

**Whooping-cough / by H. Richardiere ; translated by Joseph Helfman.**

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# WHOOPIING-COUGH

BY DR. H. H. HARRISON

NEW YORK

1885



# WHOOPIING-COUGH

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BY

DR. H. RICHARDIÈRE,

PARIS, FRANCE.

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TRANSLATED BY JOSEPH HELFMAN.

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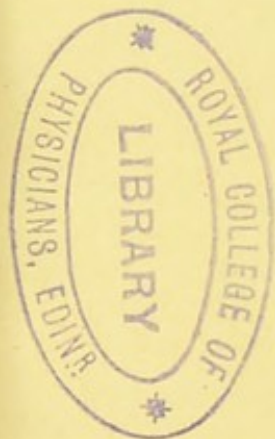
VOLUME I.

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1893.

GEORGE S. DAVIS,  
DETROIT, MICH.





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## WHOOPING-COUGH.

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### DEFINITION.

Whooping-cough is a specific, infectious, and contagious disease. Clinically, it is characterized by coughing paroxysms, more or less frequent, composed of a series of violent expirations, succeeded by sonorous and whistling inspirations. The paroxysms of cough commonly terminate by the vomiting of viscid and tenacious matters, mingled often with portions of food.

Whooping-cough has no regular, cyclical course similar to that of several infectious maladies—the eruptive fevers, for example. Its duration oscillates within limits varying ordinarily from one month to six months.

## HISTORY.

The origin of whooping-cough is completely shrouded. The precise epoch at which it made its appearance is unknown, as are also the conditions amid which the germ, now regarded as animate and susceptible of reproduction, began to develop in the human organism. It is impossible to fix with precision the date of its first manifestation in the characteristic symptoms of spasmodic paroxysms and contagious catarrh.

Like the eruptive fevers, however, whooping-cough seems to be of relatively recent origin. The principal symptoms of the malady were observed and noted in Europe toward the close of the Middle Ages. Are we hence to conclude that the germ was generated at that time? I think not. If the germ of whooping-cough was not imported into Europe during the distant expeditions and voyages marking the end of the fifteenth century, it was capable of living latent and harmless to man during long centuries, finding for the first time at this period of history the mysterious conditions of its development and acclimatization in the human organism. In truth, it is by no means rare to observe pathogenic bacteria intensify their virulence at a given moment. We have an actual example in the microbe of pneumonia, whose virulence is exalted under the influence of influenza and of certain climatic conditions, until it becomes

capable of producing veritable epidemics of infectious pneumonia, almost unknown until these later years.

The physicians of Greek and Latin antiquity knew nothing of whooping-cough. Their writings make mention of no malady whose symptoms could be referred to this disease. The Hippocratic books are silent on the subject, as are also the works of Celsus and Galen.

The first author who reported symptoms similar to those of whooping-cough seems to have been Avicenna. In mentioning a malady observed among children, he states that it caused the victim to spit blood and imparted a bluish tint to the visage. At all events, since the Arabian physician says nothing of paroxysms or of the contagious character of the malady, a great obscurity still clings to the exact cause of the symptoms he observed. His description may, indeed, apply to pulmonary tuberculosis or to the cyanotic complications of phthisis.

The term whooping-cough (*coqueluche*) was employed in France for the first time during the fifteenth century, serving to designate certain epidemic maladies which raged repeatedly and which were probably epidemics of influenza. Analyzing the symptoms observed in the epidemics narrated by Monstrelet, Rivière, Mercatus, Ambroise Paré,\* d'Aubigné, Valle-

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\* "There is an accident of the testes, termed *coqueluche*, so called because those seized with it experienced an extreme pain in the testicles and stomach, in the kidneys and in the legs, with constant fever and often with delirium and frenzy."—(Ambroise Paré, cited by Henri Roger.)

riola, we see that the patients were most often adults. The very variable symptoms consisted of fever "with delirium and frenzy." The renal pains were frequent. Nasal and bronchial catarrh were common. The paroxysms, so characteristic of whooping-cough, and the vomiting which is its consequence, are not mentioned. It is accordingly probable that the descriptions of the epidemic maladies which raged during the fifteenth and sixteenth centuries may be referred to epidemic influenza rather than to whooping-cough.

But in the epidemic described by Baillon we find the principal symptoms of whooping-cough. In this epidemic, observed in 1578, children were the principal victims. Their cough was so violent as to cause the ejection of blood from nose and mouth. Vomiting was frequent; the cough recurred in paroxysms, and the malady was often terminated by fever and a terrible dyspnœa. In this tableau drawn by Baillon, we can readily recognize the principal features of whooping-cough, simple or complicated with bronchopneumonia.

Baillon's description was as clear as possible. Yet the confusion between whooping-cough and influenza lasted another century, until Willis, in 1682, definitely established the symptoms of whooping-cough, which he studied under the name *Tussis puerorum convulsiva seu suffocativa*. The description of Willis comprises the essential symptoms of the

malady: its epidemic character, the accesses of paroxysm with threatening suffocation, the long duration of the disease, its resistance to remedies employed, etc. Thanks to this authoritative tableau of whooping-cough and its symptoms, the epidemics of the malady were thenceforward clearly individualized and referred to their true origin. Of the subsequent epidemics, which afforded opportunity to complete our knowledge of the malady and to present its most frequent complications, we may cite the Augsburg epidemic of 1724 and that of Vienna, of which De Haen was the historian.

Whooping-cough is a simple malady and easily studied, the symptoms being few. Hence, little has been added to the description of Willis. Later investigations have sought to determine particularly the nature, the causes, and the contagiousness of whooping-cough. Its treatment has been the object of numberless researches—for the most part, unfortunately, fruitless.

It would be unjust to omit from this rapid sketch the fact that whooping-cough, a malady almost peculiar to infancy alone, has been constantly studied in the Children's Hospital at Paris, and several physicians whose observations were made in this hospital have devoted very remarkable treatises to the subject. The monographs of Rilliet and Barthez, of Bache, of Trousseau, are known to all, and will ever remain authoritative. Finally, the work of the re-



gretted Henri Roger\* forms the most precious guide to any physician essaying a new description of whooping-cough. From this book we shall borrow freely, since it is the fruit of an immense practice in pædiatries and of a vast experience, subjected to the sifting of a most rigorous critical faculty.

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\* *La Coqueluche*, par Henri Roger, in Vol. II of his *Recherches Clinique sur les Maladies de l'Enfance*.

## ETIOLOGY.

The sole cause of whooping-cough is *contagion*. Long doubted, and even formally repudiated by illustrious physicians, the contagiousness of whooping-cough is now conceded by all observers. Innumerable facts attest its contagious character—for example, the epidemics of the disease, and the numerous observations which exhibit all the children of one family and the parents themselves successively attacked.

At the present time no doubt prevails respecting the contagious power of whooping-cough, in the minds of physicians or of mothers of families—whose dread of children coughing in convulsive paroxysms we all know.

Whooping-cough is never manifested spontaneously; it always proceeds from antecedent contagion. The malady may ravage epidemically a boarding-school or a village, in consequence of the arrival of one person suffering from infectious catarrh. In the large cities of Europe the disease prevails endemically, the germ being propagated and maintaining its vitality by an unbroken series of successive contagions.

Whooping-cough possessing no appreciable cause other than contagion, its ætiology is limited to the study of the adjuvant or auxiliary conditions constituting predisposition or conferring immunity. Such

auxiliary conditions are, for the most part, commonplace, as are those which favor the development of the greater number of infectious diseases.

Among the predisposing conditions the *age of the individual* stands foremost. Whooping-cough is peculiarly the disease of infancy. Its rarity in later life is, however, relative, and often is due to the fact that the adults are protected by a previous attack of the malady during infancy.

No age, indeed, is perfectly shielded: the disease has been observed at all times of life, even in old age. A case is cited by Hale White, of a woman 81 years old suffering from whooping-cough.

Infancy is attacked by whooping-cough with extreme frequency, and is exposed to the disease during all its periods.

The epoch of greatest frequency extends from the middle of the second year to the fifth or sixth year. According to R. Blache, the greatest number of cases is observed during the course of the third year. The value of figures, however, is but relative. At any moment infancy is subject to the contagion of whooping-cough. If the instances of contagion are few during the first months of life, and very numerous beginning with the second and third years, the cause of this unequal distribution is the more and more frequent contact of children with other children of the same age in running and playing, and in places of assemblage and instruction.

New-born infants possess no immunity. If born in a locality where whooping-cough prevails, they usually contract the malady. Attested observations prove that whooping-cough may attack infants during the first days of life. Bouchut saw a new-born child which was infected two days after birth, and from the tenth day suffered the paroxysms of the cough. Henri Roger, while declaring whooping-cough to be very rare during the first three months of life, yet observed several cases: one at the age of 15 days, one at a month, two others at two months, a certain number at three months. He adds that the rarity of whooping-cough during the first months of life is due solely to the fact that at this age "the infants are under more surveillance and kept more apart from the diseased."

Congenital whooping-cough, manifesting itself by the characteristic paroxysms from the very birth of a child, was once observed by Rilliet and Barthez. The violent paroxysms began on the very day of birth. The mother of this child, seized with whooping-cough a month before, was suffering from the disease at the moment of confinement.

The *seasons* and certain *meteorological conditions* were long regarded as predisposing causes. The periods of transition (spring, autumn) and the humid cold have been specially incriminated. The atmospheric conditions, held to blame particularly by authors who saw in whooping-cough only a bronchitis

or a catarrh of peculiar nature, and who slighted the infectious character of the malady, are in reality of little importance from an ætiological point of view. These conditions appear simply to favor the complications and to prolong the duration of the malady. Indeed, from the figures given by H. Roger, who tabulated 571 cases, the latter may be equally divided among the four quarters of the year. The deviations reported are so slight that they may be ignored. It may be affirmed that whooping-cough prevails with equal intensity at all seasons.\*

The telluric conditions are equally insignificant. Whooping-cough prevails everywhere—in the valleys as well as on the plateaux, on the banks of rivers and in plains arid with drought. All the countries of the earth have been visited by the malady, which becomes acclimated wherever it finds the conditions necessary to the propagation and maintenance of the contagious germ. Nowhere has it found human races refractory to its development.

The conditions of habitation are of no importance. The germ of whooping-cough develops and propagates as well in sumptuous homes—the most wholesome and best ventilated—as among the poor and wretched hovels of manufacturing cities. If whooping-cough seems more frequent amongst the

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\* The figures given by Roger are: out of 571 cases of whooping-cough, 158 occurred in the spring, 138 in the summer, 138 in the autumn, and 137 in the winter.

poor, the cause may be found in the crowded tenements and the habitual contact of neighboring children—conditions more particularly favorable to its propagation.

No temperament is a safeguard against whooping-cough. Strong and vigorous children are stricken as readily as are the feeble, lymphatic, or scrofulous. Sex has no influence. From an abstract prepared by Rosen and comprising more than 40,000 cases, it is shown that whooping-cough attacks boys and girls alike.

There is no antagonism between whooping-cough and the other maladies of infancy. Whooping-cough frequently attacks children yet suffering or convalescing from other diseases. These morbid associations are frequently seen in children's hospitals, where the little patients are treated in the wards where whooping-cough prevails constantly.

Despite all assertions to the contrary, there is no *affinity*, more or less mysterious, between whooping-cough and measles. The association of the two maladies is frequent in children's hospitals, but is due alone to the facility with which the double contagion may be imparted.

*Immunity from Whooping-Cough.*—But one single, certain cause of immunity from whooping-cough is known, and that is a previous attack of the malady. There is perfect accord on this point. A previous whooping-cough almost absolutely prevents any new

seizure. The immunity conferred by a prior whooping-cough is even more potent than that imparted by measles, scarlatina or typhoid fever against relapse from the respective disease.

Relapses in whooping-cough are altogether exceptional. Many physicians who have passed long years in children's hospitals and have practiced pædiatrics all their lives (Rilliet and Barthez, Bergeron, for example), assert that they have never observed any. More fortunately situated, H. Roger claims to have observed five cases of relapse, perfectly authentic, and recalls the fact that West and Trousseau also witnessed three cases—the former one, the latter two. A case of relapse from whooping-cough was recently reported by Le Gendre. These examples suffice to show that whooping-cough may be followed by relapse, but relapse is exceptional, and a first attack is almost certain to beget subsequent immunity.

