has been my object not to complicate this question, but to treat only of genu valgum.

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* MacEwen, "Osteotomy," p. 161.

## *On the Pathology and Treatment of Genu Valgum.*

Mr. Henry F. Baker, London.

Great interest has been excited amongst surgeons during the last few years regarding the pathology and treatment of genu valgum, owing to the introduction of several new and ingenious operations involving the division of the bones forming the knee-joint, some by severing the internal condyle of the femur and pushing it up, and others by division of either the femur or the tibia and fibula, or all of them, and then placing the leg straight without any wound of the joint.

The consideration of this subject would, I think, be much simplified if the term "genu valgum" were restricted to that variety of knock-knee in which the femur and the tibia are both straight, and the deformity takes place at the joint itself, and thus some other name should be devised for the other kinds of knock-knee which depend on a curvature of bone not affecting the joint.

During a residence of five years at the Royal Orthopædic Hospital nearly 800 cases of genu valgum have come under my notice, and in nearly all of these the deformity took place at the joint itself, and was quite independent of the curvatures of the bones that were sometimes present in the same patients, and it is to this class of cases that I wish to confine my remarks on the present occasion.

The great question to be decided with regard to these cases is whether they can be cured by gradual stretching by means of splints or instruments with or without subcutaneous tenotomy; or is it necessary to resort to the heroic measures that have been advocated of late, and to divide the bones forming the knee-joint either with or without entering the joint? Although it must be allowed that a wonderful immunity from danger has on the whole followed osteotomy, it must at the same time be remembered that cases have happened in which suppuration followed by anchylosis has taken place, and in one recorded case even death has occurred; and I think from the large number of cases in which osteotomy has been practised, it cannot have been confined to very severe examples of this deformity.

Believing, as I do, that almost all cases of genu valgum can be cured by a course of treatment which, when carried out with reasonable care, is devoid of all risks whether to life or the free use of the joint, I am pleased to take advantage of this occasion to place before the Congress in a very few words my opinion as to the points that are necessary to be attended to in the treatment of genu valgum without division of bone.

The great majority of cases of this deformity that come under the notice of the surgeon are not of a severe nature, and on the knees being placed in the extended position and allowed to touch each other, the distance between the inner malleoli will be found not to exceed three or four inches. When this is the case, a very simple and inexpensive mode of treatment is all that is required to ensure a cure—viz., wooden splints applied to the outer side of the limbs, and extending from the hips to rather below the ankles, and having straps and buckles attached to them, by means of which the knee-joints can be gradually drawn towards the splints. If the patient is kept always entirely off his feet during the treatment (and this restriction is absolutely necessary), the deformity will soon be found to yield.

In those cases which are rather more severe, with from four to seven inches

between the malleoli (and indeed in the less severe cases if the expense of the instrument can be afforded), a special instrument should be prepared and made in such a way that it should keep the knee in the *extended* position, and prevent the leg *rotating*, at the same time that, by its means, the knee-joint can be acted upon in the *lateral* direction with sufficient force to gradually overcome the deformity. An instrument that was shown by me at a meeting of the Medical Society of London in February 1880, and a description of which was published in the *British Medical Journal* on May 22, 1880, carries out these conditions efficiently.

Cases are occasionally met with of so severe a nature that it is impossible to overcome the deformity, owing to the contraction of the biceps femoris, and sometimes of the external lateral ligament and the ilio-tibial band. In these cases it is necessary to divide one or other of these structures subcutaneously, and afterwards to gradually extend the limb. After the deformity has been overcome by the means indicated above, it is absolutely necessary that for some time the knee-joint should be supported, or a relapse will assuredly take place. A large number of cases in proof of the efficiency of the modes of treatment that I have advocated here can be seen in the practice of the surgeons at the Royal Orthopædic Hospital, where during my residence I have become convinced of the truth of the statements that I have made in this paper.

## DISCUSSION.

Mr. Barker, London: Mr. MacEwen and Dr Little appear from their papers to be quite in accord in the main as to the pathology of genu valgum. With their views the speaker agrees completely, and believes with them that there are many cases of genu valgum quite unsuited for operation, while a number remain where osteotomy is the proper measure to adopt. The particular stage of the affection it is which determines more than anything the procedure to be adopted for its treatment. Instrumental treatment will generally suffice for the stage of softening, but when this has passed, and the bone has become firm again, osteotomy is called for, and will always have a large field of usefulness. The whole question as to the admissibility of Professor MacEwen's operation appears to the speaker to turn in a great measure upon this point, *i.e.*, is it a dangerous proceeding or not? If it be not dangerous, why not substitute it for a lengthy, expensive, and troublesome treatment with instruments which render repeated visits to the instrument-maker and surgeon necessary, and which is not always even then successful? A parallel might be quoted in the instance of *pes equinus* which might be cured by prolonged mechanical treatment, but which no surgeon would now hesitate to straighten at once by tenotomy on the score of the risk of the operation. Professor MacEwen's 560 operations amply prove that his operation, properly performed, has no more danger about it than ordinary tenotomy; and from a careful study of the English, French, and German literature of the subject, the speaker believes that no charge of dangerousness can be brought against the operation from other quarters. In regard to the theory of causation of genu valgum put forward in the third paper read, *i.e.*, that it is due to stretching of the internal lateral ligament, it need only be said that, if it were correct, the treatment advocated by the author (Mr. Brodhurst), namely, to divide the external lateral ligament, would be most undesirable. By this measure the joint would be left without support either of the internal or external lateral ligament, and would become most unstable. The speaker had

seen a case illustrating this in a remarkable degree. The patient, an adult female, came to him at University College Hospital complaining of a loose knee-joint, with inability to walk with any comfort without instruments. She stated that several years ago her knee had been operated on in this way by no less fervent an advocate of the method of division of the external lateral ligament than Mr. Brodhurst. She now demanded an operation for genu vulgam, which was refused owing to the lateral instability of the joint. Lameness, pyæmia, anchylosis had been alluded to in the paper very freely, and as though of common occurrence after MacEwen's operation. Such allusions (the speaker submits) it would be most desirable to have carefully substantiated, seeing that they are at variance with all that can be gathered from the literature of the subject, home or foreign, which the speaker had carefully examined. The accidents alluded to had followed other modes of osteotomy, but it had yet to be shown to have followed upon MacEwen's operation. Such statements, unless definitely confirmed, were calculated to foster an unnecessary feeling of anxiety, and to be misleading.

M. Jules Guérin, Paris: The papers which have just been read show that there are as many opinions on the nature and mechanism of this deformity as there are methods of treating it. This divergence of opinion among conscientious and experienced practitioners appears to me to depend on a confusion as to the causes which lead to the deformity. It will suffice, I hope, that I should categorise the causes, and place them in relation with the characteristics peculiar to each form, in order to clear away this confusion. The observation of more than 300 cases of genu valgum, and an experience of more than forty years, have led me to recognise four classes of cause for genu valgum, and consequently four forms of the deformity. The first cause is active muscular contraction analogous to that which, in the ocular muscles, leads to strabismus, and in the foot to talipes. The special muscles which lead to genu valgum are the tensor vaginæ fasciæ and the biceps—the one inclining the leg laterally and angularly on the thigh, the other causing rotation of the condyles of the tibia on those of the femur, whence results the external rotation of the leg which so often complicates knock-knees. To these primary and special causes we must add retraction and subsequent shortening of the external lateral ligaments of the knee. This category of cause is indisputable, and I show a series of drawings of monsters and of infants, where examples of knock-knees accompanying other forms of convulsive disorders can be plainly seen. The second form of genu valgum is the result of great weakness of the articulation of the ligaments and muscles intended to assure the verticality of the bones entering into the formation of the joints. It is quite obvious that if the vertical force of gravity is superior to the resistance of the ligaments and muscles of the knee, the joint will yield in the direction of its least resistance. This resistance is least on the inner aspect of the knee, there not being any muscle or ligament to oppose those on the outer side. This result is the more likely as the horizontality of the femoro-tibial surfaces is not always absolute. This form of cause is frequently a local manifestation of a condition of general weakness. In such cases all the joints —feet, hands, fingers, and vertebral column—by their extreme laxity attest the special origin of the genu valgum. The treatment of this form of the disease naturally excludes section of either muscles or ligaments: it necessitates the use of instruments of support, together with local *fortifiants*, such as hydro-therapy and other analogous means. The third form of genu valgum is due to rickets; its mechanism of production is quite special. The genu valgum of rickets is almost always the result of an abnormal direction of the joint-surfaces,

together with the vertical force of gravity. In my researches on rickets I have shown that the deformities of the skeleton are under a developmental law, in virtue of which the deformities are produced from below upwards—from the tibias and fibulas to the femurs, from the femurs to the pelvis, and from the pelvis to the vertebral column. Thus, when the bones of the thigh and leg commence to curve, their articular surfaces take a direction transversely oblique, from within outwards, and so bring about a lateral inclination of the tibias on the femurs. To this initial cause comes now the action of gravity, which, finding the bony system weak, increases the obliquity of the joint-surfaces. From this twofold cause there results generally a lateral depression, which, after *redressement*, leaves a wedge-shaped interval capable of holding the finger, but which diminishes from without inwards. The local treatment which such an origin of the deformity requires should not be less special than the general treatment of the disease from which it results. Locally, orthopædic instruments, the recumbent position, cold douche baths, and tonics; the whole associated with the general treatment such as rickets requires. Finally, as the result of disease of the knee, the internal condyles of the femur occasionally force the leg outwards. This fourth form of genu valgum, not very common, is caused by means which do not require any very long explanation. It is the combined result of relaxation of the ligamentous structures of the joint and of the action of gravity constantly acting on a default of horizontality of the articular surfaces. The treatment required by such complex conditions must respond to their diversity—orthopædic instruments, section of the shortened muscles and ligaments, internal and general medication appropriate to the condition which has preceded the articular deformity. The special orthopædic instrument which I have used for more than forty years, and which is adapted for all forms of genu valgum, consists of a hollowed splint (*goutière*) jointed at the level of the knee, and articulated by means of a hinge which permits the leg-portion of the splint to rotate inwards, and so bring the leg gradually into the axis of the thigh. This graduation is effected by means of an arched screw, the nut of which arrests it at any desired point. Such are the four possible forms of genu valgum, as well as the methods of treating them. These means have always succeeded in my hands, and all subjects of this disease up to the age of seventeen years have been completely and permanently cured, notwithstanding the wedged-shaped interval between the tibia and the femur. As an extreme limit of what may be accomplished by this method of treatment, I may mention the case of a young Nubian aged seventeen years, the detailed account of which may be found in the "Report of the Hospital Commission on Orthopædic Treatment, 1849." But before closing these remarks, I would ask permission to add a few words on the two surgical methods which have recently been introduced for the cure of this deformity. I now refer to osteotomy undertaken with a view to re-establish the horizontality of the articular surfaces by removing the exuberant portion of the condyle, and to crushing of the condyle by *redressement*. Neither of these methods is necessary in young subjects, and any good result which may be obtained can only be secured at the risk of more or less grave accidents, or by destroying the normal function of the joint. One can hardly imagine that the excision or crushing of the internal condyle of the femur can leave an articular surface capable of facilitating the regular movements of flexion and extension. Moreover, I would add that up to the age of fifteen or sixteen years it is always possible by the means I have indicated to secure the re-establishment of the horizontality of the articular surfaces. If in adult life, however, any very considerable deviation should seem to indicate osteotomy, it may be tried; but there must always be the prospective of a very imperfect movement,

or even of anchylosis of the joint, as a consequence. There is, indeed, no other alternative to hope for.

M. Fochier, Lyons : As the successor of M. Delore, I am anxious to advocate the method by *redressement brusque*. Genu valgum results from an altered equilibrium in the knee-joint. Up to the age of ten or twelve years it is caused by a slight rachitic curve of the bones, either of the tibia (in, I believe, the majority of the cases) or of the femur, but close to the joint in either case. This proximity of the curve to the joint is a necessary condition to bring the weight of the body on to the external condyle of the femur, and its corresponding tibial tuberosity. Meanwhile, the internal condyle, relieved of all pressure, hypertrophies, and the disease thus appears as if due to this hypertrophy, while really the hypertrophy is itself secondary to curving of the shaft of the bone. In the genu valgum of adults, this condylar hypertrophy appears to be the primary condition, and to depend on hyperactivity of the "cartilage of conjugation" at the epiphysial junction. Whatever be the cause, however, *redressement brusque* always suffices for the cure, except when the disease occurs beyond the limit of age when the epiphysis is still separate from the diaphysis ; for the lesion produced by this method of treatment is a separation of the epiphysis from the shaft ; in this manner the deformity is corrected by attacking its cause at its very seat. This simple operation appears to me very much superior to osteotomy : I will point out some of its difficulties. These difficulties are not always *en rapport* with the age of the patient, although age doubtless has much to do with them. Thus I have straightened the knee of a tall and strong young woman aged nineteen years. The chief difficulties are experienced in cases where the rickets has been well marked and has been well cured. On one occasion I was quite unable to separate the epiphysis ; but a fortnight later, on making a second attempt, I was astonished at the facility with which detachment was brought about. The idea has suggested itself that some slight inflammation had been set up as the result of the first operation, which had materially facilitated the second. In some children there are symptoms of chronic inflammation of those joints which are the seat of genu valgum ; this is no contra-indication for *redressement brusque ;* it simply suggests a more prolonged use of the fixative bandage which is usually employed after the operation. In three cases in which I have operated the white swelling has only progressed once, and that only after the fixative bandage had been left off. An application of *ferrum candens*, and two subsequent applications of the bandages, sufficed to relieve this complication.

Mr. Keetley, London : I consider Mr. MacEwen's operation to be right in principle, successful in practice, and frequently necessary. But it is a fact that in a great number of cases osteotomy is not desirable. What is the best treatment for these ? I can scarcely think with Dr. Ogston that Delore's operation is suitable for all these. An operation which involves violence on bones and epiphysial cartilages can only be absolutely safe when followed by perfect after-treatment. What surgeon could invariably guarantee this ? Still Delore's operation is well suited for *bad* rachitic cases of genu valgum while the bones are still soft ; for in such cases, while, on the one hand, treatment by splints and instruments is often extremely tedious, and even, if ultimately successful to the bone, injurious to the ligaments of the joint, yet, on the other hand, antiseptic osteotomy can never be, at all events in human hands, other than a more or less serious proceeding. Weil has recorded two deaths from it in neither of which the knee-joint had been opened, one from septicæmia,

the other from suppuration; and six cases of Mr. MacEwen's had suppurated. Moreover, every rickety child cannot hope to be operated on with the experience and skill of Mr. MacEwen. But to go back to the milder cases with soft bones, cases fit for neither osteotomy nor osteoclasis. I have carefully tried Hunter's method of bandaging in the flexed position in about six cases for three months each. Some showed very little improvement, others none. Mr. MacEwen's mode of dealing with this class of cases appears to be medical and hygienic, added to never letting the children walk or stand independently. The first part of this treatment is unexceptionable; but the second is impracticable among the London poor, unless they are purposely crippled with splints too long for their legs, and what then becomes of hygiene? I am not one of those who join in the fashionable condemnation of *iron* instruments, but the old irons for genu valgum were wrong in principle. No one can study the beautiful work of Mikulicz, and the confirmatory statements of Busch, Weil, and others, and then compare them with what is to be seen every day in our own museums and out-patient rooms, without being convinced that the pathology of genu valgum has lately been far advanced, and on a firm basis. Now, in agreement with this pathology, the centre of lateral movement of the two levers of a genu valgum instrument should be, not at the knee, as in the old instrument, but considerably above it. I have ordered two such instruments, and I do not despair of, ere long, getting them into working order, though there are practical difficulties, especially those caused by the comparative shortness of the distance of the lateral hinge above the plane of the knee-joint; for one of the problems to be solved in constructing such an instrument was to protect the ligaments of the knee-joint from all strain. The old instrument used the whole leverage of the tibia to stretch the external lateral ligament, and sometimes, as is well known, and as I can positively affirm from observations made at the Surgical Aid Society, led to deplorable cases of loose and untrustworthy joints. Moreover, somewhere or another, elastic force must be used; for how otherwise can any effect be hoped to be produced on growing bones? The old instruments stole a little elasticity from the stretched ligaments. Lastly, I am pleased to point out the thoroughly international character of the history of progress in this department of surgery.

Mr. Clement Lucas, London: Looking at the pathology of genu valgum as stated in our text-books up to quite recent times, it is remarkable how all authorities agree that it is simply the result of relaxed ligaments. Nothing is said of rickets, yet who can deny that the knock-knee met with in this country is almost always the result of rickets? Rickets is a constitutional affection, which, besides causing softening of the bones, causes weakening of the ligaments and want of tone in the muscles. The affection of the bones is but one symptom, which shows itself first in the bending of the ribs, because these are constantly on the move; but I begin to recognise rickets even before the bone affections appear. If a child has a chronically distended abdomen, with a capricious appetite, and be backward in walking, it will most certainly develop rachitic bone changes unless the diet be attended to. It is quite an accident in the original direction of force whether the limb be bent in or bent out. It is very rare for us in England to meet with cases of genu valgum which can be traced to spasm of muscles. We have to deal with rickets. When the bones have become altered in shape and the internal condyle lengthened, the cases may still be treated by splints if the bones are still soft; but if sclerosis has taken place, operation is indicated. Rupture or division of the extended lateral ligament may be of service in slight cases, but it is not indicated in severe.

Osteotomy may be performed with the most trivial risk if it be done both subcutaneously and antiseptically. Whenever a new departure in surgical treatment takes place, it is first regarded with suspicion; next carried on with enthusiasm; and then comes a very strong conservation reaction, which would limit the operation almost to its exclusion. Some such conservative reaction osteotomy has now to contend against. It happened that for eight years I had attending under my care an extreme case of genu valgum, which resisted all treatment by splints both within and without the Hospital. He eventually ceased attending, not having gained any benefit. When Dr. Ogston published his paper some three or four years ago, my thoughts recurred to this case, and I wrote to the boy, and found him in the same deformed condition as when last seen. I divided his internal condyle and completely cured the deformity. Encouraged by this result, I have performed that operation many times since, in all some seventeen or eighteen times, and I have never had any ill result. I have divided both internal condyles on the same patient and on the same day, and then there has been no rise of temperature or fever to give one the slightest anxiety. I have never caused a stiff joint. The last case was performed last Tuesday. There has been no rise of temperature since. I have not used Mr. MacEwen's operation, and I do not therefore deny its efficacy. It is used by my colleagues, but the cases that have come under my own care have appeared to me to be most suitable for division of the internal condyle. I always commence movements three weeks after the operation. The operation has been performed also by my colleagues, and some thirty or forty cases must have been performed in Guy's Hospital, and in no case has there been any ill result.

Mr. Noble Smith, London: Our thanks are due to Mr. MacEwen for introducing a safer mode of performing osteotomy than those which had before been employed; but at the same time it would appear that osteotomy had been carried of late rather too far. There can be no doubt that in many cases, as in adults, no treatment short of division of the bones can remove the deformity; but in children under ten years of age—namely, at eight, at six, and even at three years of age—such an operation cannot be justifiable. Osteotomy is said to cure genu valgum in two months or ten weeks; if such is the result, the operation is certainly a brilliant one, but we have yet to learn the ultimate result of patients so operated upon. The few cases seen by the speaker of young children operated upon by osteotomy have been examples of a failure of the operation, or rather examples of relapse after that means of treatment. Osteotomy is not without some amount of danger; it is at all times, even by Mr. MacEwen's showing, a serious operation; and if we can cure cases, as we undoubtedly can, by means of instruments, ought we not to give every patient the chance of being treated, although tediously, yet with perfect safety, and in children with a certainty of success? The speaker had watched the progress of over one hundred cases treated at the City Orthopædic Hospital by instrumental means, and all had been cured, or were progressing towards recovery. These cases had not been left to the care of the instrument-maker, as had been suggested; and the speaker considered that if the surgeon was not capable of undertaking the entire manipulation of the instruments he employed, he had no right to undertake the treatment of such cases.

Mr. MacEwen, Glasgow, replied: As far as Dr. Little's and my own views concern flat-foot, they are sufficiently near to be considered together, and show that flat-foot does accompany genu valgum in the infant, or in one who

cannot or does not walk. When the person affected with that disease *does* walk, he attempts to maintain his equilibrium; this causes the toes to be turned inwards, and they attempt to grasp the floor; the flexor tendons are powerfully contracted, the instep of the foot is strongly arched, and the outside of the foot rests on the ground. Most cases of genu valgum are able to walk, hence the majority of such cases have arched insteps, and not flat feet. Mr. Brodhurst states that flat-foot is *always* present in *genu valgum*, and that unless it be first corrected, the genu valgum will return, notwithstanding any operation that may be performed for the relief of the latter. This statement is thus at variance with mine, and is disproved by the fact that I never touch or interfere with the foot in such cases; yet the deformity of genu valgum does not return after supra-condyloid osteotomy. Regarding M. Guérin's views of the etiology of genu valgum, I differ from them, with all due respect to such a distinguished physiologist and surgeon. I cannot see the slightest evidence for believing that the muscles have any action in the production of the curvature of the bones affected in genu valgum. I consider that the majority of cases of genu valgum are the result of rachitis, or at least occur after debilitating diseases of any kind. Under these circumstances the muscles, being flabby and weak, cannot act powerfully on the bones, and therefore cannot bend them. These are not theoretical statements, but are based on the observation of hundreds of cases. I have watched such from their earliest appearance, some of them being known to me prior to the appearance of genu valgum, and in a few of such I have observed them from month to month, and have never been able in one single instance to say that muscular action was the cause of the deformity. There are many other reasons which militate against the assumption that muscular action is the cause of genu valgum, but as these are given in detail in my work on osteotomy, I need not now refer to them. With the exception of the internal condyle, which is sometimes elongated, genu valgum is not an affection of the knee-joint, nor of the soft structures which surround the joint. It is an affection of the bones, principally of the distal third of the femoral diaphysis, occasionally also of the upper part of the tibia. This view is so far supported by Mikulicz of Vienna and Margary of Turin. Under these circumstances, any treatment must be directed toward the correction of the osseous deviation. When the bones are soft, this may be done by splints, attention to hygiene, and the like; when they are eburnated, osteotomy must be performed. In no form of genu valgum ought the soft structures of the knee-joint to be interfered with; the cutting of the external lateral ligament is never indicated, and if it be done will do harm. The application of splints and orthopædic apparatus, in marked cases of genu valgum with eburnated bones is useless. Cases have come under my notice in which splints have been applied in adults with bone condition as before referred to, and all of them had loose joints resulting from the overstretching of the external lateral ligament, which were not cured of their deformity until subjected to osteotomy. The great points to be aimed at in any operation for genu valgum are (1) to remove the deformity, (2) keep the knee-joint firm, (3) retain its movement entire, (4) do these in the shortest possible time, (5) and with the greatest safety to the patient. I hold that supra-condyloid osteotomy has all these advantages. With regard to Mr. Brodhurst's observation concerning the gravity of osteotomy, one might believe, from the manner in which he put a quotation from my book, following it by a string of disasters that had resulted from operations by osteotomy, that such disasters had followed my operations. I wish it to be distinctly understood that no such disaster has ever followed any osteotomy performed by me for genu valgum. I am glad, however, that that gentle-

man has disclaimed any intention of imputing such grave results to my operation. With regard to the results of the operation itself, I refer to my paper.

The Surgical Vice-President, Mr. HOLMES, then summed up the debate. He first remarked on the different views of the pathology of the disease entertained by such eminent surgeons as Dr. MacEwen, Dr. Little, Mr. Brodhurst, and M. Guérin, from which different plans of treatment necessarily result. Thus M. Guérin, with an ample experience extending over more than forty years, has never met with a case which in his opinion would justify osteotomy, while Dr. MacEwen, in a much shorter practice, had performed 522 osteotomies on 367 patients. Dr. Little trusts chiefly to orthopædic measures (though he does not reject osteotomy). Mr. Brodhurst and M. Guérin recommend for severe cases the section of the external lateral ligament, ilio-tibial band, and tendon of the biceps; and some other French surgeons agree with M. Fochier in recommending *redressement brusque*. And, further, those who practise osteotomy recommend several different operations. In this state of things, no definite opinion can be formulated, especially considering the very recent introduction of osteotomy in the treatment of the deformity. But one thing comes out very strongly from the discussion—viz., that proper care in the initial stages of the disease would in all cases prevent any such deformity as would justify operation; and this is one of the many proofs how much improvement is still to be desired in the public treatment of the chronic diseases of the poor. As to the method of osteotomy proposed by Dr. MacEwen, the information before the Section appears to prove that the operation is an adequate and a comparatively safe one, allowing that the deformity is severe enough to justify it. But further information is still needed as to what class of genu valgum indicates this or other forms of osteotomy, and also what the danger may be found to be. Any such danger, however, is not to be considered as an objection to the operation, provided that the deformity is so severe at the time the patient is first seen as to prevent any prospect of success from orthopædic measures, or that such measures have failed after a careful and skilful application. The mere fact that death has in some cases followed the operation, even allowing that it was directly caused by the operation, is no objection to it, if the surgical indications for its performance were adequate.