Apart from this immunity conferred by whooping-cough itself, certain individuals possess a natural immunity, of whose nature and causes we are ignorant. Natural immunity from whooping-cough is not peculiar to adults, but may likewise exist in infancy. In a work by Budert on "Predisposition to Whooping-Cough," we find, indeed, that in a village near Hagenau, where an epidemic of whooping-cough prevailed, out of 418 children, only 366 had the disease. Accordingly, 52 *who had suffered no prior attack of whooping-cough* enjoyed an immunity, at least

temporary, from the disease. The figures given by Budert show that the immunity is proportionately far more potent in children of ten to fourteen years. The temporary nature of the immunity is further demonstrated by the fact that certain individuals living constantly in an environment where whooping-cough prevails, long exhibit an indemnity, and contract the disease after the lapse of several years when exposed to a new contagion.

*Propagation of the Contagion.*—Numerous observations made during epidemics of whooping-cough show that the contagion is not effective at a distance. The germ is not borne from afar by the ambient air. It is not distributed by drinking-water or propagated by the food. The malady is almost always transmitted by direct contact with a patient. The duration of contact, moreover, is very variable. In certain cases children have caught the whooping-cough as a result of passing only a few minutes in the presence of a sufferer. The contact need not be close; the immediate proximity of a whooping-cough patient is sufficient, as is shown by the following case: A lady, seated in a public garden, is watching her child, three years old, playing near by. Another person comes along and takes a seat a few yards away, placing beside her a little girl companion. In a few minutes this child is seized with a paroxysm of convulsive cough. Alarmed and justly dreading the whooping-cough, the first mother withdraws promptly with her



child, who had remained several yards distant from the little cougher. Despite the prudent flight, the child was seized fifteen days later with a whooping-cough, absolutely characteristic.—The following is another instance wherein contagion was very rapidly effected: A lady living in the suburbs of Paris, occupied with her two children (one four years and the other three months old) a property separated from an adjoining garden by a hedge. The day after her arrival, promenading in the garden with her two children, she heard a child coughing in paroxysms on the other side of the hedge. She hurried her children into the house, and prevented their returning to the garden during the following days. Nevertheless, the two children had the whooping-cough, which manifested itself several days later.

In these two cases a few minutes sufficed for the operation of the contagion. At times repeated contact seems necessary. There are, indeed, children who live constantly with others suffering from whooping-cough, yet are not seized save after the lapse of several weeks (H. Roger).

Observation shows that whooping-cough may be transmitted by sick children at a distance of several yards. We may hence conclude that it is the expired air, particularly the air expired after the paroxysm, and bearing with it the solid particles of expectoration, which is to be regarded as the vehicle of contagion.

Everything points to the belief that the animate germ of whooping-cough resides in the secretions accompanying the paroxysm. Thus we may comprehend how the germ, readily expelled during the convulsions of the paroxysmic cough, may bear the contagion to a certain distance. The expectorated matter of the whooping-cough subject should accordingly be considered dangerous and carefully disinfected.

The germ of whooping-cough, contained in the expectoration and borne along on the expired air, may cling to the clothing of people who have come in contact with the sufferers, and particularly to the clothing of physicians. Thus it may be transported a certain distance. In a case occurring in the practice of H. Roger, a child which had suffered for three weeks from typhoid fever (hence keeping its room), after several weeks developed a whooping-cough, probably communicated by its physician. All the more reason, then, for considering as dangerous the rooms occupied by whooping-cough patients, and for causing them to be shunned by children in health.

The contagiousness of whooping-cough varies at different periods of the malady. It is *nil* or very slight during the first period. It reaches its maximum of intensity during the second period at the moment of the characteristic paroxysms. Is the malady still contagious during its third period? According to certain authors—Hoensler, in particular—its maximum of virulence is attained at this period.

The latter opinion is not general, and it is conceded that, the paroxysms once terminated, the contagiousness of whooping-cough need not be feared in the great majority of cases. Whooping-cough may, however, prove contagious during the third period, and of this we have certain proof. Witness the following: In a certain family a child six years old was sick with whooping-cough. A brother, aged 11 or 12, attending a boarding-school in the suburbs of Paris, kept aloof from the family residence for nearly four months. At the end of that time, when the little patient had almost completely recovered and presented only an insignificant cough (for there were no more paroxysms, so to speak), the schoolboy was permitted to spend Sunday with his family. The room had been carefully disinfected. Despite these precautions, he contracted the whooping-cough, although the parents declare that he remained but a short time in the presence of the convalescent and that the latter had no coughing spell during the interview.

*The Contagion of Whooping-Cough.*—The manifest contagious character of whooping-cough has long given rise to the suspicion that its medium of contagion must be a living parasite, probably contained in the air expired by the sick and in the expectorated products. Linné and, later, Rosen of Rosenstein, were the first to admit the parasitic nature of whooping-cough, but were unable to give proofs in support of

their opinion. In 1867, Poulet, having found infusoria in the air expired by children suffering from whooping-cough, supposed he had discovered the parasite of this malady. In view of the epoch at which Poulet's researches were made, and the absence of all precise bacteriological principles, we may assume that the micro-organisms which he described as peculiar to whooping-cough formed part of the germs which are encountered in the organism in such great number, especially in the mouth. Several years after the communication of Poulet, Letzerich (whose researches dealt with the etiology of all the infectious maladies) supposed he had also isolated the contagious germ of whooping-cough from the products of expectoration. He described the parasites of whooping-cough as micrococci forming whitish flakes and visible in the expectorated products. According to Letzerich, these parasites of whooping-cough are developed in the tonsils, the throat, and the upper part of the larynx.

These researches possess little beyond an historical interest, in view of the defective methods employed by authors interested in bacteriology prior to the discoveries of Pasteur.

Contemporary bacteriological investigations have not yielded certain results. Among the authors who have devoted themselves to these researches we may cite Bürger, who claims to have found in the expectoration of whooping-cough patients a special microbe

never encountered by him amid other conditions. The presence of this microbe is alleged in every case of whooping-cough, its abundance being claimed to be in proportion to the intensity of the disease.

According to Bürger, this microbe is found particularly in the little flocculent masses observed in the expectoration. Its form is that of a straight rod (*bâtonnet*), the length being double the width. A constriction appears in the middle of the bacillus. Bürger was able to color this microbe by the method followed by R. Koch in tinting the bacillus of tuberculosis.

The characteristics of the microbe described by Bürger are of little importance. Moreover, since he neither cultivated nor inoculated, we may entertain some doubts as to its precise relationship to whooping-cough.

The researches of Afanassiew, controlled and verified by Wendt, are of greater value. If the results of Afanassiew were established by a rigid control, it is very probable that this author saw the true microbe of whooping-cough. The microbe which he describes was found in the liquid expectorated after the paroxysms, and he claims to have found it in every case of whooping-cough which came within his researches. The microbe of Afanassiew is a bacterium  $0\mu 6$  to  $2\mu 2$  long.

When cultivated on gelatin, this microbe develops in the form of little colonies, of a darkish yellow

color and rounded or oval form. These colonies do not liquefy the gelatin. On agar-agar the colonies present a whitish-gray coloration. The potato is the soil upon which they develop most readily and rapidly. They first form a thick layer of yellowish hue; presently the layer assumes a brown color, and in a short time the entire surface of the potato section is invaded by the colonies.

Afanassiew essayed the inoculation of this microbe, and obtained positive results after inoculating cultures in the trachea and in the lungs of young dogs and cats. He claims thus to have produced accesses of whooping-cough, soon accompanied by bronchitis or broncho-pneumonia.

The labors of Afanassiew have been confirmed by Wendt in a bacteriological review of whooping-cough. Wendt states, in fact, that he sought for the bacillus of Afanassiew in every case of whooping-cough observed by him, and always encountered it. He found it only during the paroxysmic period, and never in the first period preceding the appearance of the paroxysms.

In a recent communication to the Berlin Medical Society,\* Ritter describes another microbe as the specific cause of whooping-cough. He found it (in two children suffering from whooping-cough) enclosed in the white corpuscles and mixed with expec-

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\* Berlin Medical Society, session of Nov. 2, 1892. *Semaine Médicale*, Nov. 9, 1892.

torated mucus. Ritter's microbe is very tiny; its form is that of the diplococcus, the isolated cocci being rounded in outline and slightly flattened at the centre.

Ritter's microbe is aërobic, developing readily between  $36^{\circ}$  and  $38^{\circ}$  C. It cannot be cultivated below  $30^{\circ}$  C. nor above  $42^{\circ}$  C. The isolated cocci may be arranged in every possible manner (masses and chains, straight or curved). Two dogs were inoculated with Ritter's microbe: one succumbed to pneumonia; the other survived. Inoculated in the trachea of these animals, the microbe determined a cough similar to whooping-cough.

## GENERAL DESCRIPTION OF WHOOPING-COUGH.

The *début* of whooping-cough is commonly like that of simple bronchitis. For a certain time—one or two weeks—the children present the symptoms of an ordinary cold. Then supervene the characteristic spasms, augmenting for several days in number and intensity. At this moment, having reached its full development, whooping-cough is clearly characterized by the paroxysms and by the accompanying symptoms. For several weeks this condition persists; and then follows a relaxation, not sudden but progressive. The paroxysms diminish in number, the intervals between them become greater and greater, and finally they disappear after a variable term. When they have vanished, cure may be regarded as definitive.

Such is the regular, classic form of whooping-cough, and this course is observed in the great majority of cases. Hence it forms the general basis for a description of the malady, and we shall conform to this division, which has the merit of affording useful indications in prognosis and treatment.

The first period is the *term of début*, and it is still denominated the *catarrhal period*. During this term the catarrhal symptoms alone prevail, and the clinical tableau is that of common bronchitis.

The second period is that of *maturity* or *establishment*—the *spasmodic period*. It begins with the parox-



ysms of convulsive cough, and lasts until these have notably diminished in frequency. This is the dangerous period of whooping-cough, and formidable complications may then ensue to impart gravity to the malady.

The third period is that of *decline*, beginning when the paroxysms have diminished in number and intensity, and lasting until their complete disappearance.

*Incubation.*—Like all infectious maladies, whooping-cough is preceded by a period of incubation,—extending from the moment of contracting the malady until the appearance of the first symptoms marking the *début* of the catarrhal period. It is difficult to state its duration with precision, for the initial symptoms of whooping-cough are most often ill-characterized and difficult to refer to their true origin. In the hospital it is almost impossible to verify the period of incubation. In city practice, when the parents begin to suspect a possible previous contamination and watch their children closely, precise information is at times obtainable. In two cases I have been able to fix the duration of incubation at eight or nine days. The first case was that of a child who had been seated in a public conveyance near another child suffering from whooping-cough. The mother, recognizing the nature of the paroxysmic cough of the second child, made haste to leave the conveyance. Nevertheless, *her* child was seized with bronchitis eight days after, and

finally had the usual paroxysms of whooping-cough. In the other case, the whooping-cough began with cold and fever eight days after the contamination.

Several observations made by H. Roger are in accord with the two foregoing cases, with respect to the duration of incubation. They show likewise that when whooping-cough is contracted from the same origin by two children of the same family, there may be a difference of twenty-four to forty-eight hours in the appearance of the phenomena of *début* (Obs. XXVIII, in the work of Roger).

The figures limiting the expiration of the incubation period to eight or nine days are greater than those fixed by some writers. Guersant fixes the period of incubation at five to six days; Oppolzer at seven days; Gerhardt at two to seven days. These varying figures may be readily explained, for in such matters it is difficult to arrive at absolute precision. A week, however, may be regarded as approximately the duration of the incubation period.

There is no ground for associating the duration of incubation with the gravity of the malady: a mild whooping-cough may follow a very brief incubation; conversely, a serious attack may supervene after prolonged incubation.

During this period the child presents no abnormal symptom: health is perfect; naught presages the invasion of the disease.

## PERIOD OF DÉBUT ; CATARRHAL PERIOD.

After a period of incubation comprising approximately a week, whooping-cough begins. The *début* may occur in various ways.

The most common beginning, observed in the great majority of cases, manifests itself by symptoms of bronchitis. The child commences to cough—in a manner, however, not at all characteristic; he seems to have caught cold, and most often it cannot be attributed to a chill. Physical examination of the chest discloses a number of sibilants and sonorous rhonchi related to bronchitis. The general phenomena are in no wise peculiar. The child loses appetite, is restive and nervous as in any indisposition. The bodily temperature varies; at times it is quite high; from the *début* the thermometer may register  $39^{\circ}$  C ( $102.2^{\circ}$  F.) and more, especially during the evening, and for several days. At other times the temperature is somewhat above the normal, but without exceeding  $38^{\circ}$  and several tenths. In some cases fever may be altogether absent at this period.

In one case where a prior contamination gave ground for apprehending the development of whooping-cough, the *début* was in the form of bronchitis without any elevation of temperature. The temperature, taken several times a day by the anxious parents who dreaded the whooping-cough, remained normal

for several days; it did not show any elevation until the fifth or sixth day.

When whooping-cough thus begins, the symptoms observed at this period suggest an ordinary cold. Only at the end of several days does the cough undergo modifications which reveal the truth.

In another mode of *début*—one of much greater rarity and far less significance—the first symptoms are those of coryza and nasal obstruction. The child has frequent accessions of sneezing, suffusion of the eyes and swelling of the lids. The nasal mucous membrane secretes abundantly. We fancy that a simple nasal catarrh is beginning, but at the end of several days the catarrh progresses and the child begins to cough. The general symptoms are quite as insignificant as in the method of invasion first studied.

A very rare mode of *début* was that reported by Rilliet and Barthez, viz., an attack of stridulous laryngitis. This mode is of as little significance as the others; for all or nearly all the maladies of infancy, accompanied with laryngeal congestion, may begin by an attack of false croup. The one condition necessary to the production of this syndrome seems to be the existence of a certain degree of œdema—at times, of the glottis; at other times and perhaps most often, a very acute œdema of the uvula and palatine arches. When stridulous laryngitis marks the *début* of whooping-cough, it is rapidly followed by

the development of the ordinary symptoms of laryngeal and bronchial catarrh.

According to some authors the catarrhal period may be altogether absent. The beginning of whooping-cough is then characterized by the regular paroxysms. Rilliet and Barthez declare that they have been able to assure themselves that whooping-cough has begun in this manner. In an epidemic amongst children less than a year old, Trousseau observed the absence of the catarrhal period in two out of fifteen cases. West also noted the paroxysmic cough from the very *début*. Absence of the catarrhal period—the beginning being marked by paroxysms—was never observed by H. Roger. Conversely, the catarrhal period may be prolonged abnormally; in some cases it persists several weeks ere the appearance of the paroxysms.

Whatever may be the mode of its commencement, for several days the only manifestations of whooping-cough are symptoms of catarrh—most often bronchial, at times laryngeal or nasal. The child seems to suffer from a mild attack, the general symptoms being often those which are observed in an ordinary cold. The temperature is normal, or exceeds the normal by only a few tenths of a degree. This condition persists for several days without any ground (save where there has been a prior contagion) for suspecting the development of whooping-cough.

At the end of this period several signs serve to

indicate the road to diagnosis. First of all, the initial catarrh, instead of ameliorating, despite confinement to the room and appropriate treatment, augments in intensity. The cough becomes more and more frequent, though auscultation fails to note an aggravation of the symptoms perceived at the commencement of the malady.

And while the cold seems to progress, the general condition becomes sensibly modified. The agitation and sullenness of the child increase daily. Sleep is less calm and is frequently broken by the cough. Appetite disappears. The fever, at first scarcely noted, becomes quite pronounced in the evening. The pulse is more rapid.

The cough is transformed and often becomes very frequent, manifesting itself by repeated expiratory convulsions following in close succession. Several convulsions of cough (at times eight, ten, and more) succeed one another without interval. The modifications of the cough are often more appreciable during the night, causing anxiety, even at this early period, and impeding respiration momentarily. We have not yet reached the paroxysm with sibilance, several times repeated; but an attentive observation will obtain the first indications—the outline, as it were. The character of this cough, recalling that provoked by the presence of a foreign body in the air-passages, attracts attention and suggests the possible invasion of whooping-cough. At times the

cough is incessant and gives the patient no repose. H. Roger dwells much on this feature of the cough, and sees in it a good indication of transition from the first to the second period.

The timbre and rhythm of the cough are not alone modified. It becomes looser, and is accompanied with the expulsion of mucus more or less adherent. Not rarely this mucus is already viscid and clings to the lips and nostrils; already the inspiratory whistling may be perceived in certain accesses of cough.

These changes in the cough announce the end of the first period, and are daily accentuated. The paroxysm then supervenes with its ordinary symptoms; now begins the second period, and whooping-cough with its entire array of symptoms is established.

The duration of the first period, from its inception to the appearance of the first paroxysm, is quite brief. It varies generally between one and two weeks, being slightly affected by the character of the epidemics and individual idiosyncrasies. According to Rilliet and Barthez, the duration of this period oscillates between four and ten days. In the experience of some authors it has been abnormally prolonged. Lombard (of Geneva) observed it to persist a month to six weeks during the epidemic of 1833.

It has been stated that the younger the patient, the briefer this period. In infants at the breast, the paroxysms may ensue two or three days after the first symptoms which mark the *début* of the malady.

West maintains that the duration of this period has an important bearing on prognosis, claiming that the gravity of the disease increases with the length of the first period. But West's opinion is formally contradicted by our experience with the whooping-cough of new-born infants. Among the latter the term of the first period is most often very short, though the whooping-cough prove particularly grave.



## SECOND PERIOD (SPASMODIC PERIOD).

The second period of whooping-cough is the most important in the malady, and begins with the paroxysms which are its essential characteristic and impart to it the name of Spasmodic Period. Up to the appearance of the paroxysms, diagnosis is impossible and whooping-cough may be confounded with a great number of affections of the respiratory passages. But when the paroxysms exist, doubt is no longer possible to an experienced observer, and they assure the diagnosis of whooping-cough.

The paroxysms are not only the most important element of diagnosis, but the prognosis of whooping-cough also depends in great measure upon their number and intensity. Hence it is most important to know the paroxysm thoroughly and the features by which it may be distinguished in diagnosis.

In observing a paroxysm of whooping-cough in a child (in such matters, a single attentive observation is worth more than any description), the following is noted:

Either without apparent cause and in the midst of evident tranquillity, or as a result of one of the provocations which will be mentioned later on, the child is suddenly seized with a series of convulsive expirations, generally loud and violent. Five, ten, fifteen, twenty times in a paroxysm, the "whoops" succeed one another, imparting to the thorax and

often to the entire body a violent shock. During this expiratory phase of the paroxysm the face is red and congested. The tongue is impelled violently against the roof of the wide-opened mouth. Inspiration is arrested throughout these convulsive expirations, hence hæmatisation is momentarily suspended; and if the "whoops" are oft-repeated, the visage becomes blue and the face congested, with an apparent menace of asphyxia. Then, suddenly, a great inspiration occurs, which for an instant interrupts the expiratory convulsions. Inasmuch as a violent spasm of the glottis prevails at this moment, the air, penetrating into the thorax through the constricted lips of the glottis, produces a very peculiar noise. The sudden inspiration makes a loud report. The accompanying noise has been compared to a whistling, and again, more justly, to the clucking of a hen. This noise or "whoop" is the source of the Anglo-Saxon name of the disease. The noise may be well enough imitated by inspiring a great quantity of air and simultaneously contracting the soft palate.

The loud inspiration once effected, the paroxysm is not terminated, but recurs in the form of renewed expiratory spasms, soon interrupted by a second noisy inspiration. The paroxysm thus pursues its course through several accessions. A paroxysm of whooping-cough may comprise but one access, but that is exceptional: almost always several may be counted (four, five, and even more).

If the recurring accessions are prolonged beyond measure, the situation may grow ominous, for asphyxia, averted for an instant by the sudden entry of air into the thorax, becomes during the convulsive expirations more formidable at each access. The children, in their despair, rush into the mother's arms, hoping there to find a relief for their sufferings. The older patients instinctively lean the head against a wall or some piece of furniture; they extend the arms and cling to the objects within reach, as much to avoid the violent shock of the paroxysm as to give a point of support or fulcrum to the levers formed by the accessory muscles of expiration. But these efforts do not suffice to combat the progress of the asphyxia, the signs of which are readily recognized in any paroxysm which is somewhat prolonged: the face becomes cyanosed, the eyelids tumefied, the conjunctiva injected, the eyes suffused, and the veins of the neck swollen. During this time the pulse is extremely rapid, the heart accelerating its beats in order to propel into the lungs the blood which fails to find there the elements necessary to its oxygenation.

When the paroxysm is prolonged it produces immediate danger, which is unfortunately realized in a certain number of cases. In the immense majority of cases, however, it ceases at the most dramatic moment. Its end coincides with a very important phenomenon—an expectoration of peculiar nature. The purpose of the paroxysm, indeed, seems to be the

expulsion of certain secreted matters. These dense and adhering substances are borne along slowly by the expiratory convulsions. At the termination of the paroxysm they are seen clinging to the soft palate and to the posterior wall of the pharynx; by a final effort they are ejected by mouth and nostrils in a final access of cough. The expulsion of these viscid substances may be effected all at once or at several times, and is always difficult, for the tenacious matter is pushed, as it were, by successive efforts, rather than borne away mechanically. Reaching the lips, it often adheres there until detached by the fingers of the child or the aid of the parents. When the expectorated matters are accompanied with an abundant salivary hypersecretion (which happens often enough), they are more easily detached. They are then commonly vomited and expelled in a terminal access of cough.

The expectoration which puts an end to the paroxysm is a phenomenon of great and specific importance. The rarity of expectoration during infancy imparts to it such importance that every time a child's cough is accompanied with an abundance of expectorated matter, whooping-cough must be suspected.

The expectoration of whooping-cough has certain peculiarities which must be made familiar. It is formed of whitish and viscid *glairs* or mucus resembling somewhat the white of egg, when the whoop-

ing-cough is not accompanied with bronchitis. If there be concomitant bronchitis, the expectoration is formed of mingled *glairs* and greenish mucus. At times these mucous substances form little masses or "chunks" and assume the aspect of the mummular expectoration observed in the third period of pulmonary tuberculosis (H. Roger).

Examined under the microscope, the glairs of whooping-cough present a number of rare white globules and cells of pulmonary epithelium. In this matter, expectorated at the end of the paroxysm, Afanassiew found microbes which he considered as pathogenic in relation to whooping-cough. These microbes are not present in the form of pure cultures, but are mingled with the microbes which dwell normally in the mouth and pharynx.

It happens often that the expectorated products are mixed with portions or débris of food, the ordinary effect of the whooping-cough paroxysm being to provoke vomiting.

The origin of the vomiting is mechanical. Caused by the shock of the cough, it is comparable to the vomiting of tuberculous subjects who exhibit a violent cough after their repasts. The vomiting following the paroxysm is sufficient to warrant our considering it as one of the essential factors on the same plane with the expulsion of the viscous glairs.

Recapitulating, then, the paroxysm is composed of three principal elements:

1. Convulsive expirations more or less repeated (expiratory phase).

2. One or several loud inspirations interrupting the convulsive expirations (inspiratory phase).

3. An expulsion of glairy and mucous matters, often accompanied with the vomiting of food (excretory phase).

Such is the paroxysm of whooping-cough with every foreign element eliminated. The description would be incomplete did we not mention the mental anxiety of the child, unable to control respiration, struggling to establish the equilibrium of his vital forces, and yet menaced with imminent asphyxia. Frequently, moreover, the paroxysm is accompanied with a certain number of mechanical accidents, such as hæmorrhage of the nose, mouth, and auditory meatus; hernias, produced by the violent cough; prolapse of the mucous membrane of the rectum, similarly determined, etc., etc.