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## *Tracheotomy in Croup and Diphtheria.*

Professor GEORGE BUCHANAN, Glasgow.

1.—Tracheotomy is justifiable in diphtheria as well as in croup (if they are not identical diseases). This proposition might be considered unnecessary at the present time, but not many years ago it would not have been so. Even now, there are some who maintain that it is useless to operate in diphtheria, because it is a general disease, and the false membrane is but a local manifestation of it. I do not here wish to enter into the question of identity, but there is one phase of both affections which makes them clinically identical—that is, the tendency to death by suffocation. In order to make my position clear, I define diphtheria to be a disease, characterised

by an effusion of false membrane on the fauces, with a tendency to this membrane to extend through the glottis into the trachea, and even down to the bronchi and lesser bronchial tubes. In croup there is also the false membrane in the trachea, with a tendency to the membrane to extend downwards, but not upwards. The clinical difference then is the situation of the false membrane at the outset.

2.—*The type of the disease in which tracheotomy is justifiable.*—Both croup and diphtheria may be found in an asthenic as well as a sthenic type.

In the asthenic type the tendency is to death by rapid failure of the vital powers. Diphtheria occurs in this form much more frequently than croup. The false membrane is present, and often causes suffocation before the vital power is quite exhausted, but the asthenic symptoms show that the disease is exhausting the patient, independently of any want of air in the lungs. In this type tracheotomy should never be performed; it cannot save the patient, and scarcely, if at all, mitigates the suffering.

In the sthenic type the vital powers are not exhausted early, and the patient, if not relieved, dies of suffocation, caused by the diminution of size of the air passages by the false membrane, and by the frequent spasm of the glottis which accompanies it, and produces the stridor during inspiration so characteristic of it.

3.—*The stage of the disease in which tracheotomy should be performed.*—In a sthenic case, as soon as it becomes apparent that remedies are not producing any appreciable effect, and the disease is progressing unchecked, then tracheotomy should be performed before the struggles of the patient and want of proper aëration of the blood so tell upon the vital powers as to make recovery doubtful. I believe that in suitable cases more lives might be saved by early resort to tracheotomy; for the operation, if skilfully performed, is not a dangerous operation either *per se* or in its results.

4.—*The diagnosis of cases suitable for the operation.*—There are two signs which I rely on as indicative of necessity for tracheotomy:—

(*a.*) Stridor, indicating, on the one hand, a cavity ready to receive air if it could get access, and a passage constricted either by false membrane, or spasm, or both. The louder the stridor the more urgent the call for tracheotomy.

(*b.*) The movements of the chest as seen on inspection. I always cause the child to be stripped naked, in a warm atmosphere, and observe the movements. Approaching suffocation may be caused in these diseases either by the partial occlusion of the trachea, or by the bronchi and smaller bronchial tubes being stuffed with false membrane and viscid effusion. In this latter case the thirst for air—"*besoin de respirer*"—is as great as in the former, for the lungs, being already distended with air, imprisoned by the tough tenacious substance in the tubes, can neither contract nor expand efficiently, and as a consequence the chest remains puffed out and comparatively motionless.

In the former case the lungs are normal, ready to receive air, and the respiratory muscles are stimulated to the most violent efforts to expand and contract the thorax cavity. During the inspiratory movements the lungs fail to expand, in consequence of the air being denied entrance, and, as a result, all the flexible parts of the chest are sucked inwards, the intercostal spaces recede, and even the costal cartilages are bent in. In such a state of matters tracheotomy is urgently demanded.

5.—*The operation itself.*—I have nothing very special to bring forward. I adopt Trousseau's maxim, "Operate slowly, very slowly." The incision in the skin should be free, and should extend from nearly opposite, or little below

the cricoid for an inch and a half. Cut after cut must be made in the middle line until the trachea is visible. Everything which bleeds must be tied; and I have no hesitation in notching upwards, and even in dividing, the isthmus of the thyroid body. I have never seen hæmorrhage from it which could not easily be commanded by a ligature. Whatever bulges into the wound—veins, cellular tissue, apices of the thymus gland—must be held aside by retractors, and the golden rule I believe to be, "Never plunge the knife into the trachea till the white rings are clearly seen in the bottom of the wound."

*Chloroform.*—The greatest advantage is obtained by administering chloroform when the child is vigorous and the struggles are severe; but when there is a tendency to asphyxia, as indicated by the blue, cold, and clammy face, it is both unnecessary and dangerous. Often I have operated with success when the patient lay without wincing much, owing to the carbonisation of the blood. The opening of the trachea restores the colour and warmth in an incredibly short time.

6.—*After-treatment.*—I order all drugs to be discontinued, and trust solely to nourishment and attention to the tube. I have no reason to give for this but the success which has followed its adoption.

The tube must be kept cleansed from any mucous or other discharge, and from the viscid secretion which so often tends to block it up. The nature of the material which sticks in the tube is often indicative of the progress or recession of the original disease. If it is very viscid, and easily dries and crusts the tube, the respiration has a whistling sound, and indicates an inflammatory condition of the mucous membrane further down, always an unfavourable complication. In such a case it is necessary to take a feather moistened with tepid water and pass it down through the tube into the trachea and mop it out freely. Often I have seen a pellet of thick inspissated mucus shot out after such mopping, giving rise to free respiration.*

When the secretion is copious mucous, even purulent, there is little tendency to obstruction, and the chief trouble is to clear it away from the mouth of the tube, but often it is freely coughed out through the tube.

Viscid, easily-dried secretion with whistling respiration is an unfavourable sign, while copious moist secretion with much gurgling and coughing is a good sign.

The atmosphere around the patient should be temperate, fresh, and free from draughts; if possible, regulated to about sixty-five degrees of heat.

I have given up the keeping the patient in a steam-bath by imbuing the atmosphere with steam introduced under a cover or tent. I am satisfied it is both unnecessary and injurious, the patient being kept in a state of perpetual moisture from his damp clothes.

Many years ago I found that in spite of this precaution the secretion in the tube became viscid and dry in a case with tendency to this kind of effusion. In one case, after I had mopped out the trachea and got away the offending pellet, the attendant kept a piece of sponge moistened with hot water over the mouth of the tube, which kept the entering air moist within. Since that time I have trusted to the use of a moist hot sponge when necessary, but I believe even this is not required. A bit of gauze laid loosely over the tube acts as a respirator in moistening the air, but in my last case I found even this not to be necessary, and in future I shall dispense with all precautions except keeping the temperature equable, and seeing that the patient is not exposed to draughts, with a bit of gauze over the mouth of the tube for a day or two.

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* See *Brit. Med. Journal* for April 10, 1880.

7.—Statistics of my own practice:—Total tracheotomies for any cause, 54; cured, 22; died, 32. Total tracheotomies for croup and diphtheria, 50; cured, 19; died, 31. Tracheotomy for croup, 17; cured, 7; died, 10. Tracheotomy for diphtheria, 33; cured, 12; died, 21.

Taking the two diseases croup and diphtheria together, tracheotomy saved the lives of one out of every $2\frac{2}{3}$ children operated on, who would undoubtedly have died if the operation had not been performed.

## DISCUSSION.

Dr. Leale, New York: The very valuable paper, viz., the question raised by Professor Buchanan, "Is Tracheotomy Justifiable in Diphtheria?" may be answered in the affirmative, but I believe that it should be performed only *after all other means have failed, and when death is impending by suffocation.* I have operated where the disease extended below the bifurcation of the trachea, and when with the small elbow forceps and sponge the passage has been cleared, while the patient was held with head lower than the thorax to expedite the drainage of the mucus by gravitation. I once had recovery follow where the alarming symptoms were only so far relieved after two hours' efforts at artificial respiration that the patient could breathe independently of assistance from the physician. I have been called to perform tracheotomy when the patient presented the most unfavourable symptoms, and when cyanosis had existed, and when physicians present have said recovery could not follow, but when after an emetic has produced its effects, and the expectoration facilitated the removal of the exudation, the patient recovered, without an operation. I firmly believe that tracheotomy is sometimes performed where the danger has been increased by the new incision becoming a starting-point for the rapid advance of the disease, and also that it is our absolute duty to give the patient this his only chance for recovery, after all other means have failed, an operation by which death by apnœa can be averted.

Dr. Ranke, Munich: It has given me real pleasure to listen to Dr. Buchanan's paper, because Dr. Buchanan's views coincide thoroughly with my own experience of tracheotomy in cases of diphtheria. I have performed tracheotomy since April 1878 in twenty-six cases, and sixteen out of the twenty-six have recovered. I lay the greatest stress on early operation. If a child with pharyngeal diphtheria has become hoarse, and shows laryngeal stridor and a difficulty of breathing, which has already led to ever so short an attack of real dyspnœa, that child ought to be operated on at once. At least 90 per cent., if not more, of patients in that condition are sure to die if the operation is not performed. This being the case, tracheotomy should not be delayed, as experience has shown that it makes all the difference in prognosis, whether the operation is performed when the child is still comparatively strong or when its powers have been brought down by the disease to their minimum. In Munich, for instance, tracheotomy was formerly invariably unsuccessful in all cases of diphtheria, because it was always performed too late. I have amongst my recoveries a girl, five years of age, on whom I operated although she had pneumonia of the left lower lobe. But the child being still strong, tracheotomy was performed, and resolution of the pneumonia took place on the sixth day after the operation. I also preserve between two glass plates the bronchial cast of a boy of the age of six years and eleven months, which cast represents a regular tree of ramifications down to the smallest bronchi. That boy recovered, having been operated upon while he had still sufficient strength to cough up the membranes.

Age of course has a great deal to do in regard to prognosis. Of the 26 cases, 4 were between two and three years old, and of these 3 died; 9 were between three and four, and of these also 3 died; 6 were between four and five years old, and of these 2 died; of 4 between five and six 1 died, and also 1 of 2 between six and seven years. The only patient who had attained his seventh year recovered. Older children being, *cæteris paribus*, stronger, have correspondingly a greater chance of recovery. As to albuminuria, in 4 of the 16 cases that recovered, the urine had not been examined. Of the remaining 12 cases, albumen was found to be present in 9, and in 3 only it was never discovered during the whole time of treatment. Besides an early operation when once laryngeal symptoms have fairly established themselves, I lay the greatest importance on systematic ventilation in the treatment of diphtheria generally. I insist upon having two rooms at my disposal for the treatment of every case of diphtheria, whether tracheotomy be performed or not; the one room being kept with its doors and windows wide open, whilst the sick child occupies the other. At least twice a day, and each time for several hours, the rooms are exchanged, so that the patient has always pure air to breathe. In winter time the temperature of the room which has been ventilated must of course be raised again to its normal standard before the child is brought in. This *two-room treatment* (zwei-zimmer Behandlung), as it is called, I have carried through, I might say, pedantically for the last five or six years, and my pupils have adopted it from me; and I have every reason to attribute to it the greater part of my and their good results in treating diphtheria. Bed-curtains and other hangings, as well as carpets, I banish as much as possible from the sick-room. A third point in procuring good results after tracheotomy, and also a point of vital importance, lies, in my opinion, in the nursing. One ought to have particularly skilled and experienced nurses for tracheotomy cases. The management of the tube, principally during the first day or two, requires a great deal of skill and often a quick resolution, which can only be gained by manifold experience. I therefore always take my nurses for tracheotomy cases from *one* institution, in order gradually to get there a staff of really experienced nurses. As to medicinal treatment, I generally use none after tracheotomy, vapour inhalations being almost my only remedy, and I even only use these when expectoration becomes viscid. As to food, I exclusively give good broth and milk; wine I administer only in cases of decided weakness.