The duration of the paroxysm of whooping-cough is subordinate to the number of accessions. Generally it is quite brief, and varies from a quarter-minute to one or two minutes. In exceptional cases the duration may be longer. Rilliet and Barthez have observed the paroxysm to last a quarter-hour; in such cases there are always several accesses, and they probably involve subintractant paroxysms.

The paroxysm terminated by the expulsion of mucus and glairs, the scene changes completely. The

child, a prey just before to the liveliest agitation, returns quickly to his normal state. His fears vanish. His tears are dried. A smile reappears on his lips. If aroused from sleep by a paroxysm, he tranquilly resumes his slumber. If awake, he returns to his play or occupation so suddenly interrupted. Often the child who has vomited feels the need of nourishment and asks for food. The crisis is succeeded by a state of well-being and complete calm, which lasts until the appearance of the following paroxysm. During the intervals of the paroxysms, health appears normal if there be no complication.

The number of paroxysms varies with the gravity of whooping-cough; in the average cases, twenty may be counted during the twenty-four hours. In mild cases the number is less, and in grave attacks as many as sixty, eighty, and more may be counted during the twenty-four hours.

At times the intervals between paroxysms are tolerably regular; again, they predominate at this or that period of the twenty-four hours. According to the major number of authors, the paroxysms are most frequent during the night. H. Roger, who regards movement as a provocative of the paroxysms, concludes that they are more frequent during the day.

The paroxysms often supervene without appreciable provocation—during the night, for example, when the child is in profound slumber and when no exterior cause can explain the appearance of the crisis. In

such instances the paroxysm is preceded by a short period of agitation, during which the child turns several times in bed. Respiration is accelerated, and suddenly the patient is awakened by an access of cough. His first movement is to sit up, and he remains in that position until the termination of the paroxysm permits him to resume his broken slumber. Supervention of the paroxysm during the day may be provoked by accessory circumstances, which must be well studied in order to avoid their repetition and to guard against an excessive frequency of the paroxysms. These accessory conditions are almost all excitations of the nervous system, which in infancy is so impressionable. Thus, the paroxysms supervene readily in consequence of some denial or unwelcome command, or of any emotion whatsoever, sad or joyful.

Sometimes a strong impression upon the organs of sense, such as a violent noise or a pronounced odor (such as that of tobacco-smoke, for example), suffices to produce a paroxysm. Exertions made by the child, walking against the wind, running, playing, may all provoke the crisis. Henke deems deglutition a quite frequent cause—which would explain the ordinary occurrence of the paroxysm during meals. Repletion of the stomach may play the same rôle.

Merely hearing a paroxysm of whooping-cough has at times the immediate effect of provoking a coughing spell in other sufferers. Paroxysms thus provoked are quite frequent in children's hospitals,



where the paroxysm of one patient is not uncommonly followed by a number of accesses in other little sufferers. Physicians officiating in children's hospitals are acquainted with these simultaneous paroxysms which embarrass their visits and disturb their observations by the deafening noise.

Supervening without appreciable cause, or provoked by an exterior circumstance, the paroxysm is generally preceded by abnormal sensations, perceptible to children of a certain age. Usually it is announced by a titillation of the larynx and trachea. Sometimes it is preceded by a sensation of weight and thoracic constriction, or by sudden dyspnœa accompanied with great acceleration of the respiratory movements. Having before experienced them, the child knows these sensations well. Taking warning and expecting the imminent appearance of a paroxysm, he quits his play or occupation, becomes quiet and "waits for his spell," as they say. If you question him at this moment, he scarcely answers the queries addressed to him. Observing closely, accelerated respiratory movements are perceived. At times we may detect at a distance sonorous retro-sternal râles, corresponding to the movement of the mucosities whose expulsion is the purpose of the access. This period of preparation for the paroxysm is scarcely a few seconds in length, and ceases with a burst of the characteristic cough. These precursory phenomena are not observed save by children old

enough to tell what they feel. In early infancy the paroxysms seem to supervene suddenly and, usually, without premonitory symptoms.

*Abnormal Forms of Whooping-Cough.*—The paroxysm of whooping-cough, as I have described it, characterized by three essential phenomena—*paroxysmic* or *convulsive cough*, *loud inspiration*, and *expectoration*—is the classic paroxysm commonly observed. It is at times attended with a few additional symptoms, also of a spasmodic order. A quite frequent phenomenon is the sneezing which begins with the cough and persists for a part of its duration. This sneezing generally coincides with an abundant hypersecretion of the nasal mucous membrane, and is followed by the discharge through the nostrils of nasal mucosity, which mingles with the mucus proceeding from the throat and bronchi. Not seldom an augmented lachrymal secretion is observed in connection with the hypersecretion of pituitary mucus.

In some exceptional cases the accessions of sneezing may constitute the entire paroxysm of whooping-cough. Two conditions may be then presented. In the one the accessions of sneezing manifest themselves at the beginning of the second period and for a certain time constitute the sole appreciable spasmodic symptom. Later on, the malady pursuing its evolution, the paroxysmic cough appears and then imparts the proper significance to the accessions of sneezing. This rare occurrence was twice observed by Trousseau.

The paroxysm may at times be completely absent, and may be replaced during the entire duration of the malady by accessions of sneezing. Similarly, the paroxysm may be replaced either by crises of hic-cough with hissing or whistling inspiration, or by "spasms\* in appearance exclusively pharyngeal." H. Roger, who has reported the existence of crises of pharyngeal spasm, states that he has seen them amid such conditions and environment that he was forced to regard them as masked forms of whooping-cough paroxysm.

*Effects of the Paroxysm.*—Having studied the paroxysm in itself, we must indicate its immediate consequences on the condition of the child. Its principal effect is the interruption of normal respiration. During the series of expiratory spasms which succeed one another, inspiration is suspended; respiration and hæmatisation are suppressed. An immediate consequence is the menacing asphyxia resulting from this arrest of respiration. The signs of acute asphyxia are quickly manifested. The child suffers from lack of air. He exerts his accessory muscles of inspiration to obtain a greater dilatation of the thorax and facilitate the entry of a slight quantity of fresh air. For this purpose he instinctively seizes anything which may serve as a point of support. He stretches out his arms and clenches his hands about some piece

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\* *Saccades*, literally *jerks*.

of furniture or the bars of his bed. He seeks a support for the head, to avoid the painful shock communicated by the spasms of cough. Despite these instinctive efforts, the glottis, spasmodically closed, excludes the air: the paucity of respirable air becomes greater and greater, and signs of asphyxia appear. The face becomes turgescient and cyanosed, the lips blue, the veins of the neck distended with blood which is there arrested or is repelled from the thorax. The right heart disposes imperfectly of the venous blood which it contains, and its repletion produces a reflux of the blood into the jugular veins. To surmount these obstacles to venous circulation, the heart beats tumultuously and the pulse is accelerated notably. As the expiratory spasms become more and more frequent, it seems as though the asphyxia were irremediable and the infant about to succumb to the terrible crisis. But happily an amelioration almost always ensues at the moment of greatest peril. Inspiration is suddenly reëstablished, producing its special tone. Air rushes to the lungs, and the blood is supplied with oxygen. This momentary relaxation permits the child to continue his struggle with the asphyxia. At each accession the asphyxia becomes threatening, more threatening than before, for the inspirations have not been renewed sufficiently to afford proper oxygenation; hence, the child would finally succumb in the struggle did not the paroxysm terminate and permit a return of the normal respiring function.

The impeded access of air to the thorax has the effect of embarrassing the circulation, as is shown by the distended veins of the neck and by the capillary stasis in the plexuses of the facial skin and mucous membranes. The impeding of circulation produces puffiness of the visage, œdema and swelling of the eyelids. The puffiness is sometimes momentary and disappears with the paroxysms; it persists when the paroxysms follow one another closely, and then becomes a revealing symptom of whooping-cough. The circulatory impediment has the further consequence of facilitating the vascular ruptures which are the ordinary accidents of whooping-cough. They occur in the vessels of the nasal mucous membrane, and in those of the conjunctiva or the membrane of the tympanum, each time determining hæmorrhage more or less profuse.

The immediate effects of the paroxysms comprise further certain mechanical accidents which should be anticipated as far as possible, for they may give rise to persistent infirmities. The accompanying effort is often sufficiently violent to produce hernia of the intestine through the hernial orifices (inguinal, crural, umbilical hernia), etc.

Prolapse of the rectal mucous membrane through the anal sphincter may also be observed during violent paroxysms.

An accident less grave but more frequent is the involuntary expulsion of urine and fæcal matter.

When the child is not carefully watched, this accident, insignificant in itself, may produce erythema of the nates; this is especially to be feared in children suffering from diarrhœa.

*Stethoscopic Examination During the Paroxysm.*—Examination of the thorax is difficult during the paroxysm; moreover, it affords little useful information. Percussion is absolutely normal. If auscultation be performed before the paroxysms, we may quite often perceive a number of sonorous or humid rhonchi. These rhonchi are at times loud enough to be heard at a distance, as we have stated in studying the precursory symptoms of the paroxysm. They are frequently absent.

While the paroxysm persists, auscultation is negative. The air does not enter the lungs at all or else the quantity is inappreciable. Auscultation discovers, besides an almost complete absence of vesicular murmur, the reverberation of the cough or the transmitted inspiratory noise produced at the moment of access. The paroxysm terminated, we again hear the vesicular murmur, most often accompanied, as before the crisis, with humid râles due to the presence of the mucous matter. At times the râles are so faint that they may be supposed to occur in the small bronchi. These râles rapidly disappear.

*Pathological Physiology of Whooping-Cough.*—Several theories have been advanced to explain physiologically the symptoms of whooping-cough paroxysm.

The interest now attaching to most of them is purely historical.

The theory which gives the most satisfactory physiological explanation of the paroxysmic symptoms is that based on the results of Rosenthal's celebrated experiment. M. Jaccoud has very happily applied this experiment to the pathogeny of the paroxysm.

The results of Rosenthal's experiment are well known. After experimental section of the pneumogastric below the origin of the superior laryngeal nerve, centripetal excitation of the internal branch of the superior laryngeal nerve determines, by reflex action on the bulb and the pneumogastric itself, relaxation of the diaphragm, the almost complete occlusion of the glottis, and the expiratory convulsion.

One cannot fail to be struck by the analogies which this physiological experiment presents with the paroxysm of whooping-cough, in which a high degree of glottic spasm and the expiratory spasms are produced.

Indeed, there seems to be good ground for holding that the cause of the paroxysm is an irritation of the superior laryngeal nerve or of the tracheo-bronchial filaments of the pneumogastric nerve, the excitation of which, according to M. Jaccoud, would have the same effect.

Of what nature is this irritation of the superior laryngeal nerve, and why is it intermittent?

It must be admitted that the irritant cause is of

peculiar nature, for of all the catarrhs and of all the laryngeal affections whooping-cough alone gives rise to the special irritation which causes the paroxysm.

The special irritant substance seems to be incontestably the mucous matter expectorated after the paroxysm. This mucous product, according to the bacteriologists, contains the micro-organism which imparts a specific character to whooping-cough, wherefore it is probable that it is this micro-organism which irritates the superior laryngeal nerve at the level of its terminations in the mucous membrane. The irritation is the work of the microbe itself or of the products which it secretes.

The intermittence of the paroxysm is caused by the intermittence in the secretion of the mucous products. The latter are secreted slowly; they first accumulate in the small bronchial ramifications; then, borne along by the movements of respiration, they reach the level of the mucous membrane of the larynx, where their presence causes irritation in the terminations of the superior laryngeal nerve.

At this moment, as in Rosenthal's experiment, the spasm of the glottis and the expiratory shocks are produced by reflex action. These phenomena depend upon the bulb, and require absolute functional integrity in this nervous centre. Such integrity exists at the beginning of the paroxysm; it ceases when the asphyxia produced by the paroxysm has caused its effects to be felt. Under the influence of the



asphyxia, the blood reaching the bulb is not fitted to furnish the elements necessary to the normal performance of its function. The action of the bulbar centres is then exhausted, and the glottic and expiratory spasms undergo relaxation; inspiration is therefore again possible. The sudden inspiration suspends the threatening asphyxia and permits the bulb to receive a supply of blood again endowed with sufficient reparative power. The spasms then reappear, and the paroxysm again begins, and ceases only with the definitive termination of the spasmogenic irritation of the superior laryngeal nerve after the expulsion of the secreted mucous product.

The respiratory filaments of the pneumogastric are not the only branches of the nerve on which are reflected the irritation of the superior laryngeal ramus. The gastric and cardiac filaments of the nerve participate in the reflex process, as is shown by the vomiting spells and the acceleration of pulse—symptoms almost uniformly observed in the paroxysm.

*Other Symptoms of the Second Period.*—The paroxysm forms the chief and essential symptom of whooping-cough; most of the other symptoms observed during the second period of the malady are its direct consequence. When the paroxysms are few, as in mild cases of whooping-cough, wherein we may count only fifteen, ten, and at times even a lesser number, during the twenty-four hours, the general

condition may be good during the intervals. The child enjoys a period of calm long enough to permit recovery from the crisis. No symptom persists between two paroxysms separated by a sufficiently long interval. In such cases, when observing an infant between two paroxysms, the physician hesitates to pronounce him ill. In order to diagnose whooping-cough, we must then rely on the account of the parents who describe with more or less precision the symptomatology of the paroxysm.

But such a condition occurs rarely. In the majority of cases the more numerous paroxysms are accompanied with secondary symptoms of a certain importance.

An inspection of the child often reveals a peculiar condition of the face. The puffiness of the tissues, due to the impeded venous circulation during the paroxysms, may, when the latter are sufficiently repeated and intense, persist in the intervals. A swelling of the eyelids, more or less pronounced and simulating the palpebral œdema of Brightic origin, is observed. This symptom is all the more striking, since the rest of the body is emaciated by the disease and by the vomitings which disturb nutrition. The puffiness of the visage is sometimes accompanied with a violet coloration of the integuments, comparable to that of cyanosis.

In a considerable number of cases the children present an *ulceration of the frenum of the tongue*, the

importance of which has been exaggerated, but which should always be sought, for it may be a revealing symptom of whooping-cough.

Ulceration of the frenum of the tongue, noted by Amelung, Braun, Bruck, Zitterland, Lersch, Gamberini, has been particularly studied by Bouchut, and by his pupils Charle and Houradieu. Its characteristics and pathogeny have been definitively fixed by H. Roger.

This lesion consists in a slight ulceration measuring several millimeters in diameter, of oval form, its great axis being horizontal. The ulceration is seated on the mucous membrane of the mouth, at a level with the frenum of the tongue, of which it forms an almost complete cross-section. Its most ordinary seat is the middle part of the frenum. Sometimes, as a result of a peculiar arrangement of the teeth, it occupies the lateral parts of the lower surface of the tongue, or is fixed upon the tip of this organ. It is generally a unit when it occupies the frenum. When the lesion extends to the sides of the tongue, several little ulcerations are often observed.

At its beginning this lesion forms a transverse section of the mucous membrane. Later the surface of the section assumes a granular aspect, then being transformed into a pultaceous and adherent coating of grayish color. The ulceration generally remains superficial and involves only the upper part of the mucous skin. In some cases it may completely de-

stroy the skin and become deeper-seated. In a case presented to the Academy of Medicine by Bouchut, the mucous membrane had been completely destroyed, exposing at the base of the ulceration the lingual muscle and the nervous branches of the hypoglossis.

The sublingual ulcerations of whooping-cough are not present in more than half the cases. Their presence is of real importance, for the researches of Bouchut and H. Roger have shown that the ulcerations, as we have described them, are to be found only in whooping-cough. They are absent in all the other maladies of infancy attended with spasmodic cough. The existence of the sublingual ulceration permits, according to Bouchut, a certain diagnosis of whooping-cough.

The pathogeny of the sublingual ulceration is now well established. The ulceration is unquestionably due to traumatism. The tongue, violently thrust out of the mouth during the paroxysms of whooping-cough, rubs its base against the sharp points of the lower incisors. It is thus lacerated, hence the primitive section at the seat of the traumatism, viz., at a level with the frenum. The laceration thus produced is maintained by the repetition of the traumatism, which prevents cicatrization. The production of the pultaceous exudate is a common feature of any ulcerative process of the buccal mucous membrane. It is observed in aphthous, ulcerated vesicles, in dental ulcerations due to the presence of roots or fragments,

and in the ulcerations produced by biting the tongue in epilepsy. This pultaceous exudate is probably the work of the buccal microbes. It is not peculiar to whooping-cough.

To demonstrate that the sublingual ulcerations are truly the consequence of traumatism, their absence in mild cases of whooping-cough is cited—there the tongue is not violently thrust out of the mouth; moreover, the ulcerations are almost always lacking in children not yet provided with teeth.

When the ulcerations are not seated at the level of the frenum, they are topographically related to the state of dentition. H. Roger cites the case of a child whose two median incisors had been prematurely lost, and who had two little lateral ulcerations determined by the external incisors.

Several ulcerations have been incontestably observed in whooping-cough patients without teeth. In such instances, however, the theory which connects them with traumatism is not at fault. Indeed, Dr. Bouffier, who reported seven cases of sublingual ulceration before the appearance of the teeth, assured himself that it had been caused by the parents when introducing their fingers into the mouth to aid in the expulsion of the mucus. In this operation the nails had produced by direct action ulceration of the frenum of the tongue.

In *résumé*, sublingual ulceration is always of traumatic origin, and cannot be considered as a spe-

cific lesion of whooping-cough. The manner in which the lesion is produced refutes the opinion of Dr. Delthil, of Nogent, who would regard it as a specific lesion of whooping-cough, related to a vesicular eruption on the buccal mucous membrane. The pathögeny of the lesion, the epoch of its appearance, its demonstrated absence in certain cases, are in conflict with this opinion.

This symptom is, however, of real importance, and is valuable in the diagnosis of whooping-cough, for practitioners as experienced as Bouchut and H. Roger have never observed it save in whooping-cough.

Terminating the list of symptoms yielded by examination of the infant, it remains to be added that extended conjunctival ecchymoses at times prevail; these ecchymoses, though not peculiar to whooping-cough, should suggest it, for they are extremely rare in infancy save as related to traumatism.

*Temperature.*—The temperature is very variable in whooping-cough of simple and uncomplicated character. The curve of temperature does not present a regularity comparable to that observed in the majority of infectious diseases. Yet the study of the temperature should not be neglected, for it furnishes valuable indications from a diagnostic point of view.

Generally speaking, the temperature pursues the following course:

In mild cases it remains normal during the entire duration of the malady, not even ascending during the first period.

In cases of moderate intensity we note at the *début* and during the period of augmenting paroxysms an elevation of the evening temperature, which, however, is never great—perhaps a little above  $38^{\circ}$  C. ( $100.4^{\circ}$  F.). Usually the morning temperature is normal, and the evening elevation is not always constant; it is not of daily occurrence. The temperature may be perfectly normal for several successive evenings. When the number of paroxysms ceases to augment, and the malady has become stationary, the temperature again becomes normal, and so remains until the end if no complication supervenes.

In serious cases the evening temperature is always elevated. At times this elevation is very marked and reaches  $39^{\circ}$  C. ( $102.2^{\circ}$  F.) and more (H. Roger). In such cases of whooping-cough, the temperature remains elevated throughout the course of the malady, descending only towards the close.

The cause of the elevation of temperature is the infection of a virus, for such elevation is observed in the absence of all complications. Inflammatory complications cause a rise of the thermometer. Though there exists no thermometric rule in connection with whooping-cough, the temperature furnishes useful indications with respect to the gravity of the malady. Normal in mild cases, it rises in direct ratio to the intensity of the malady.

The age of children suffering from whooping-cough has a real influence on the temperature. The researches of Roger have, indeed, demonstrated that in the first year of life whooping-cough is uniformly febrile. It is true that at this age the disease is commonly intense and more serious than at any other epoch of life.

Most of the complications elevate the temperature. The simplest of all, bronchitis, constantly gives rise to fever. "Whooping-cough is all the more febrile as it is the more catarrhal" (H. Roger). The pulmonary complications, and those of broncho-pneumonia in particular, produce a sudden elevation of temperature.

A temperature of  $40^{\circ}$  C. ( $104^{\circ}$  F.), ensuing suddenly and persisting without marked oscillations, should inspire fear of one of these complications. It is an almost certain index if attended with notable dyspnœa and an acceleration of the pulse.

*Pulse.*—The pulse is rarely normal in whooping-cough, being almost invariably accelerated. Instead of the normal number, 100, 110, and 120 pulsations may be counted, and at times the number reaches 130 or 140 per minute. These are the figures observed during the intervals between paroxysms; during the latter, the number of pulsations is still greater, augmenting in a uniform manner. The pulsations become excessively frequent if broncho-pneumonia supervenes as a complication.



*Dyspnœa.*—The dyspnœa, save during the paroxysms, is *nil* in light or moderate cases of whooping-cough; in grave attacks it is constant. Then becoming formidable during the paroxysms, it persists during the intervals. It is not at all pronounced when the child reposes, but becomes marked when any exertion is made or even a simple movement.

*Nutrition.*—In mild cases nutrition is not appreciably disturbed, but a different condition prevails in the serious cases, when the paroxysms are frequent and followed by the profuse vomiting of food. Nutrition then becomes cruelly impeded. The food is scarcely ingested ere the paroxysms of cough cause its expulsion, and at times attempted feeding is sufficient to provoke a paroxysm. The work of the reparative forces becomes almost impossible in consequence of the incessant vomiting. Nutrition languishes; the child grows pallid and, sometimes, excessively emaciated, assuming a cachectic aspect. The enfeebled organism then becomes a soil all prepared for the germination of the infectious maladies by which the sufferer from whooping-cough is threatened, with special reference to pulmonary tuberculosis.

*Urinology.*—The urinology of whooping-cough has scarcely been outlined. Analysis of the urine voided by whooping-cough patients, made regularly during a prolonged term, are as yet few in number. But we know that albuminuria is almost constantly absent save in case of complications. Despite the assertions

of Gibb, glycosuria is likewise absent (H. Roger and Henry Barth).

*Vomiting.*—The vomiting observed in whooping-cough is two-fold. It consists at times of glairous matter, expelled by the ordinary efforts of vomiting. This glairous matter is similar to that ejected by simple expectoration at the termination of the paroxysms; when it is very abundant it may provoke the mechanical effects of vomiting. At times—and most often—the glairous matters are mingled with the foods which are expelled in the exertion of coughing, by a mechanical process analogous to that which provokes the vomiting of tuberculous subjects who suffer paroxysms of cough after eating. The alimentary vomitings of whooping-cough are never due to a form of dyspepsia related to some alteration in the chemical functions of the stomach. Whooping-cough does not determine gastritis. Aside from the vomiting, there is no symptom to indicate any alteration whatsoever of the stomach: appetite is conserved; digestion proceeds readily and painlessly; there is no meteorism after the repasts. The vomiting is exclusively of mechanical origin, provoked by the shock of the diaphragm during the convulsive cough. When the paroxysm is terminated and the vomiting has emptied the stomach, the patient suffers no gastric pain and no nausea. There is no reluctance to eat again, and often the patient calls for food. Aliments then partaken of may be digested and absorbed if no new paroxysm supervenes for some time.

The vomiting of food becomes specially formidable if whooping-cough is characterized by closely following and prolonged paroxysms. An abundant repast is very apt to cause vomiting; excessive repletion of the stomach favors it, by provoking the paroxysms. Hence the patients should be cautiously and sparingly fed with nutritious substances of slight volume. As far as possible the meals should follow immediately after the paroxysms; in this way, if the paroxysms do not succeed one another too closely, digestion will be nearly completed ere the appearance of the next crisis. In mild cases of whooping-cough there may be no vomiting of the food.

According to some authors, the vomiting of whooping-cough is not always of mechanical origin; the cause may be a functional or organic alteration of the pneumogastric nerve. Guéneau de Mussy holds that the vomiting, like the paroxysmic cough, is due to compression of the pneumogastric by the hypertrophied tracheo-bronchial ganglia.

*Auscultation and Percussion.*—We have already studied the stethoscopic phenomena during the paroxysm. It remains for us to set forth the results furnished by auscultation and percussion in the interval of the paroxysms.