Mr. Golding Bird, London: Assuming the operation of tracheotomy to be done early, as I hold it should be, two questions of after-treatment result. The opening being made in the trachea, if the wound is held open with dressing forceps and a light is reflected into the windpipe, the posterior wall may be seen, pink, clear, with longitudinal rugæ. Such is the healthy appearance, and shows the membrane lying in the thyroid region above the wound. In such a case a tracheotomy tube may be employed. If, however, membrane is seen in the trachea, or if, after wearing the tube, membrane begins to appear, the tube should be withdrawn and the wound of the trachea kept open with a dilator, in order that the least forcible resistance may be offered to the passage outwards of mucus or membrane. Cleaning with feathers, &c., also is rendered easy. No harm follows the use of the dilator; for even if slight abrasion does take place at the level of the original wound in the lateral tracheal walls, there is not the same fear from it as from ulceration, the result of the silver tube. This latter causes, when it does so, ulceration in the anterior wall low down, and is in danger of entering the left innominate vein. But by whatever means the tracheal wound is chosen to be held open, I maintain that this method is

mechanically superior to that with the tube, where membrane, thick mucus, or indeed any foreign body, has to be expectorated.

Dr. JACOBI, New York: Tracheotomy is indicated when there is *strangulation*, and the *seat of it is in the larynx*. No sort of complication gives a contra-indication, *not even pneumonia*. The latter will increase the number of inspirations (*which are slow in uncomplicated croup*), and has been taken to form a contra-indication to the operation. To operate late is always a disadvantage. When I could operate early (seventy cases published 1868), I had 20 per cent. of recoveries. Since that time, being called to operate late, I lose many more, and have lost fifty cases in succession. Where the symptoms of laryngeal stenosis are present, being frequently relieved then getting worse again, the case may be one of catarrh, and may sometimes be left alone. Mr. Golding Bird thinks slight ulcerations are not harmful. I have an exception to that rule, losing a child eight days after tracheotomy from hæmorrhage originating in just such a tracheal ulceration.

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## *On the Surgical Treatment of Croup and Diphtheria by the Introduction of Tubes into the Trachea through the Mouth.*

Mr. WILLIAM MACEWEN, Glasgow.

Mr. MacEwen, referring to the introductory remarks made by Dr. West on "tubage," stated that "tracheal catheterism" and tubage were widely different. Tubage of the larynx was introduced into France by M. Bouchut. He used an instrument of a cylindrical shape, measuring one inch in length, and furnished with two elbows at its upper part designed to rest on the vocal cords. A thread of silk was attached to this tube whereby it could be removed from the larynx. Whatever merits this instrument may have had, M. Bouchut could not furnish any case which had recovered after this tube had been used. This fatality might have been the result of the original disease quite independent of the instrument. On the other hand, "tracheal catheterism," as introduced by the author some years ago, consisted in using a tube 10 to 14 inches in length, the one extremity of which was passed into the trachea, the other projecting from the mouth. The method of introduction and the details of several cases were given in full in the *British Medical Journal*, July 24th and 31st, 1880, and to these the reader is referred.

The first two cases in which tracheal catheterism was practised by the writer were adults suffering from œdema glottidis. In one the œdema resulted from a severe scald; in the other it supervened on chronic ulceration of the larynx. They were both sent into the Royal Infirmary, Glasgow, with the intention of having tracheotomy performed, and if tracheal catheterism had not been practised, an opening into the trachea would have been imperative. In both, the tubes were finally withdrawn after thirty-six hours, having answered the purpose for which they were inserted. In two cases of children this treatment has been practised by other surgeons. One by Dr. Fraser of Birkenhead, in a child affected with croup, the other by Dr. Lantnany, also in a child similarly affected. In both these cases these surgeons were satisfied with the manner in which the tubes acted, and in both recovery was complete. In one case of

croup with false membranes (diphtheria?), occurring in a boy of eight years, the catheter was inserted, and false membranes were expelled through it, and the patient breathed freely and easily. This patient recovered from the laryngeal affection, the tube was withdrawn, and he was pronounced convalescent. Four days later he became affected with pneumonia, from which he afterwards died.

These, then, are five cases in which these tubes have been used with success, four of these having recovered and are living at the present time; the fifth recovered from the laryngeal affection but succumbed to pneumonia contracted during convalescence. So that it may be said that the five cases hitherto tried have been successful.

Besides these cases, tracheal catheters were introduced in two patients who had to undergo operations for the removal of epithelioma of the back part of the tongue and pharynx. Instead of performing tracheotomy and introducing Trendelenburg's tamponade canula, the catheter was introduced into the trachea, the upper portion of the glottis being occluded by an india-rubber ring placed on the outside of the catheter so as to increase its external circumference. Chloroform was administered through the tube, which projected several inches from the mouth. It was thus easy to exhibit the anæsthetic during the continuance of the operation, while it in no way interfered with the operator, and it also prevented blood from entering the trachea.

## DISCUSSION.

Dr. ALEX. ROBERTSON, Glasgow: I saw three of the patients referred to by Mr. MacEwen, who were shown by him at meetings of medical societies in Glasgow. They had all fully recovered. In one of the cases the tube had been in the larynx for thirty-six hours continuously, except for a few minutes on two or three occasions. I carefully observed in that case the state of the voice. It was perfectly normal, and the patient said it was as strong and clear as ever it was. I also saw the child aged eight years spoken of by Mr. MacEwen, after the tube had been in for three days. The little patient was quietly breathing through the tube, which projected about three inches beyond the lips. By examining with a lighted candle, I satisfied myself that the breathing was *through* the tube, not by the side of it. The respiratory murmur was heard by me at the base of the lungs, showing that the admission of the air was quite free. Fearing that the tube might act as a conductor to fluids in any attempt to swallow, I satisfied myself on that point by causing the nurse to give the child a spoonful of milk, which it swallowed with little difficulty, obviously none going into the larynx. Mr. President, I regard this mode of treatment, which I think is mainly Mr. MacEwen's, though M. Bouchut previously made some not very successful attempts by a somewhat similar method, as a valuable addition to the means of treating a most important class of cases. How far, however, it may supersede the operation of tracheotomy it would be premature to say.

## *Traitement de l'Angine couenneuse et de la Diphtérite par la Dissolution des Fausses Membranes au Moyen de la Papaïne.*

Dr. Bouchut, Paris.

La diphtérite est une maladie primitivement locale devenant rapidement infectieuse, et elle peut être guérie sur place par les agents antiseptiques et par les dissolvants de la fibrine parmi les quels il faut citer la *papaïne* extraite du *Carica papaya.*

Les faits que j'ai déjà publiés en 1879 sur la dissolution et la digestion rapide des ténias des lombrics et des fausses membranes du croup devaient me conduire au traitement de l'angine couenneuse par la *papaïne.*

J'avais dû hésiter dans ces essais par la raison que si des fausses membranes baignant dans une solution de papaïne se dissolvaient et se digéraient en quelques minutes, il n'en est pas de même pour des fausses membranes diphtéritiques collées sur les amygdales et qu'en ne fait que laver avec un pinceau chargé de papaïne. Les deux expériences n'ont rien de comparable.

Mais, M. A. Wurtz de l'Académie des Sciences ayant montré que la fibrine, imprégnée par une solution concentrée de papaïne et lavée ensuite sous un filet d'eau pendant un jour, n'en était pas moins bien digérée et convertie en peptone si on la mettait dans une étuve à 40°.

Dès lors, je fus en droit d'appliquer la papaïne au traitement de l'angine couenneuse, à son début, puis qu'il suffisait de toucher la fibrine avec un pinceau imbibé de solution de papaïne pour en espérer la dissolution.

Je mis alors, indistinctement, toutes les angines couenneuses qui entrèrent dans mon service de l'hôpital des enfants, les cas graves ou légers, avec ou sans adénite cervicale, avec ou sans albuminurie, de manière à ne pas laisser croire à un choix des cas les plus favorables, et d'une guérison facile par toutes les méthodes de traitement.

Je pris une *solution de papaïne* à deux grammes pour huit grammes d'eau distillée avec cinq centigrammes de salicylate de soude pour éviter toute fermentation.

Je suis arrivé au chiffre de 46 cas d'angine couenneuse traité de cette façon, et sur ces 46 cas j'ai eu 40 guérisons.

Un de ces enfants avait en même temps une otite membraneuse du conduit auditif externe dont la pellicule diphtéritique très épaisse a été dissoute et digérée en 24 heures.

Dans plusieurs autres cas, il y avait de la conjonctivite diphtéritique et la fausse membrane touchée plusieurs fois par jour avec la papaïne a disparu. C'est un très bon traitement de la conjonctivite diphtéritique.

Les fausses membranes ne se dissolvent pas instantanément comme dans un verre, mais elles l'amincissent graduellement ou se détachent saus reparaître, et en trois jours les enfants sont guéris.

Il suffit de toucher les fausses membranes toutes les deux heures avec un pinceau imbibé de la solution, et dans l'intervalle je fais faire des gargarismes ou des douches dans la gorge avec une solution de benzoite de soude, de bicarbonate de soude ou d'eau phéniquée a deux grammes pour 1000 grammes d'eau.

## *Des Résections articulaires chez les Enfants, et de leur Influence sur l'Accroissement des Membres.*

Prof. L. Ollier, Lyons.

Les résections articulaires donnent en général de bons résultats immédiats chez les enfants, c'est sans doute pour cette raison que quelques chirurgiens les ont multiplées outre mesure et n'ont pas craint de réséquer chez de très jeunes enfants des articulations qui eussent guéri toutes seules avec du temps, une bonne hygiène et un traitement local approprié. Eu égard à la fréquence des maladies articulaires dans l'enfance, les résections sont rarement indiquées, mais il est des circonstances cependant où elles constituent le seul moyen de sauver le membre et la vie du sujet. Dans les trois premières années de la vie, les suppurations articulaires, lorsqu'elles ne sont pas le résultat d'une maladie générale que rendrait inutile toute opération, guérissent le plus souvent par l'ouverture des foyers purulents ; il est des cas cependant où il faut enlever des épiphyses ou des portions osseuses nécrosées. L'affection tuberculeuse des os et des articulations sévit durant toute l'enfance et la jeunesse, mais ici encore bien que l'ablation de toutes les parties malades paraisse théoriquement le meilleur moyen de guérison, on obtient de très beaux succès par l'expectation, tant sont énergiques les tendances réparatrices de la nature, surtout lorsqu'on peut les aider par toutes les ressources de l'hygiène combinées avec une thérapeutique rationelle. Parmi ces ressources qu'on ne peut malheureusement procurer qu'à un petit nombre d'individus, je citerai le séjour sur les bords de la Méditerranée et les bains de mer. La rénovation organique que s'opère sous l'influence d'une athmosphère maritime change bientôt l'aspect misérable des enfants scrofuleux lorsque leurs organes internes ne sont pas altérés. Cet effet se produit rapidement sur ces sujets étiolés qui pullulent dans les quartiers pauvres des grandes villes. On les voit prendre du teint et de l'embonpoint, et des articulations fongueuses et criblées de fistules ne tardent pas à se cicatriser. C'est là un des meilleurs arguments en faveur de la curabilité de la tuberculose, de la tuberculose du jeune âge en particulier.

Mais ce n'est pas sur ce point de vue des résections que je veux insister. Je tiens surtout à démontrer les inconvénients des résections pratiquées chez les enfants au point de vue de la croissance des membres. Nous trouverons dans ces considérations, non pas un argument contre la résection entreprise pour sauver la vie des malades, mais un motif de plus pour persévérer dans le traitement expectant lorsque la vie n'est pas immédiatement menacée.

Toute résection articulaire pratiquée dans l'enfance nuit à l'accroissement ultérieur du membre. C'est là une proposition que j'ai appuyée autrefois sur de nombreuses expériences faites chez les jeunes animaux, et que je crois utile de démontrer encore, en précisiant le danger spécial de diverses résections. Comme les résections chez les enfants ont été beaucoup plus souvent pratiquées en Angleterre qu'en France, ou pourrait trouver dans ce pays de riches matériaux pour résoudre la question ; malheureusement je ne connais pas depuis les beaux travaux de M. Humphry de Cambridge, de travail d'ensemble sur ce sujet, et je n'ai pu me procurer tous les documents que j'aurais désiré ; mais il y a là un sujet intéressant qui tentera, il faut l'espérer, le zèle de quelques jeunes chirurgiens. C'est par la comparaison de cas nombreux, observés longtemps après l'opération, qu'on pourra combler les lacunes que je laisserai nécessairement dans ce travail.

Rien ne peut empêcher un certain degré d'arrêt du développement en longueur, quelque réguliers que soient les processus réparateurs et quelque abondante que puisse être la régénération des extrémités osseuses. Aussi faut-il se méfier des observations dans les quelles un chirurgien, un peu trop épris de son œuvre, annonce qu'à la suite d'une résection d'une grande articulation il a trouvé après la guérison complète les deux membres parfaitement égaux. Il y a là une erreur d'appréciation. Jamais chez les animaux auxquels j'avais pratiqué des résections articulaires, ou diaphysaires étendues, je n'ai trouvé pareille égalité.