During the first period of the disease, auscultation reveals more or less numerous râles of a bronchial character (hence the name “bronchial period”). During the period of maturity the signs of bronchitis

diminish in number, and at times disappear completely; in the intervals of the paroxysms, respiration is normal and percussion negative.

Immediately before the paroxysm, and during the access itself, râles more or less numerous are perceived. The paroxysm at an end, if the whooping-cough is normal, there persists in most cases no stethoscopic sign serviceable in diagnosis.

In a certain number of cases the symptoms of bronchitis persist, more or less pronounced. But it must be borne in mind that the signs of bronchitis are in no respect specially characteristic of whooping-cough: they are bronchial symptoms and nothing more, and with these signs alone diagnosis cannot be made. Only the diagnosis of bronchitis would thus be possible.

Despite the slight significance of the stethoscopic symptoms, a daily and attentive examination of the thorax is necessary. Such examination is, indeed, of the greatest importance in the diagnosis of complications. The most serious and frequent complications of the disease are the thoracic, which are usually insidious in their development. Auscultation of the patient will disclose their *début*, indicate their nature, and at times, through appropriate therapy, prevent, if not their development, at least their aggravation.

Percussion of the thorax is normal in uncomplicated whooping-cough.

Guéneau de Mussy holds that percussion of the

interscapular region will yield valuable indications. This author insists, in fact, that whooping-cough is accompanied with constant hypertrophy of the mediastinal ganglia, manifested on pleximetric examination. The hypertrophy of the mediastinal ganglia may be discovered by percussion of the region right and left of the vertebral column between the rhachis and the inner edge of the scapula. The dull zones existing in this region are alleged to be related to the ganglionic tumefaction and to render possible an exact appreciation of its dimensions. Unfortunately such a pleximetric examination is extremely difficult. It is no easy task to seize the shades of very delicate timbre revealed by percussion of the interscapular region. Too many errors are daily committed in the percussion of voluminous and superficial organs to permit our attaching a great value to the perceptible variations of tone in a region where the organs to be explored are covered with numerous muscular layers.

*Laryngoscopical Study of Whooping-Cough.*—Laryngoscopical examination, always difficult with children, becomes peculiarly so in whooping-cough, where the application of the laryngeal mirror often immediately provokes a paroxysm. Hence the use of the laryngoscope is not to be recommended with young patients. With adults, laryngoscopical examination, according to Herff, yields useful indications respecting the state of the laryngeal mucous membrane. It may likewise facilitate a study of the provocative causes of the paroxysm.

Herff noted on himself, throughout the duration of an attack of whooping-cough, a superficial inflammation of the mucous membrane lining the larynx and the trachea. This inflammation was specially marked in the inter-arytenoid region, and was most appreciable in the posterior part of the glottic cleft and in the lower surface of the epiglottis. The region immediately below the glottis exhibited a very pronounced red color. This appearance of the laryngeal mucous membrane was permanent, and persisted during the intervals of the paroxysms.

At the moment when the latter supervened, Herff observed the presence of flocculent, viscous mucus on the posterior wall of the larynx at the level of the glottis. The presence of this mucus seems to be an important factor in the production of the paroxysm, which may be terminated if we succeed in removing the mucus.

The contact of an instrument (a sound, for example) with certain regions of the larynx, may provoke an accession similar to that constituting the paroxysm. The instrument then acts as does the flocculent mucus, whose presence at the moment of the paroxysm has been stated. The regions of the larynx, contact with which may artificially provoke a paroxysm, are quite limited; they are: (1) the arytenoid region, and especially the subjacent part; (2) the lower surface of the epiglottis. This second region gives rise to less violent accesses than the first. Touching the other parts of the larynx never provokes a paroxysm.

### THIRD PERIOD (PERIOD OF DECLINE).

The duration of the spasmodic period of whooping-cough has no fixed limits, and varies considerably according to the gravity of the malady. Speaking approximately and including solely cases of mean intensity (the most frequent in medical practice), the second period reaches its full development (period of maturity) at the end of ten to fifteen days; the period of maturity, during which the paroxysms remain stationary in number and intensity, lasts from four to five weeks; and then commences the third period.

The decline of whooping-cough is insensible. Rarely does any critical, formal symptom announce the *début* of convalescence. The urine, in particular, is not modified with the *début* of the period of decline. We observe no polyuria, azoturia, or variations in the quantity of phosphates or chlorides, which mark the beginning of convalescence from a certain number of infectious maladies. In some cases, however, convalescence may be announced by repeated eruptions of boils, particularly abundant on the face and scalp, sometimes also by an eruption of ecthymatous pustules. H. Roger, who reported these eruptions at the beginning of convalescence, does not regard them as critical symptoms; he holds that the furuncles and pustules of ecthyma are related to the state of the skin, which is often moistened with abundant perspiration during the movements and efforts of the paroxysm.

Since the appearance of these eruptions coincides precisely with the diminution of the paroxysms, at a moment when the transpirations are less frequent, it seems more rational to associate the latter with the eruptions of furuncles and ecthymatous pustules which are observed during convalescence from other infectious maladies—typhoid fever, for example.

At the beginning of the period of decline, the temperature presents no notable modifications. The general condition is not sensibly changed. The diminution of the paroxysms alone announces the commencement of this period. The number of paroxysms is at first less by several units than that of preceding days. The diminution becomes more pronounced from day to day, soon becoming sensible to the child's attendants without regular numeration. And while diminishing in number, the paroxysms likewise relax their intensity. Their duration is briefer, and the accompanying phenomena are less violent. The dyspnœa, less intense during the crisis, is not accompanied with the menace of immediate asphyxia. The efforts are less violent. The expectoration which terminates the paroxysms, occurs more readily. The mucosities, less adhesive, are expelled with greater facility. The vomitings, attending the period of maturity, become less abundant and soon disappear, at first intermittently and soon definitely.

Simultaneously the paroxysms gradually part with their most significant characteristics. The in-



spiratory sibilance is absent in certain paroxysms, which are then limited to expiratory shocks more or less numerous. In some cases the expiratory spasms cease to recur in frequency, and the paroxysms are aborted almost at their *début*. The child has one or two expiratory spasms, and then after a loud inspiration the paroxysm ceases. These aborted paroxysms recall those of the close of the first period. They announce the decline of the whooping-cough, just as the paroxysms of the first period announced its development.

With the transformation in the character and appearance of the paroxysms, during their interval a cough is observed, similar to that of simple bronchitis at its decline. In conjunction with the paroxysms, which are at times complete and at times aborted, the child exhibits a regular catarrhal cough similar to that attending the *début* of whooping-cough, but looser and less trying.

When the cough presents these modifications for several days, we may affirm that whooping-cough has entered definitely into the period of decline.

The general condition then becomes sensibly improved. If the temperature was elevated more or less notably during the period of maturity, it again becomes quite normal. The pulse, before accelerated constantly, returns to the normal number of pulsations. Dyspnœa disappears. On stethoscopic examination of the chest, the existence of bronchitic

râles is perceived. These humid and persistent râles, bearing relation to the cough with catarrhal timbre, are often more abundant than in the period of maturity.

Nutrition proceeds regularly, and the appetite may be excessive if the whooping-cough has caused frequent and abundant vomiting. Hence the repasts must be carefully watched, for indigestion or gastric embarrassment may impede convalescence and produce a relapse.

The accidents which form an almost common train to the paroxysms of the period of maturity, cease to manifest themselves. Puffiness of the visage diminishes insensibly until it vanishes. The sublingual ulceration becomes cicatrized. The amelioration of all the accidents of whooping-cough continues in a course parallel with the progressive diminution of the paroxysms.

The disappearance of the paroxysms takes place by irregular stages. In the earlier period of convalescence they soon decline from twenty and twenty-four a day to ten and even less; but later they give way more slowly—often persisting when the disease seems at an end, thus retarding recovery more or less.

The period of decline rarely exhibits a regular evolution. Sometimes, after several days of improvement, the paroxysms are observed to return brusquely with the frequency and intensity marking the period of maturity, persisting with these character-

istics for several days and finally resuming their progressive decline. These relapses of whooping-cough at times supervene without appreciable cause. In some instances they are occasioned by irregularity of the regimen or by defective hygiene. An over-abundant alimentation, a premature or too prolonged promenade in the cold air, may alike prove the source of mischief. The relapse may likewise be due to some malady (an attack of measles, gastric trouble, usually acute bronchitis), accidentally contracted.

The authors who have mentioned these offensive recurrences are unanimous in declaring them as generally of short duration. They last several days, and then the whooping-cough resumes its progressive decline. When provoked by an accidental malady, they generally have the same duration as this malady during its period of maturity.

The aptitude of whooping-cough patients to cough in paroxysms, on the occasion of any malady which may attack the air-passages, is not limited to convalescence from whooping-cough. It may persist for several months, and even several years, according to the claim of Rilliet and Barthez. Certain individuals, who had previously suffered from whooping-cough, may again be seized, as a concomitant of bronchitis, with a paroxysmic cough absolutely similar to that of whooping-cough. In such cases the paroxysmic cough is of brief duration and is noted especially at

the *début* of the malady. According to H. Roger, this return of the paroxysms is particularly observed during the months following the whooping-cough. Later on it is said to be extremely rare.

Affections of the respiratory passages are peculiarly apt to cause a return of the paroxysms in old whooping-cough subjects. In some cases, however, especially if the prior whooping-cough were intense, recurrence of the paroxysms may be observed independently of any affection of the respiratory passages. They may be provoked by an effort, or by a lively emotion such as a feeling of resistance (Rilliet and Barthez).

The returning convulsive paroxysms have certain characteristics which distinguish them from the true paroxysms of whooping-cough. They present the expiratory spasm and the inspiratory sibilance, but they are not followed by a special expectoration. They possess the spasmodic (nervous) nature of the paroxysm, without the catarrhal traits. At times they are even confined to the whistling inspiration.

These recurring and convulsive paroxysms should not be taken for relapses of whooping-cough. I have before stated that such relapses are most rare—that there is perhaps no other malady so seldom followed by relapse.

## DURATION OF WHOOPING-COUGH.

It is difficult to fix the duration of whooping-cough. The various authors who have essayed to do so (Rilliet and Barthez, West, H. Roger) are unanimous in stating that uncomplicated whooping-cough has a duration wavering between one and a-half and two and a-half months. Cases of the disease lasting only a month are rare; those persisting for several months are not exceptional. Frequently, for several weeks not more than one or two paroxysms afflict the suffering child during the twenty-four hours. But these last paroxysms do not yield, and while they persist the malady cannot be considered as cured. From a prophylactic point of view, it would be of great interest to know whether whooping-cough amid these conditions is still contagious. Unfortunately this question is yet much disputed. In doubtful cases the physician must act as though the whooping-cough were still necessarily contagious, and order the rigorous isolation of the children still exhibiting paroxysms, however infrequent. The duration of whooping-cough may be prolonged by a relapse supervening during the period of decline when the malady seems definitely ended. These relapses are generally of short duration, and do not bear in their train the ordinary complications of whooping-cough.

## ACCIDENTS AND COMPLICATIONS OF WHOOPING-COUGH.

The malady described in the preceding pages is whooping-cough, pure and simple, disengaged from all elements foreign to the essential symptoms of the disease. In the clinic it is seldom observed in such simple form. Most often the few symptoms of the malady are reinforced by a certain number of accidents. Among these accidents several are sufficiently frequent to enter into the general description of whooping-cough. These are the accidents to which we have already referred, and which are closely related to the paroxysm and the efforts it determines, constituting the *petty tribulations of whooping-cough*. And few cases, even the mild, fail to present a number.

By the side of these accidents, which are almost all of mechanical origin, there are others of a more serious nature, depending upon secondary inflammations conjoined to the principal element of the infectious malady. They constitute the true complications, imparting to whooping-cough its gravity and (in the majority of mortal cases) its fatal termination. These inflammatory complications are peculiarly liable to assail the respiratory apparatus, but they do not altogether spare the other organs, of which almost all are exposed to attack in different degrees; and the complications manifest by their presence the general

and infectious character of the malady in which they are observed.