Les membres peuvent paraître égaux quelques semaines après l'opération, au moment de la guérison de la plaie ; mais l'inégalité entre les membres opérés et le membre sain se prononce de plus en plus.

Sur une jeune fille de 15 ans à qui j'avais enlevé la moitié supérieure de l'humérus, 125 millimètres, j'ai constaté quelques mois après, une différence de moins d'un centimètre entre les deux bras, mais trois ans plus tard cette différence était de six centimètres.

La méthode sous-périostée, qui a tant perfectionné les résultats des résections au point de vue orthopédique et fonctionnel, est impuissante à nous redonner la longueur de l'os réséqué. Jamais même dans les cas les plus favorables, l'os ne prend sa longueur normale. Si la portion reproduite peut être aussi longue que la portion enlevée, elle est toujours plus courte que la portion analogue du côté opposé ; celle-ci continuant à grandir, la première n' ayant plus les moyens de s'accroître en longueur ou ne les ayant que d'une manière transitoire et imparfaite.

J'ai bien constaté dans mes expériences la reconstitution temporaire d'un cartilage de conjugaison et la persistance d'une couche cartilagineuse entre les noyaux de réossification épiphysaire et la masse diaphysaire de nouvelle formation ; mais ce cartilage ne tarde pas à disparaître en s'ossifiant et l'os ne peut plus grandir en hauteur. Dans le cas de résection d'une extrémité osseuse, cette inégalité entre l'os réséqué et l'os sain du côté opposé, s'accuse d'autant plus que l'extrémité retranchée prend une plus grande part à l'accroissement normal de l'os.

Les diverses résections des grandes articulations diffèrent considérablement eu égard à l'arrêt d'accroissement auquel elles exposent. La loi d'accroissement des os des membres, telle que je l'ai depuis longtemps formulée, nous fait prévoit ces différences.

Au membre supérieur, pour les os du bras et de l'avant-bras, ce sont les extrémités éloignées du coude qui prennent la plus grande part à l'accroissement du membre ; d'où l'arrêt de l'accroissement de l'humérus après la résection de l'épaule, et l'arrêt d'accroissement de l'avant-bras après la résection du poignet chez les jeunes enfants. D'où par cela même le peu d'inconvenient relatif de la résection du coude, bien qu'elle porte sur les trois os qui constituent cette articulation.

Au membre inférieur, au contraire, les os de la cuisse et de la jambe, ce sont les extrémités constituant le genou qui prennent la plus grande part à l'accroissement du membre. Delà la gravité des résections totales du genou pratiquées dans l'enfance que j'avais annoncée dès mes premières expériences et que les faits cliniques sont venus depuis lors confirmer surabondamment.

C'est l'ablation du cartilage de conjugaison qui est la cause immédiate de l'arrêt d'accroissement. Delà la nécessite de diviser les résections articulaires eu résections intra-épiphysaires, c'est-à-dire, coupant l'os dans la substance de l'épiphyse et laissant intact le cartilage de conjugasion, et en résections ultra-épiphysaires, c'est-à-dire coupant l'os au delà de l'épiphyse et enlevant en même temps le cartilage de conjugaison.

Mais il ne faudrait pas croire que les résections superficielles intra-épiphysaires, laissant intact le cartilage de conjugaison soient sans influence sur l'accroissement ultérieur de l'os. Si l'on mesure, après l'enlèvement de la croissance, les os qui ont subi ces résections superficielles, on les trouve toujours sensiblement plus courts que les os sains analogues. Il n'y a pas seulement le déficit osseux dû à la portion enlevée et qui ne s'est pas reproduite il y a aussi un arrêt d'accroissement de l'os dû à la perturbation qu'a éprouvée la nutrition du cartilage de conjugaison. Pour le genou cet arrêt d'accroissement peut aller jusqu'a 5 et 6 centimètres. C'est que sans doute quand on réfléchit aux arrêts d'accroissement de 20 et 30 centimètres qui peuvent être le résultat d'une résection totale ultra-épiphysaire pratiquée dans l'enfance, mais c'est un motif de plus pour insister sur les moyens de traitement plus conservateurs encore que la résection, tels que l'immobilisation prolongée, l'arthrotomie antiseptique et l'évidement ou le raclage des fongosités articulaires. On doit d'autant plus insister sur ces moyens que l'ankylose du genou, et même de la hanche en bonne position, laisse en général un membre plus utile qu'un membre réséqué.

Si les fongosités des arthrites ou ostéo-arthrites chroniques donnaient lieu fatalement chez les enfants, à la généralisation de la tuberculose on devrait agir autrement. Il faudrait se hâter de réséquer et même souvent d'amputer le membre ; mais il n'en est pas heureusement ainsi et la plupart des arthrites fongueuses suppurées, arrivent à se cicatriser, chez les enfants, lorsqu'elles ne sont pas précédées ou accompagnées de lésions tuberculeuses internes. Une arthrite fongueuse est sans doute une source d'infection générale, mais elle est aussi la manifestation d'un état diathèsique, antérieur à l'évolution du produit tuberculeux.

Sans cette double notion étiologique de la tuberculose, il est impossible de se rendre compte de la marche de cette maladie. En ne tenant compte que de l'infection qui a sa source dans l'arthrite fongueuse, on est porté à accorder trop de confiance au traitement local qui a pour but de supprimer le foyer infectieux ; et on attribue à la résection une influence curative que l'avenir dément malheureusement dans beaucoup de cas. Il ne faut pas oublier cet effet que toute ostéite ou arthrite développée spontanément chez un enfant ou un adolescent doit inspirer les plus grandes craintes pour la santé future. La phthisie pulmonaire fera dans l'avenir de nombreuse victimes dans cette catégorie de sujets.

D'autre part, en ne voyant que l'état diathèsique, on s'endort sur les dangers de la lésion locale et on laisse le poison tuberculeux infecter, par sa résorption, les divers systèmes organiques ; surtout quand on n'a pas soin de l'annihilir par la destruction des fongosités au moyen du fer rouge ou des divers caustiques chimiques.

C'est pour cela que pour certaines articulations en particulier, genou, cou-de-pied, carpe, tarse, l'ouverture antiseptique des foyers articulaires ou osseux (épiphysaires ou juxta-épiphysaires) le raclage des fongosités, l'évidement des foyers tuberculeux intra-osseux, la cautérisation ultérieure des tissus abrasés, constituent chez l'enfant des moyens de guérison qui nous dispenseraient souvent des résections totales.

Chez l'adulte, on doit se comporter autrement, et l'on ne peut guérir que par l'amputation des lésions qui cédent chez l'enfant à l'ablation des quelques parties nécrosées ou à l'abrasion ou à la cautérisation des fongosités. On ne saurait trop s'appesantir sur la différence du traitement des maladies osseuses selon les âges. Un os jeune se rapproche beaucoup plus des parties molles qu'un os d'adulte. S'il devient plus souvent malade, il guérit encore plus

facilement ; les processus réparateurs étant d'autant plus rapides que le sujet est plus jeune.

Et cependant il ne faut pas s'illusionner sur la valeur des opérations élémentaires qui peuvent dans certains cas nous dispenser des résections. La résection totale d'une articulation est un moyen de guérison plus sûr plus durable ; plus on enlève de tissus, plus on dépasse les limites des foyers tuberculeux, plus on a de chances de faire une opération radicale. A ce point de vue l'amputation serait préférable à la résection ; mais si nous différons la résection à cause des inconvénients qu'elle a au point de vue de la croissance du membre, à plus forte raison doit-on rejeter une mutilation irrémédiable.

Ces réserves faites sur le pronostic des lésions fongueuses suppurées des articulations, il faut se rendre compte des avantages et des inconvénients de la résection pour chaque grande articulation.

Là où la mobilité articulaire est indispensable au bon usage du membre, au coude, par exemple, il faut réséquer les extrémités osseuses dans le but de faire reconstituer une nouvelle articulation, mobile et solide à la fois, sur le type physiologique de l'articulation primitive. On réséquera d'autant plus cette articulation que l'opération ne nuit pas sensibilement à l'accroissement du membre ; l'accroissement du membre par l'épaule (extrémité supérieure de l'humérus) et le poignet (extrémités inférieures du radius et du cubitus), étant environ six ou sept fois plus grand que l'accroissement qui s'opère par toutes les extrémités qui constituent le coude. C'est-à-dire, que si le membre a à s'allonger dans l'avenir de 30 centimètres ; quatre au cinq tout ou plus manqueront par le fait de la suppression des cartilages de conjugaison qui seront sacrifiés par l'opération.

Dans la seconde enfance l'abrasion et la cautérisation articulaire sont toujours suivies d'une perte, plus ou moins complète des mouvements ; mais il n'en est pas ainsi dans la première enfance, durant les trois premières années, et j'ai vu plusieurs fois des mouvements incomplets, mais assez étendus se rétablir après la suppuration spontanée ou la destruction artificielle des fongosités. C'est une raison de plus pour ne pas se presser de réséquer à cet âge les articulations en suppuration, à moins qu'il n'y ait des désordres osseux étendus ou des nécroses épyphysaires avec décollement. Dans ce derniers cas la résection ou l'ablation des épiphyses nécrosées est très nettement indiquée ; et il faut la faire le plus tôt possible, car en attendant on laisse non seulement les forces s'épuiser, mais les membres s'atrophier de plus en plus par suite des troubles de nutrition qui accompagnent les suppurations articulaires.

C'est même dans les cas de ce genre que la résection, en tarissant la suppuration et mettant le membre à même de fonctionner, favorise indirectement l'accroissement du membre. Des résections de la hanche pratiquées vers l'âge de trois ou quatre ans, nuiront à l'accroissement dans certains cas moins que des coxalgies suppurées entretenues par la présence de la tête fémorale nécrosée ou du moins décollée de la diaphyse. Si le sujet vit et résiste à la suppuration, il aura un membre atrophié dans son ensemble, et finalement plus court qu'un membre réséqué au dessous des trochanters.

Il ne faut donc pas être absolu au sujet des résections dans l'enfance. Si les resources de la nature sont grandes à cet âge, il est des formes d'ostéo-arthrite qui ne peuvent pas guérir spontanément et qu'il faut se hâter d'opérer dès qu'un diagnostic exact est posé. La question de l'accroissement ultérieur du membre, si importante lorsqu'il s'agit d'arthrites suppurées qui ne menacent pas immediatément la vie du sujet, devient tout-à-fait secondaire lorsque la résection est le meilleur moyen de conserver la vie.

Si, donc, nous nous élevons contre l'abus des résections dans l'enfance, nous

sommes loin de les rejeter. Si elles sont, relativement au nombre des arthrites fongueuses, chez les enfants, beaucoup moins souvent indiquées que chez les adultes, elles constituent dans certains cas le seul moyen de guérison. On en a abusé dans certains pays, mais dans d'autres contrées on a commis l'erreur inverse. En France, en particulier, la plupart des chiriurgiens ont exagéré leurs tendances conservatrices dans le traitement des lésions articulaires chez les enfants, et quoique cet excès soit peut-être moins préjudiciable que l'excès opposé, il n'en doit pas moins être condamné.

C'est par une appréciation judicieuse des affections curables par l'expectation méthodique et des formes morbides incurables par les ressources de la nature qu'on se tiendra éloigné des opinions extrêmes et qu'on arrivera à formuler les préceptes rationnels de la thérapeutique articulaire.

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## *On the Treatment of Scrofulous Inflammation of Joints.*

Professor HUETER, Greifswald. (Abstract.)

The author first referred to the experiment of Villemain showing that tuberculosis is an infectious disease, and then stated that scrofulous disease of joints was part of a general tubercular disease, as might be proved by transplanting some of the fungous matter into the anterior chamber of the eye of a rabbit. After about four or five weeks it developed, and he had been able to demonstrate the tubercular animal side by side with the patient, who under suitable treatment had partially recovered. The treatment consisted in the injection of solutions of carbolic acid into the midst of the diseased tissue: about 1 gramme of a 2 to 5 per cent. solution suffices for each injection. By means of a hypodermic syringe it is quite possible to penetrate even bone when diseased. The syringe must penetrate the cortical layer so as to get into the medulla.

If after three weeks of this treatment the case does not get well, excision of the diseased joint may be practised. But early excision is necessary. Excision is not only a curative agent, but it is also useful, because it allows the surgeon to get a full view of the extent of the disease, so that he can remove every part of it.

He referred also to the use of the gouge, of the cautery, and of iodoform.

If an examination of the lips of scrofulous children be made, almost as large a number of white corpuscles will be found as in a case of leucocythemia. As recovery takes place they become fewer and fewer. In other cases again they increase in number, and then relapses are to be feared.

### DISCUSSION.

Mr. JOHN H. MORGAN, London: English surgeons are hardly prepared to accept *in toto* the views of Professor Hueter as to the pathology of these affections. It is desirable that the early pathology of these cases should be more studied, and with it the clinical symptoms which accompany the commencement of disease in the synovial membrane or in the end of a bone. I am not prepared, in some cases, to reject the treatment by free incision or injection, but from my own observation the treatment of synovial membranes by sulphuric acid or of the bones by potassa fusa has been followed by failure.

With regard to excision, I find that the advocates of that form of operation do not give sufficient merit to the results obtainable by rest, such rest as is given in cases of children suffering from disease of the hip, under the care of Mr. Marsh and myself. The subject of excision is a large one, and spoken of generally, it had not yielded all the results which were expected of it; and excision should not be talked of collectively, but according to the results obtained in each joint. Under these circumstances the remarks of M. Ollier are valuable, especially those which point to the share of the epiphyses in determining the length of the limb. But for one joint only—the elbow—can excision be claimed a successful operation. Finally, it seems to me that we should try to make diagnosis of lesion at an earlier date; and if that were done it might be possible to treat the local disease by some means, but regarding always the desirability of not interfering with epiphysial growth, and therefore to avoid excision in young children.

Professor Sayre, New York: If we diagnosticated these cases of diseased joints in their very earliest stages and treated them properly, we should in the majority of cases have a favourable result, and not be compelled to resort to exsection. The first and most important point is *rest*—absolute and complete rest of the joint involved; and as the disease is one of malnutrition, the general health must be sustained. If the rest given to the joint is obtained by confining the patient to the bed, the general health is impaired by the confinement, and therefore the treatment should be, to apply some means that will secure rest to the particular joint involved, combined with sufficient extension and counter-extension to prevent pressure on the inflamed parts. At the same time the appliance should be so adapted as to allow the patient to take full and free exercise in the open air, thus placing the patient in the best position for increased nutrition. If suppuration follows, let out the pus; and if caries increase, notwithstanding the best treatment that can be given has been adopted, then excise, and in many instances the most favourable results may be anticipated. I have exsected the hip-joint in seventy-three cases,* and in some of them the result has been almost perfect, both as to motion and length of the limb, as can be seen by these various photographs. But, as I first stated, if we diagnosticate the disease early and treat it properly, we will have favourable results in the majority of cases. Time is too short to say what I wish; but as my views are well known, it is unnecessary.

Dr. Gustavus Krauss, Darmstadt: I think that before we can accept the teaching that "in scrofulous disease of joints, resection is indicated in those cases in which, after the injection of the antisepticum into the centre of the diseased spot, an improvement had not been effected," we ought to carefully weigh all collateral circumstances, to follow the course of the disease in our mind's eye as far as possible, as well as to consider the condition of joints as we find them after the disease has expanded itself, side by side with our means for rendering such joints useful by operation. Thus the comparative number of cases where a movable joint is obtained as against an immovable one ought to be specially considered, because a more favourable prognosis as to utility can be given in the cases where motion is retained. I believe that movable joints are more numerous than anchylosed ones, and that, as a rule, especially in the joint (knee) now under consideration, by means of tenotomy and a carefully applied orthopædic treatment very useful limbs might be obtained, and which, more especially as regards movement and shortening, compare favourably in

* See Vol. II. of these Transactions, p. 338.

these respects with resected limbs. Thus I hesitate to accept unreservedly the rules that have been laid down (by Hueter) as to the treatment of scrofulous joints.

Dr. Fochier, Lyons: I cannot allow to pass without contradiction Professor Hueter's statement relative to the inefficiency of immobilisation in chronic joint disease in children. In the country of Amedée Bonnet many white swellings are cured simply by immobilisation, and the method is not at all likely to be discontinued. On hearing Professor Hueter's statement I am almost led to believe that the disease varies in different countries. As regards resection of joints in children, after having in the past operated freely, I now seldom ever practise this operation. I have lately been able to send a certain number of my cases to the shores of the Mediterranean, and thanks to this climate I have seen some remarkable cures of suppurating joints. On the other hand, after resection of the joint or removal of fungosities, the child remaining in the hospital, its general condition was not improved, the wound again became unhealthy, and assumed much the appearance it had before any operation was undertaken. Quite recently I sent off two cases to the seaside—one a recurrent swelling of the elbow after operation, the other a suppurating fungous arthritis of the same joint: both patients returned after a while cured. Confining my remarks to what I have myself seen, I may say that the articular cartilages are often intact in children. The bone and the synovial membrane are infiltrated, and even suppurating in places, while the cartilaginous part of the joint is hardly if at all eroded. This it is which explains the possibility of re-establishing movement after a suppurating arthritis, and becomes another reason for discountenancing resections, which, *à priori*, have no advantage except that of preventing anchylosis.

Mr. Samuel Benton, London: confined his remarks to the treatment of "chronic scrofulous disease of the knee-joint," and expressed his conviction that prolonged rest in the treatment of these cases might be carried too far. It is not rest, but walking exercise and skilful rubbing of the joint, after all the adhesions have been properly broken down, which is the surest method of securing a useful limb. To break up the adhesive bands requires a sudden, sharp, and quick movement, whilst the patient is under the influence of an anæsthetic, and the patient should be encouraged to walk about directly after the operation. The adhesive bands must be broken where they are thinnest—in the centre; stretching the adhesions or tearing them from their papilla-like insertions is harmful; and after breaking down a stiffened joint to put it up on a back splint is often waste of time. Cases in which the bony structures are affected or in which there are open sinuses communicating with the joint are not suitable for this kind of treatment, but all other cases of chronic strumous disease of the knee-joint can be got well by skilful rubbing and movement after the adhesions have been properly broken down. I have successfully treated several cases in this way, and may mention one of a little girl aged seven years, who had had scrofulous disease of the knee-joint for three years. At the time of the first operation there was pain and considerable serous effusion into the joint, great thickening of synovial membrane, atrophy of muscle of thigh and calf, the limb was flexed nearly to a right angle, the tibia dislocated backwards and every symptom of destruction of cartilage; this case was cured by repeated forcible movement, walking exercise, and skilful rubbing.

Mr. Holmes, London: The time has so nearly expired, that I can only say

a very few words, and these will chiefly relate to Dr. Hueter's paper. With much that fell from M. Ollier I heartily concur, but am obliged, with regret, to leave the various topics of his address unnoticed. As to the idea that scrofulous disease of the joints is a self-infecting disease, almost locally malignant, as Professor Hueter's ideas imply, it seems opposed to the fact, so constantly observed in practice, that such affections constantly disappear when treated in the case of rich people's children by rest, good air, residence at the seaside, &c. If similar hygienic requisites could be obtained in the case of the poor, probably few operations on joints would be practised. Nor do I feel disposed at once to accept Professor Hueter's rules of practice. Repeated injection of diseased joints, still more the perforation of inflamed bones with a needle in several places, and the introduction of an injection into the medullary tissue, followed after a very short trial by excision, appears to me too rough and "heroic" a method of treatment for a disease so chronic, and which general experience shows to be so curable. Had time allowed, it would have been interesting to have heard more as to the diagnosis of the seat of disease in bones by percussion and auscultation.

After the PRESIDENT had announced that this discussion concluded the work of the Section—

Dr. JACOBI, New York, rose and said: Gentlemen, the vastness of the preparations for the International Medical Congress has been surpassed by its success only. Particularly the foreign members will leave London with a profound gratification, amounting almost to surprise, at the results accomplished, the order of the proceedings, the warm reception on the part of their English confrères, and the generosity and the hospitality displayed by them. In regard to this Section, we have a special cause for congratulation. In the person of our President, Dr. Charles West, we have had a presiding officer who, with his great reputation as a medical man and writer and his knowledge of languages, combined all the great and desirable qualities of a chairman. Patient, urbane, universally both kind and able, he has contributed considerably to the success of our meetings. I move that the thanks of this Section be voted to Dr. Charles West for his earnest, thoughtful, and successful services in our behalf; and to the other officers for their interest in our meetings, their uniform kindness, and for the success attending their industry and punctuality.

Professor SAYRE, New York, re-echoing the sentiments, seconded the vote, which was carried by acclamation.

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## *On Some Points in the Etiology and Treatment of the Tuberculous and Scrofulous Diseases of Infancy and Childhood.**

Dr. THOMAS MORE MADDEN, Dublin.

As the value of clinical experience depends mainly on the extent and accuracy of the observations on which it is founded, I may premise that I have been connected, since its opening, with the first special hospital for sick children established in Dublin.

* This paper was accepted by the Council, but from want of time was not read during the meeting.

In this institution we have now recorded on our books nearly 23,000 cases of the diseases of infancy and childhood, which have been treated in our wards and extern departments. Of these 1744 were intern patients.

In a large proportion of the cases thus brought before us, the scrofulous or tuberculous diathesis was the *fons et origo malorum*, and within the past few years the number of cases thus caused has increased from 35 per cent. in 1872, to 48 per cent. in 1881, of all the diseases treated at the hospital.

First amongst the causes of this greater frequency of tuberculous diseases may be reckoned the growing indisposition to suckle their children which is now noticeable amongst women of all classes, and the unsuitable or injurious character of some of the preserved or artificially prepared substances which are too commonly substituted for the only natural aliment of an infant.

The probability of tuberculosis being thus occasioned is a question of great practical importance. In his recent treatise on "Bovine Tuberculosis in Man," Dr. Creighton has shown that tuberculosis, in the human subject, is in many instances identical with the tubercular epizootic known as *Perlsucht*, or bovine tuberculosis. And in the acute form of tuberculous disease, so frequent in infancy and childhood, the disorder has been observed by Cohnheim, Klebs, and various other writers to "resemble closely the infectious diseases in its zymotic origin, from a specific virus, whether generated in the body from caseous matter, or introduced from without."

In many other zymotic disorders, milk is well recognised as the agent by which the septic germs of disease is carried. The propagation of typhoid fever in this way has been distinctly traced by Dr. Cameron of Dublin to the use of milk thus infected. And the evidence adduced by Dr. Creighton as to the pathological identity between the tubercular *Perlsucht*, affecting the serous membranes of animals, fed, for the purpose of experiment, on the milk of diseased cows, and human tuberculosis, shows the possibility of the disease being thus propagated in the human subject, who is equally liable to this source of infection when fed on the milk of unknown cows, as the disease is common, and does not materially lessen the quantity of milk.

Secondly, to some extent the larger proportion of tubercular or scrofulous diseases observed among our patients may be ascribed to the increasing inebriety and poverty of the families of the labouring classes; for, notwithstanding the great increment of wages, the families of even well-paid mechanics and artisans are now poorer than was formerly the case. This is owing not so much to the corresponding rise in the price of the necessaries of life within the last few years, as it is to the daily increasing habits of drunkenness in both sexes, in whose offspring the toxological effect of alcoholism is evinced by physical deterioration and increased tendency to scrofula, tuberculosis, and other diathetic diseases. This point is continually forced on the notice of the medical officers of our hospital. In treating the tuberculous children of the poor we are often obliged to supply, by cod-liver oil, Parrish's or Dusart's syrup, extract of malt, and other preparations of the same kind, the want of food of which these wretched children have been defrauded by their drunken parents, and the privation of which is a prime factor in the development of chronic tuberculosis or scrofula.

Thirdly, local circumstances have, I believe, much to do with the prevalence of scrofulous and tubercular diseases in Dublin. The situation of the city in the low-lying, badly-drained valley of the Liffey, the densely inhabited tenements, too generally devoid of the most necessary sanitary requirements, in which the poorest class of a poverty-stricken population are crowded together, have unquestionably a deteriorating influence on the physical condition of the

ill-fed scrofulous children, who from thence recruit our hospitals and prematurely fill our cemeteries.