The methodical classification of the complications of whooping-cough has often been attempted. They have several times been divided into mild and grave complications. Certain authors have proposed to study separately the accidents and the complications, the accidents being referred to the spasmodic (nervous) element, and the complications to the inflammatory element. Such classifications, properly intended to facilitate study, work mischief in prejudging the nature of these phenomena and in imposing upon them a pathogeny determined in a manner frequently arbitrary, when in reality several elements are often united to explain the supervention of the complications. The prevailing theories or classifications were proposed at a time when the notion of secondary infections was unknown and when ignorance prevailed respecting the rôle played by these superadded infections in the pathology of the infectious maladies. Now, the whooping-cough subject does not escape the secondary infections. A great number of complications, formerly considered as intimately bound up with primitive infection, appear now as independent of this infection and as manifestly due to superadded infections. A knowledge of the secondary infections in whooping-cough compels a rejection of the ancient classifications.

In the present state of science it seems preferable

to set forth the complications of whooping-cough according to their special seat and local environment. We shall dwell especially on the complications currently observed, which the practitioner must apprehend and combat, beginning with a study of the accidents conditioned by the paroxysm.

*Vomiting.*—The vomiting of whooping-cough is of two kinds. The one, occurring immediately after, or even during, the paroxysm, is of such frequency that it enters into the common symptomatology of whooping-cough. This vomiting is purely mechanical and is caused by the compression of the stomach in the spasmodic movements of the diaphragm. It is more painful than serious, and does not disturb nutrition save when too frequently repeated. These vomitings are not related to an organic lesion, nor even to a functional disturbance of the stomach. A proof of this is the fact that, the paroxysm once terminated, food then taken may be digested without difficulty if no new paroxysm supervenes during the process of digestion.

Another, rarer, form of vomiting is of real gravity. This supervenes apart from the paroxysms, and seems conditioned by gastric troubles or by an alteration of the pneumogastric nerve. Such vomiting commonly supervenes without appreciable cause. Sometimes it is occasioned by the slightest attempt at alimentation, and ensues as soon as the food reaches the stomach. In other cases the ingestion of the food is well borne,



and the vomiting supervenes only during the digestive period, being preceded simply by a brief nausea. The vomiting thus determined may become incoercible, and any attempt at feeding inevitably results in its recurrence. The situation then becomes very grave. The infant loses ground and emaciates with extreme rapidity. Cachexia is not slow to supervene. If the vomiting be not arrested, the patient may die of inanition. Even in the absence of this rare termination, the vomiting, by the anæmia that it produces, facilitates the supervention of nervous complications and particularly of convulsions.

The vomitings may ensue apart from, and independently of, any other digestive trouble. At times they are accompanied with profuse diarrhœa; denutrition is then extremely rapid, and death may speedily follow.

Ordinarily, at the onset, the child vomits only after the paroxysms. Later on, the vomiting occurs in the intervals, though it does not on that account cease to attend the crises of convulsive cough.

*Hæmorrhages.*—Hæmorrhages are frequent in whooping-cough. Two elements enter into their pathogeny: infection and traumatism. Whooping-cough is a general malady, in which all the tissues and all the systems of the organism suffer in varying degrees the influence of the infectious principle. The vessels (arterial, venous, and capillary) do not escape this influence. Though no special study has

been made of the state of the vascular system in whooping-cough, it may be admitted that this system has its part in the alterations of the organism. As in other infectious maladies, the walls of the vessels probably present alterations which result in a diminution of the resistance of the vascular tunics. In addition to these mysterious anatomical alterations of the walls, the paroxysmic traumatism intervenes. One of the common effects of the paroxysm, even when moderate, is to impede venous circulation and consequently congest the capillary plexuses, particularly those of the cephalic extremity. Vascular congestion, seated in vessels whose walls are altered, may cause dilatation and rupture, if the sanguineous repletion be reinforced by the efforts and spasmodic movements of the paroxysm.

Hæmorrhage may thus occur frequently in whooping-cough as a consequence of the coughing paroxysm. Certain hæmorrhages are almost normal. Epistaxis, for example, is observed in a great number of whooping-cough patients. If moderate and limited to the loss of several drops of blood during the paroxysm, this hæmorrhage is of no importance in itself and in no wise aggravates the prognosis of the malady. In some cases epistaxis is profuse and frequent. Supervening at first only on the occasion of a paroxysm, it later appears during the intervals, and may become a cause of anæmia. Moreover, as in all hæmorrhages, the repetition of the losses of blood

renders the patient anæmic through the depletion of the red blood-corpuscles, and facilitates renewed hæmorrhages, which become more and more difficult to arrest. This repeated epistaxis then constitutes a veritable complication.

When the epistaxis is profuse, blood may be swallowed, may remain some time in the stomach, and may be subsequently ejected with the vomited substances. In such cases we may be led to suspect hæmatemesis, and to attribute to a gastrorrhagia what is only the consequence of an epistaxis. Hæmatemesis is altogether exceptional in whooping-cough, if, indeed, it has ever been observed. H. Roger knows of no authentic case, and holds that the suspected signs of it proceed always from swallowed blood, a consequence of prior epistaxis.

Hæmorrhage of the mouth is observed as a consequence of sublingual ulceration, or of a laceration of the mucous membrane resulting from the violent contact of this membrane with the dental arches. The blood may also proceed from a fungoid condition of the gums, or alterations of the gingival mucus in consequence of alveolo-dental periostitis. Hæmorrhage of the mouth is rarely profuse. Almost invariably the blood flows drop by drop, and is expelled mingled with paroxysmic mucus, which is then tinted red.

Inspection of the face occasionally reveals ecchymoses of the oculo-palpebral conjunctiva. Sometimes

there is a slight ecchymosis seated on one of the sides of the cornea, limited in extent. In other cases ecchymosis is more extended and occupies the greater part of the sub-conjunctival cellular tissue. When hæmorrhage is caused by laceration of one of the little vessels of the conjunctiva, the blood becomes readily infiltrated into the loose cellular tissue of the region and invades the entire surface of the ocular conjunctiva. The uninvaded cornea appears surrounded with an ecchymotic circle of dark red. The eye is tumefied and protruding. In extreme cases, sub conjunctival ecchymosis is not limited to the ocular conjunctiva; it gains access to the cellular tissue of the eyelids, which it excessively infiltrates and tumefies.

These conjunctival hæmorrhages are of no prognostic importance in themselves. In imparting a very peculiar appearance to the visage of the child, they may serve as an additional aid to the diagnosis of whooping cough, when they are not explained by some prior traumatism.

Purpura has been twice observed (H. Roger).

The existence of hæmoptysis has been denied by H. Roger, who claims that the so-called hæmoptyses of whooping-cough are only cases of epistaxis, wherein the cough expels the blood derived from the nasal or pharyngeal mucous membrane. Trousseau, however, claims to have observed it several times, and does not consider this form of hæmorrhage as involving a grave prognosis.

Hæmorrhage of the auditory meatus, or otorrhagia, has been reported by Gibb and Triquet. It is generally slight, being limited to the loss of several drops of blood during the paroxysm. In a case cited by H. Roger, however, the blood gushed from the auditory meatus during the very intense paroxysms. This hæmorrhage may be unilateral or bilateral.

The pathogeny of hæmorrhage of the ear has been well outlined by Triquet. It is due to laceration of the tympanic membrane, caused by the sudden forcing of air into the tympanum during the expiratory spasms of the paroxysm.

Otorrhagia may occur in children who have had otitis media and are not yet cured. In such cases the flow of blood is not due to rupture of the vessels of the tympanum; it is caused by laceration of the vessels of the mucous membrane of the tympanum, whose softened tissues, often ulcerated by prior inflammation, are abnormally fragile.

The vessels of sound tissue are not, indeed, the only source of hæmorrhage in whooping-cough. Non-cicatrized wounds and ulcerated surfaces may bleed during the paroxysms. Abrasions of the mucous membrane of the mouth, ulceration of the lips, may give rise to a flow of blood—sometimes through congestion of the “proud flesh” of cicatrization, at other times through laceration of recent cicatrices.

Profuse hæmorrhage through laceration of the

commissure of the lips has been observed by Joseph Franck.

Ulcerated tumors may also bleed during the paroxysms. Trousseau observed a flow of blood at the surface of an erectile tumor during the paroxysms of whooping-cough.

The hæmorrhages of the disease are not exclusively external. The flow of blood may be internal, suffusing the parenchymæ and the serous membranes.

Internal hæmorrhages thus produced are much more rare than the external, and only a few cases have been cited. Meningeal hæmorrhage has been reported by Boivin, who found, in the autopsy of a child dying of whooping-cough, an effusion of blood beneath the arachnoid.

Renal hæmorrhage was observed by H. Roger in an infant who succumbed to broncho-pneumonia during the course of a whooping-cough. The same author has observed hæmaturia from hæmorrhage of the mucous membrane of the renal pelvis or from renal apoplexy.

In two cases cited by West and R. Croker, in which two children were stricken with right hemiplegia with aphasia, these symptoms could be explained by a hæmorrhage in the mantle of the cortical layer of the brain or in the cerebral ganglia.

In place of occurring from a single centre, cerebral hæmorrhage may supervene from a number of disseminated foci. F. Widal exhibited to the Ana-

tomical Society the brain of a young child who died on the twentieth day of whooping-cough complicated with broncho-pneumonia. The white brain-matter presented numerous small hæmorrhagic foci, varying in volume from the size of a pin-head to that of a kernel of grain. Each focus corresponded to a capillary which occupied its centre. The cerebellum and the marrow displayed similar foci in lesser number. The child had suffered from epileptiform crises during the last days of life.

# WHOOPIING-COUGH

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PARIS, FRANCE.

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TRANSLATED BY JOSEPH HELFMAN.

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GEORGE S. DAVIS,  
DETROIT, MICH.



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# WHOOPIING-COUGH.

## THORACIC COMPLICATIONS.

The thoracic are the most frequent complications of whooping-cough, and are of peculiar importance by reason of their gravity.

Before proceeding to study the consequences of an inflammatory condition of the thoracic organs, it is proper to say that sometimes, in the absence of any appreciable complication, an intense whooping-cough causes death by the very difficulty of respiration and by disturbances of hæmotosis. The fatal termination is then due to progressive asphyxia, no lesion of the thoracic organs being produced.

Death from asphyxia is observed in very intense whooping-cough when the paroxysms are incessant and will not permit adequate respiration. In ordinary attacks of the disease, asphyxia threatens during the paroxysms when normal respiration is suspended; but the paroxysms once ended, respiration is restored; hæmotosis, momentarily interrupted, may proceed, for its arrest is intermittent and brief. It is quite different when the paroxysms are abnormally prolonged and repeated; hæmotosis can no longer proceed as under normal conditions; the dyspnœa is continued; and the child, becoming gradually enfeebled, succumbs with all the symptoms of progressive asphyxia,

though there be no complication due to an organic alteration of the respiratory apparatus.

This mode of termination in grave whooping-cough, indicated by Guéneau de Mussy, is rare. Most commonly the asphyxia is caused by a morbid modification constituting a veritable complication.

*Bronchitis.*—Bronchitis is one of the ordinary elements of whooping-cough. Catarrhal inflammation of the bronchi always exists during the first period, and generally attends the malady to its conclusion. Frequently the symptoms of bronchitis are alone appreciable.

During the second period, complete absence of bronchitis is rarely observed, the latter commonly manifesting itself to auscultation by snoring and sibilant râles. Bronchitis, accordingly, accompanies whooping-cough during a great part of its evolution, and cannot, indeed, be considered as a complication save when of abnormal intensity; it then exaggerates the bronchial secretion and thus augments the number of paroxysms.

Intense bronchitis is recognized by auscultation of the chest. Continual snoring and sibilant râles are heard in great number, if the large bronchi are alone invaded; subcrepitant râles, if the inflammation has extended to the lesser bronchi. Intense bronchitis often produces elevation of the temperature and more or less marked dyspnœa, in accordance with its gravity and extent.

Bronchitis, commonly without serious prognosis, must be watched, for at any time it may be followed by broncho-pneumonia.