In considering the etiology and pathology of tuberculosis, and especially of pulmonary tubercular disease, those of us whose professional career extends over the last couple of decades must be struck with the entire change in the nomenclature now applied to these disorders as compared with that used in our student days. And in noticing this, some have been tempted to inquire whether the change of doctrine thus expressed is as real as it is apparent, or is this but another illustration of the tendency to employ new sesquipedalian Teutonic verbiage in the place of plain English words, without any corresponding change of idea being conveyed thereby, which is noticeable in other special branches of modern medicine. Many of the polysyllabic pseudo-scientific terms in which some writers appear to delight are almost irresistibly suggestive of that "patched and piebald language" of which Hudibras

> —— "had supplies so vast and large,
> For he could coin or counterfeit
> New words with little or no wit;
> Words so debased and hard, no stone
> Was hard enough to touch them on:
> And when with hasty noise he spoke 'em,
> The ignorant for current took 'em."

The prevailing doctrine concerning the pathology of pulmonary tuberculosis is to some extent the revival of an old idea in altered phraseology. But this is too common nowadays to excite any attention, for we can hardly read up the literature of any department whatever of medical science without being struck with the frequent coincidences between long-forgotten opinions of our ancestors and the newest revelations of modern science.

Thus the present discussion as to the etiology and pathology of pulmonary tuberculosis as a consequence of scrofulous taint was anticipated years ago by Dr. Matthew Ballie, who described the diffused deposits in phthisical lungs as being evidently scrofulous, asserting that whether found in the lungs or any other organs in which this scrofulous matter co-existed with tubercles, both are equally susceptible of undergoing caseous degeneration.

It would be impossible to consider in a short paper so wide and so vexed a question as the relation of scrofulous affections to tuberculosis; but without entering on any lengthened or pathological disquisition, I may venture to say that I still adhere to the doctrine which was impressed upon my mind many years ago by clinical observation in the wards of the scrofula-haunted civil Hospital of Algiers, where I had an opportunity of seeing every phase of this disease. And I have no hesitation in reiterating the opinion that the scrofulous diathesis is the prolific source of all tuberculous diseases, whatever part of the body may be affected—whether it be the lungs, the meninges or substance of the brain, or spinal cord, the mesenteric glands, the cancellous structure or articulating surfaces of the bones, or the external glandular system.

The pathology of scrofulous lymphatic glands, as recently described by Professor Treves (*British Medical Journal*, April 30, 1881), and by those other pathologists who, with him, hold the distinctive character of scrofulous inflammation, I venture to think, would rather sustain the older doctrine of its identity with tubercular disease. In both we have a morbid process, commencing in the medullary or deeper portions of the affected glands or structure; in both we have the formation of large cells with glistening protoplasm, indistinguishable from the "giant cells of phthisis," as described by Virchow, within the lymphatic and in the diseased tissues; in both we have similar reticulated products

of inflammation, the coagulated lymph, later on becoming opaque and retaining the rounded form. As the disease advances these opaque or caseous spots become foci of irritation, leading to suppuration, and giving rise to purulent cavities or infiltration, extending over greater or small areas of the inflamed structure. It would be useless to trace this analogy between tubercular and scrofulous inflammation through all its stages.

In my work on change of "Climate in the Treatment of Consumption," &c., the first edition of which was published many years ago, as well as my later guide to "The Health Resorts of Europe and Africa," I have dwelt upon the connection between tuberculosis and the scrofulous diathesis, and I am well pleased to find that the views I therein expressed concerning the causation of pulmonary tuberculosis are practically identical with those now generally adopted, and which rest on the basis of advanced modern pathology. As Dr. Pollock says, "Scrofula is the key to the whole sequence of these changes. . . . A scrofulous disease of mucous membrane, or of joints, or of a lymphatic gland, has furnished the detritus of a caseation, which was afterwards carried by vessels or lymphatics into the lung, and originated the destructive changes in question."

Miliary tuberculosis, although by no means confined to the pulmonary structures, or necessarily more frequent therein than in other organs, is still, when primarily attacking the lungs, best described by the old name of acute or galloping consumption.

In the Dublin Hospital for Sick Children we have now seen a large number of cases in which pulmonary miliary tuberculosis has passed through all its successive stages within a period of from three to four weeks, from its first manifestation until its victim's death. In one of these cases less than a fortnight elapsed between the commencement and fatal termination of the disease.

This fearful acceleration of the race to death, and the generally attendant deposition of similar tubercles throughout the body, especially in the meninges and substance of the brain, the peritonæum and liver, &c., in such cases leaves little room to doubt the accuracy of Buhl's view, viz., that "miliary tuberculosis is an infectious disease, produced by an auto-inoculation with caseous matter in the body."

The accidental or catarrho-pneumonial origin of phthisis, in many instances, is most evident in the rapid development of pulmonary tubercles, as the sequence of bronchial catarrh and pneumonia in the strumous children of the poor, debilitated by semi-starvation and exposed to cold and damp. Virchow holds that caseous retrograde metamorphosis is invariably antecedent to miliary tuberculosis, and Dr. Pollock observes that tubercle is but a secondary superadded result, arising from inflammatory residua of inflammation that have undergone caseation.

Notwithstanding any pathological objections to the contrary, I believe that none whose experience is founded on actual clinical observation at the bedside in the wards of an hospital, and not confined to histological investigation, will question the fact that some cases of miliary tuberculosis are essentially chronic in their course. In such cases, it would be almost impossible to draw any clear symptomatic distinction between pulmonary miliary tuberculosis, when thus chronic and true, or catarrho-pneumonial phthisis, as developed in early childhood. In both cases we have to deal with a wasting disease, the most evident general symptoms of which are cough, hectic fever, acceleration of pulse, increased frequency of respiration, sudamina, diarrhœa, heightened morning temperature, with evening falls. The physical signs discoverable in auscultation and percussion have, moreover, almost the same sequence

in both cases. In the former, however, we have in addition the symptoms occasioned by the secondary or concomitant deposit of miliary tubercle in the organs and tissues.

The rapidity with which the prodromal catarrh of scrofulous children becomes developed into phthisis is often almost inexplicable, considering the multiplicity and complexity of the pathological changes involved in the progress of the disease, from the first invasion of an attack of bronchial catarrh to the caseous degeneration, and more or less complete alveolar disintegration of the affected lung.

*Mesenteric phthisis, or tabes mesentèrica*, is a not infrequent form of tubercular disease in our hospital practice, where some thirty-seven intern and nearly five hundred extern cases of this kind have now come before us. In these cases, the prominent symptoms were great emaciation, increased or perverted appetite, at first obstinate constipation, and in the more advanced stage diarrhœa. The skin is harsh and rough, the tongue was generally preternaturally clean and red, the child's breath offensive, and, on examination, the abdomen is then found enlarged, tense, and generally tympanitic; so that it is a matter of some difficulty to ascertain the enlargement of the diseased glands, which in two cases I have seen situated immediately below the umbilicus, and in one in the right hypogastrium. These tubercular masses varied in size from a small hazel-nut to that of an orange.

With regard to the treatment employed, it is difficult in our hospital, the disease being essentially chronic, to ensure that long-continued attendance, or that steady perseverance in the regimen and remedies ordered, that are essential in such cases. Still, however, I have had sufficient evidence of the curability of this disease under favourable circumstances, and may therefore briefly allude to the by no means novel treatment which appeared most serviceable. In all cases the patient should be placed on a nutritious diet, varied, of course, according to the child's age, but always containing a fair proportion of well-cooked fresh meat with vegetables. In the first stage of the disease, purgatives, generally in the form of a mixture containing turpentine and Peruvian balsam, were administered for a considerable time for the purpose of unloading the prima via, as well as of stimulating the intestinal glands, and removing that hard tympanitic distension of the belly which is so general in this stage of the disease. At the same time cod-liver oil and syrup of iodide of iron were exhibited. Small doses of grey powder were then generally prescribed, and great benefit was derived from the inunction of a very dilute calomel ointment over the enlarged glands.

Amongst the medicines which I have found of most use in the treatment of this disease in private practice, the pancreatic emulsion, for which the profession is indebted to Dr. Dobell, holds a high place.

*Scrofulous disease of joints.*—A considerable number of cases of scrofulous diseases of the knee and ankle joints, as well as a few cases of morbus coxæ, and scrofulous disorganisation of the elbow joint, have come under treatment at our dispensary. In the majority of these instances, the constitutional evidences of the scrofulous diathesis were well marked, but in some few cases there was no sign of scrofula other than the local strumous inflammation of the affected joint.

It would be useless to follow, seriatim, the chronic strumo-tubercular disorders which have come before us, in the practice of the Dublin Hospital for Sick Children, as these include almost every disease of this class. The following table, however, shows the comparative frequency of some of these diseases in 1744, intern cases treated in that institution:—

TABLE.

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| Phthisis, chronic and acute . | 108 | Scrofulous disease of spine . . | 51 |
| Scrofulous disease of cervical and other lymphatic glands . | 105 | Mesenteric disease . . . | 43 |
| Scrofulous disease of the joints | 95 | Tubercular meningitis . . | 16 |
| Scrofulous disease of skin . | 59 | Specific scrofulous ulcers . . | 42 |
| Phlyctenular ophthalmia . | 59 | Scrofulous osteoporosis . . | 8 |
| | | Hepatic tuberculous disease . | 8 |

The existing prevalence of the scrofulous or tuberculous diathesis is also of considerable practical interest in another and much neglected aspect. I now allude to the light that is thus thrown on the causation of the increasing frequency of the disorders which, in after-life, come under the care of gynæcologists. For, as I have shown elsewhere, the scrofulous diathesis is the most common predisposing cause of chronic endocervitis or endometritis, and consequent sterility, and various other female functional derangements. As this subject is one which has attracted much less attention than its importance demands, and indeed is still unduly ignored in practice by gynæcologists, I take this opportunity of expressing my views as to the frequent connection between a strumous childhood and the subsequent occurrence of the most common forms of chronic uterine or peri-uterine disease, the matter being one which comes fairly within the scope of the present discussion.

In the first place, therefore, I may mention that my attention was originally called to this point by observing that amongst the patients attending the Dispensary for Diseases of Women attached to the Dublin Lying-in-Hospital, a considerable proportion of those suffering from subacute uterine or peri-uterine disorders, such as chronic endometritis, cervicitis, areolar hyperplasma of the cervix, chronic ovaritis, menstrual disorders, tendency to repeated miscarriages, and sterility resulting from the same cause, were persons obviously of the scrofulous diathesis, or were actually suffering from some other evident scrofulous disease at the same time.

I am confirmed in this view by my experience during the last three years in the Gynæcological Department under my charge in the Mater Misericordiæ Hospital, where I have traced, in many instances, the clearest etiological connection between scrofulous taint and chronic uterine or ovarian disorders.

In the cases of cervicitis, leading to ulceration, which were thus caused, the character of the local complaint was clearly expressed with the unmistakable scrofulous type. The utero-vaginal secretion, in these cases, closely resembling the glairy discharge of the specific nasal catarrh of scrofulous children, and being attended by a similar tendency to the production of excoriations of the mucous membrane, gradually extending to the submucous tissues. Like other forms of scrofulous inflammation, that affecting the cervix uteri is apt to thus lead to the formation of ulcerations of the characteristic strumous aspect, irregularly circular in shape, superficial in depth, pale and flabby looking, possessing little natural sensibility, tedious beyond patience when neglected or maltreated, but readily curable when appropriate anti-strumous constitutional treatment is conjoined with the use of the suitable local applications required, and too generally exclusively relied on in such cases.

It need hardly be observed that it would be manifestly impossible to attempt even the most superficial outline of the general treatment of the scrofulous and tuberculous diathesis, within the narrow limits of this paper. This treatment may be considered in a twofold aspect—the first being dietetic and hygienic, and the second pharmaceutical; and on each of these volumes might be written.

Therefore, in the few minutes now at our disposal, I shall merely allude to one or two of the remedies from which I have seen benefit in the general treatment of chronic strumo-tuberculous disorders; and add some observations, founded on my own experience, concerning the curative influence of change of climate, and the use of certain mineral waters, in such cases.

Amongst the remedies most generally applicable in all chronic scrofulous or tuberculous disorders, none are so important as those which act dietetically, as well as medicinally, by rectifying the primary error of nutrition, which is the starting-point in all such cases. Foremost amongst these are cod-liver oil, maltine, extract of malt, and the various preparations of iron, especially the combinations of the lacto-phosphate of iron, with lime and other salts, such as Dusart's, Parrishe's, or Fellowes' compound syrups.

In the majority of the strumo-tuberculous cases under my care at the hospital, I have administered cod-liver oil, either by itself or combined with an equal quantity of syrup of iron, or what I regard as the most valuable method of administering remedies such as this, to infants as well as of those older patients whose stomach rejects the medicine—by inunction.

The carrageen moss, or *Chondrus crispus*, although now little employed beyond the immediate vicinity of its habitat on the south-west coast of Ireland, is one of the most valuable food medicines which can be used in the chronic disorders now under consideration. This species of alga, of which the sea-shores of Clare furnish an inexhaustible supply, was originally recommended in the treatment of pulmonary tuberculosis and scrofulous diseases, by Mr. Todhunter of Dublin, upwards of half a century ago; but at the present day its use as a highly nutritious article of food, as well as an antistrumous specific, appears to be almost entirely unknown, and hence I take this opportunity of again bearing my testimony to its importance as a cheap, abundant, easily prepared, agreeable, and generally most serviceable article of food for children suffering from any of the chronic tubercular diseases which are connected with or originate from the strumous diathesis.

Iodine is admittedly the only medicine that can be regarded as possessing anything like specific power in these cases, and the reason more benefit is not derived from it appears to me to be owing to the non-employment of the metallic iodine recommended by the older writers. And my own experience would lead me to recommend the long-continued administration of metallic iodine in the minute doses originally suggested by Lagol, and given in combination with the iodide of iron and iodate of quinine, until distinct iodism is produced. A combination of this kind, which I have found of singular benefit in the treatment of the scrofulo-anæmic cases, so commonly met with in the out-practice of the hospital, consists of two to four drops of tincture of iodine, half a grain of iodate of quinine, and a grain of iodide of iron, given two or three times a day, in any suitable vehicle.

With regard to local applications to the parts implicated by scrofulous disease, there can be no question of the utility in some cases of the deep hypodermic injection of a few drops of tincture of iodine into the substance of scrofulous glands, nor of the use of cataplasms of the *Fucus vesiculosus* in similar cases and in strumous ulcerations. In the latter I have seen great benefit from the application of poultices of this common sea-wrack, and as I am not aware that this simple but most efficacious application is mentioned in any of the text-books on diseases of children, I now refer to it.

These sea-wrack poultices require to be persevered in, without intermission, for a considerable time, and to be changed at short intervals. When used in this way, and being combined, of course, with appropriate constitutional treat-

ment, I have often seen the most obstinate scrofulous sores, that had resisted all other remedies, in a short time after the application of the wrack poultices, commencing to assume a healthier aspect, gradually contracting in size, and leaving hardly any trace of their existence.

I might readily adduce cases in support of this assertion, but one will suffice however.

J. H., aged about ten, the scrofulous child of scrofulous parents, was treated at the dispensary and in the infirmary from December until the end of the following April. For a considerable time previously he had been under treatment in another hospital, where the disease was at last pronounced incurable, and when brought to the dispensary, it was chiefly in order that he might obtain a certificate for the Hospital for Incurables. On first sight I was inclined to agree in the opinion that the case was a suitable one for that institution. The child was worn and hectic looking, and on being examined, a large sore was found extending on the right side from the angle of the jaw in front along the sterno-mastoid muscle, and backwards along the trapezius from its origin to the scapular insertion, and spreading over the forearm as low down as the insertion of the deltoid; whilst on the opposite side the ulceration corresponded very closely with the extent of the platysma myoides. Throughout this vast tract of disease the ulceration was foul and unhealthy looking, the edges were thickened and everted, and the surface covered with an offensive and ichorous discharge. A mixture containing cod-liver oil with syrup of iron, and minute doses of iodine, was ordered, and the ulcerated surface was covered with sea-wrack poultices; these were changed frequently during the day, and kept constantly applied for fully three months, at the end of which there remained only two small healthy granulating ulcers, one on the right forearm, and the other on the neck. The child, though still thin and pale, had quite lost the cachectic worn aspect he presented when first brought to the dispensary. He was now pronounced convalescent, and was sent to the country for change of air.

The hygienic and climatic treatment of the strumo-tubercular diseases of early life is a subject of considerable practical interest, as hygiene and climate are all-important in the cure of all chronic disorders of this class. And although, unfortunately, we can only extend the advantages of favourable hygienic and climatic conditions to that small proportion of our patients who are the children of the wealthy, still so great are the remedial advantages of such treatment, that it obviously demands our most careful consideration.

The therapeutic influence of change of climate and the use of various mineral waters in the treatment of chronic tubercular and scrofulous diseases of childhood and youth, as well as in other chronic disorders, is a subject on which I can speak with some confidence, having devoted attention to it during several years of foreign travel in pursuit of health or in attendance on invalids in the winter health-resorts of Europe and Africa, as well as at the spas of Germany, France, and Italy. And I may here repeat that the accuracy of my views on these topics has been proved by the freedom with which they have been borrowed from my works on "The Health Resorts of Europe" and "The Spas of Germany, France, &c.," by other writers.

In these works, as elsewhere, I have long maintained that in chronic tuberculosis, and in all scrofulous affections of childhood and youth, no remedies are so generally useful as certain mineral and thermal waters, when used at their sources and conjoined with change of climate. The disorders we are now considering are essentially chronic in their progress, and hence the action of our remedies must be almost as gradual; for it is vain to expect that a brief

course of any remedy can suddenly undo the effect of years of disease. In mineral waters of different kinds, suited to the exigencies of each special case, we have therapeutic agents of undoubted power, the action of which on the animal economy is generally so gradual that they require to be continued for long periods to produce their curative effect.

Three distinct classes of these waters may be used in the treatment of the different forms of chronic strumous and tubercular complaints. The first are the iodated and bromated saline springs, the Iod-und-Bromhältige Kochsalz-wässer, as the Germans term spas containing iodine and bromine, generally in the shape of bromide of manganese and iodide of sodium, dissolved in a muriated saline water. Springs of this kind are seldom thermal.

The most important of these iodated or bromated spas are Wildegg, in Switzerland, which is a most powerful remedial agent in all glandular scrofulous diseases. This water must be used with considerable caution, however, and in small doses of from 2 to 4 ounces twice daily, as in larger quantities it soon produces complete iodism.

Amongst these iodated and bromated springs, besides that just mentioned, we have sufficiently wide choice from which to select a suitable spa for the treatment of glandular enlargements and other scrofulous disorders. If the circumstances permit of the patient going to Germany, we have there, at Kreuznach, Halle, or Salzhausen, strongly iodated waters, whilst for those for whom our English watering-places would be more suitable, we have at Woodhall Spa a mineral spring hardly inferior in therapeutic value to any of its foreign rivals.

The second class of mineral waters above alluded to as applicable to the treatment of some chronic strumo-tubercular disorders are the simple chalybeates, such as Spa or Schwalbach. These waters, which contain little more than the carbonate of the protoxide of iron, with an excess of carbonic acid, are powerfully tonic and stimulant in their action, increasing the number of red corpuscles and amount of hæmoglobuline in the blood, and are thus of special service in the diseases we are considering.

Saline chalybeate springs, such as the Statelbrunnen of Homburg or Kissengen, or Tunbridge Wells, or the Kissengen Spring of Harrowgate, may also be used in certain scrofulo-tubercular complaints.

Another class of mineral waters also applicable, though in a somewhat more limited class of scrofulous or tuberculous cases, are the cold sulphurous waters, such as Lisdoonvarna in Clare, Enghein-les-Bains, the old well of Harrowgate; the warm sulphurous spas, such as Aix-les-Bains, Eaux Bonnes; and the other Pyrenean thermal sulphurous springs, Aix-la-Chapelle or Schiznach—although frequently prescribed in tuberculous affections, and most useful in appropriate cases, especially of glandular enlargements, these, however, require to be used with very great caution in such cases, being powerfully stimulating in their action on the vascular and cerebro-spinal nervous system.

The thermal arseniated mineral waters of Royat, Mont Dore, and St. Nectaire, in the volcanic district of Auvergne, may be used in the treatment of scrofulous glandular enlargements, and some cases of strumous disease of the joints, with great advantage.

In change of climate we have another, and often the most efficient, remedy for chronic tuberculosis. In no patients may we more confidently hope for the beneficial results of change of air than in the case of children predisposed, either by hereditary descent to scrofulous diathesis, or physical conformation to consumption. The constitution, as yet unformed, may be expected to receive and retain whatever impression a pure, bracing, and mild air may produce, and

thus pass in safety over that critical period which intervenes before puberty. The climate required for this predisposition to phthisis is one characterised by a dry, moderately warm, and bracing atmosphere, dryness being the essential condition necessary. The health resorts which are now most in vogue for the treatment of this class of patients are the cold, dry tonic climates of Alpine districts, such as Davos-Plaz, in the elevated plateau of the Engadine, and other similarly situated mountain sanataria. For my own part, I regard the preference given to these cold, bracing mountain climates, although very judicious in the case of children who are suffering merely from the general debility of the undeveloped strumous diathesis, as mistaken when extended to developed tuberculosis, and especially to pulmonary tuberculous disease. The diminished pressure of the atmosphere in these Alpine health resorts, and the sudden variations in its electrical condition and temperature as well as pressure, to which such places are generally subject, must influence the balance of the circulation and tend to occasion congestions and hæmorrhages.

The primary effect of a cold, dry Alpine climate and pure mountain air, such as that enjoyed at Davos, is unquestionably tonic, and braces up the strumous child whose disease has been fostered by life in the impure, variable, and generally humid atmosphere of some of our great centres of population.

When a scrofulous youth is removed from such unfavourable conditions to a dry, cold climate, such as that just referred to, the respiration becomes more energetic, more oxygen being required and more carbonic acid being exhaled, to supply which more food, especially of a fatty character, is consumed. Thus the blood is enriched by a greater proportion of hæmoglobuline, the red corpuscles, the oxygen carriers, are augmented in number, and the general nutrition of the system is improved.

But this picture is not without an adverse view; and, on the whole, the advantages of Alpine climates, even in such cases, are by no means so certain as some believe.

Whilst those who, though of the diathesis we are considering, are still moderately vigorous may thus benefit from the cold mountain climates now in fashion as health resorts, those in whom the constitutional inroads of scrofulous or tuberculous disease are already pronounced will be very differently affected. For whether the influence of cold may be therapeutic or pathological will depend mainly on the state of health and power of resistance of the individual exposed to it, the very young and old, or those debilitated from any cause, being of course the most susceptible to the depressing action of great cold.

In such cases the blood repelled from the skin is thrown on the internal structures of the body, deranging the balance of the circulation and occasioning congestions, especially of the brain and lungs. And even where these effects are not occasioned, a probable result of subjecting debilitated scrofulous children to any of these cold climates would be, by depriving them unduly of their natural caloric, to arrest the development and lower the activity of mind and body.

For these reasons, therefore, I would much prefer some of the moderately warm, equable, and dry climates as winter resorts for the generality of tuberculous children or youths. Of such climates we have an abundant choice. And of these, according to my own experience, acquired during some years of health travel, those which best combine the required advantages are Western Australia, Upper Egypt, Heyeres and the Riviera generally, and Malaga.

In selecting a winter residence for children suffering from or predisposed

to tuberculous diseases, attention should be paid to the facilities and inducements it affords for open-air exercise. Consumptive children are generally disinclined for the least fatigue, and love to hang over the fire, whence their friends fear to disturb them lest they might thus "catch fresh cold." But how mistaken is this view, in the case of the strumous-tainted inheritors of the predisposition to phthisis, is too obvious to need any comment. For such children, above all, free exercise and exposure to the pure fresh air and sunlight are essential, as by withholding these the tuberculous diathesis may be developed in any child. Hence, in choosing a health resort in the cases referred to, preference should be invariably given to one, the climate of which will permit, with safety, of the maximum exposure to the open air, and the situation of which will afford the largest inducement and opportunities for free outdoor exercise.

In these respects the new Alpine sanataria are as unfavourably circumstanced as the older health resorts of Pau and Mentone, from which they differ in all other respects, climatic and topographical.

For the same reason, none of the artificial sanataria now sometimes recommended, or any home climate, be it ever so suitable in other respects, can compensate for the lesser amount of open-air exercise which can be there taken, when compared with the amount which the invalid would enjoy throughout the winter in a dry, warm, and equable climate such as that of Malaga. And, moreover, in our British wintering resorts, not only are valetudinarian children confined to the house for all the wet and cold days that prevail, but also they are further injured by the use of coal fires and gas lights, which in English houses fill the air with carbonaceous particles that must necessarily irritate the sensitive lungs of a child predisposed to pulmonary tuberculosis.