In most instances the bronchitis of whooping-cough is limited to the large and medium bronchi. In some rare cases, and particularly in certain epidemics, it gains access to the lesser bronchi and thus gives rise to a suffocative catarrh. The bronchitis then assumes an extreme gravity and rapidly carries off the patients, who succumb after presenting the classic symptoms of capillary bronchitis.

Bronchitis, as a complication of whooping-cough, sometimes supervenes without appreciable cause, being apparently due to the infection. Most frequently it is caused by an accidental circumstance such as a premature venture out of doors, or a chill.

*Broncho-pneumonia.*—This is the most formidable complication of whooping-cough. Its frequency has been the subject of different estimates by writers, varying widely according to the epidemics and the scene of its action, and depending principally on the gravity of the disease. Rare and even exceptional in whooping-cough of slight intensity, broncho-pneumonia is frequent where the paroxysms are violent and repeated. The surroundings are of prime importance. Broncho-pneumonia is rare in urban practice; it is frequent in the hospitals, where it sometimes rages epidemically in the halls where the whooping-cough subjects are isolated. When it rav-

ages a community where whooping-cough prevails, it is particularly formidable, for it may seize upon children whose mild form of whooping-cough would seem to promise immunity against this complication. It then assumes the manner of a contagious malady, and may be compared to the broncho-pneumonia which supervenes as a complication of measles.

Generally speaking, broncho-pneumonia ensues as a complication in one-fifth the total number of whooping-cough cases of whatever intensity (Joffroy). In a review of 431 cases of whooping-cough, H. Roger cites 68 cases of pneumonia and broncho-pneumonia. Of these 68 cases, 51 resulted fatally.

Certain secondary causes favor the development of broncho-pneumonia:

Age, first. The younger the child, the more frequent the broncho-pneumonia, being extremely so among children at the breast. Up to the age of two years it is still frequent—in such degree that in children of less than two years the appearance of broncho-pneumonia may be apprehended, even though the whooping-cough be one of slight intensity (Desruelles, Damaschino).

Cold and humid seasons play a certain part in favoring the appearance of bronchitis *à frigore*, which, at a given moment, may be complicated with broncho-pneumonia.

Its development is not influenced by temperament, although certain writers declare it more frequent in feeble children.

Prior thoracic affections predispose to broncho-pneumonia. The same is true of intercurrent infectious maladies, especially of measles. When whooping-cough is complicated with measles, broncho-pneumonia is almost invariably mortal (H. Roger).

The influence of cold, regarded as incontestable by ancient authors, seems doubtful. The cold does not seem to act save in favoring the bronchitis which may be finally complicated with lobular pneumonia.

At what period of whooping-cough does broncho-pneumonia develop? It is exceptional during the first period, not being then observed save occasionally among new-born infants. It is almost always observed during the second period when the paroxysms are in full development. During this period, also, its frequency varies according to the age of the malady. Rare during the first week, it is specially frequent during the second and third weeks. Later on it becomes much less frequent, and is altogether exceptional during the last period, when it occurs only by accident—if another infectious malady supervene, notably measles.

The *début* of broncho-pneumonia is most commonly insidious, and its appearance is marked by two principal symptoms: fever and dyspnœa.

The fever is generally intense from the outset. The temperature, but slightly elevated in uncomplicated whooping-cough, rises considerably from the *début* of broncho-pneumonia, the thermometer regis-



tering  $39^{\circ}$  to  $40^{\circ}$  C. and several tenths. The pulse, already accelerated in typical whooping-cough, becomes extremely rapid. It is not rare to count 140 or 150 pulsations from the outset. At the same time the skin is hot and dry. The visage is red and animated. The cheekbones are red or violet.

Dyspnœa prevails from the first days, revealing itself by movement of the nostrils—a well known and important indication of thoracic complication in the diseases of childhood. The movement of the nostrils sometimes renders it possible, ere any examination is made, to diagnose the existence of a broncho-pneumonia at its *début*, and it suggests the possibility of this complication when the pulmonary lesions, still central, are not perceptible to stethoscopic examination.

Dyspnœa manifests itself also by an augmented number of respirations—always considerable,—by distension of the veins of the neck, by the substernal and supersternal inspiratory sounds, by the purple color of the face.

The cough assumes special characteristics. The Hippocratic axiom, “*Febris spasmos solvit*,” applied by Trousseau to the fever of broncho-pneumonia complicating whooping-cough, is not absolutely true of the cough and the paroxysms. And yet, if the supervention of a broncho-pneumonia does not suppress the paroxysms, it modifies their characteristic features; while remaining as frequent, they become

less violent. The cough grows dull and smothered. The whistling or sibilant access loses its peculiarities, often ceasing to be even perceptible (Rilliet and Barthez). The returning crises are briefer and less numerous. At the outset the paroxysm is still followed by expectoration; and in proportion to the want of vital power produced by the broncho-pneumonia the ejection of expectorated matters grows even more difficult. Subsequently expectoration diminishes and may even cease completely.

At the *début* of broncho-pneumonia physical examination of the chest yields little useful information. Later on, by percussion we may perceive the existence of dull zones, and, by auscultation, of subcrepitant râles and souffle. The broncho-pneumonia of whooping-cough has no other special physical signs beyond those observed in any broncho-pneumonia.

Its evolution as a complication of whooping-cough is generally rapid. Dyspnœa progresses steadily to a fatal termination, which is the common consequence of broncho-pneumonia in whooping-cough. Death, generally speedy, supervenes after a few days.

At times, however, cure may be effected when the lesions have been purely congestive and characterized as such by their mobility as revealed on stethoscopic examination.

The broncho-pneumonia of whooping-cough may be prolonged and become chronic. After an acute

phase of several days, marked by fever and dyspnœa, the temperature falls; convalescence promises to begin; but, after a brief remission, the evening temperature again rises, dyspnœa reappears, and presently are manifested the clinical symptoms of chronic broncho-pneumonia.

The chronic broncho-pneumonia of whooping-cough is most frequently attended with a considerable dilatation of the bronchi, imparting to it a special aspect, and producing veritable cavities or vomicas, with expectoration of a gangrenous odor. The general condition recalls that of tuberculosis. The emaciated children, profoundly cachectic, present continued dyspnœa. Fever prevails every evening.

Death is the termination of chronic broncho-pneumonia, and generally ensues after several months of cachexia.

When this complication assumes a chronic form, whooping-cough ceases to manifest itself by its habitual signs. The paroxysms disappear. The cough persists and is then related to the lesions of the dilated bronchi, not to the whooping-cough.

The expectoration accompanying this cough differs from that following the paroxysms, being formed of purulent matter of gangrenous odor; in this matter little plugs of mucus are found, similar to those described by Dietrich.

The broncho-pneumonia of whooping-cough is comparable, from an anatomical point of view, to

the other varieties of broncho-pneumonia. All the anatomical forms of broncho-pneumonia may be observed, the most frequent being the spleno-pneumonic form as described in Joffroy's thesis.

Kromayer, who studied the anatomical condition of three children, dead of lobular pneumonia as a result of whooping-cough, has described some interesting microscopical alterations. Two orders of lesions, he states, may be observed. In the first group the pulmonary alveoli contain round cells and detached epithelial cells; small quantities of blood and fibrin are there found; the alveolar spaces are enlarged; the connective tissue surrounding the bronchi is slightly modified and presents only rare leucocytes. In the second group the alterations are more interesting and explain the frequent passage of this broncho-pneumonia into a chronic state, as also the dilatation of the bronchi and the pulmonary cirrhosis resulting therefrom. In these broncho-pneumonias we see surrounding the foci an abundant proliferation of peri-bronchial and peri-vascular connective tissue. The walls of the alveolar ducts are thickened. In addition to this interstitial proliferation of the connective tissue, the endothelial cells of the alveoli are multiplied, become cubic and form circular or tubular trains. The supra- and intra-lobular bronchi are filled with cylindrical epithelial cells, with leucocytes and products of alveolar secretion.

The foci of broncho-pneumonia in the second

group are more numerous. Kromayer considers them as characteristic of this variety.

The analogy existing between the broncho-pneumonia of whooping-cough and the other varieties of secondary broncho-pneumonia, anatomically as well as clinically, warrants the supposition that it is due to the same pathogenic micro-organism, that is, to the streptococcus whose preponderating rôle in the pathogeny of the broncho-pneumonia of infectious maladies has been demonstrated by Mosny. It cannot be ascribed to the microbe held to be pathogenic of whooping-cough by Afanassiew and Wendt, for these authors have not found the *bacillus tussis convulsivæ* in the pulmonary lesions.

Like the other broncho-pneumonias of infectious maladies, that of whooping-cough is secondary and due to superadded infection. It seems to be conditioned by the streptococcus, whether this microbe proceed from the mouth or the first air-passages where it may exist amid normal conditions (Von Besser), or whether it be derived from the external air where it may be held in suspension, as demonstrated by the experiments of Straus and Wurtz. The presence of the streptococcus in the expired air accounts for the frequency of broncho-pneumonia in hospital wards, where it often rages epidemically.

Whooping-cough prepares the organism for infection from the streptococcus. The secondary infection, according to Mosny, may be explained in several ways:

by chemical action, the primary infection modifying the normal acid reaction of the lung; by physiological action, the primary infection impeding the phagocytic function of the cells of pulmonary epithelium and of the leucocytes; by mechanical action in modifying the broncho-pulmonary epithelium and promoting the invasion of the organism by the streptococcus.

Such invasion of the lungs is the principal cause of the broncho-pneumonias of whooping-cough. In rare cases other micro-organisms intervene, which seem to play a rôle in the development of this complication, or at least of some of its varieties. In three cases of broncho-pneumonia, developed during whooping-cough, and followed by cure, Haushalter established, during the evolution of the pulmonary complication, the presence of the *gilded* streptococcus in the patient's blood. This microbe has yielded virulent cultures which, injected into the circulation of rabbits, have determined fatal broncho-pneumonia.

The broncho-pneumonia of whooping cough may produce a series of secondary complications which are rather conditioned by the pulmonary complication than by the primary infectious malady. They have no features peculiar to whooping-cough. In this connection we have before mentioned the chronic dilatation of the bronchi, which is an ordinary consequence of chronic broncho-pneumonia.

*Acute dilatation of the bronchi* is quite frequent in whooping-cough. It is conditioned by the concomi-

tant bronchitis, and is not of great importance from a prognostic point of view. It is not characterized by special stethoscopic signs, and is often one of the revelations of an autopsy. Acute bronchial dilatation is commonly generalized.

*Emphysema* is frequent in whooping-cough. Its presence is readily explained in this malady where the shock of the cough exhibits a violence rarely existing in the other affections of the respiratory passages. Two forms are observed: alveolar and interlobular emphysema.

*Alveolar emphysema* exists in acute form in most whooping-cough patients, and is almost uniformly revealed by autopsy. According to H. Roger, it disappears after cure of the infectious malady. Trousseau claims that it may persist and finally assume the form of constitutional emphysema.

*Interlobular emphysema* is much more serious. It may be limited to the lungs or may become general—may gain the cellular tissue of the mediastinum, and manifest itself in the cervical region by the presence of gaseous, sonorous tumors whose contents may be emptied by gentle pressure, causing access of air into the cellular tissue. This emphysema is observed only in grave whooping-cough. Vorthrup has seen it accompany pulmonary abscess in an infant two months old.

With this purely mechanical lesion we may associate pneumothorax, observed by H. Roger; the

mode of its development was almost latent. The effusion of air into the pleura was slowly absorbed without causing liquid effusion. The patient was cured.

*Hernia of the lung* was reported by Adler in an infant, occurring at the level of the nipple-line and at the height of the sixth rib, where it appeared like a tumor of the size of a plum. This tumor evidently consisted of a pneumocele; fluctuated; was sonorous; and varied in volume during the respiratory movements. It was susceptible of complete reduction and gave rise to a crepitant murmur. Auscultation of the tumor revealed the vesicular sound.

*Pleurisy* is very rare in whooping-cough, and when it does occur it is most commonly the cause of a broncho-pneumonia. It has been observed, though rarely, during convalescence from whooping-cough, independently of any pre-existing pulmonary complication.

Pleurisy, as observed in whooping-cough, is serous or sero-purulent.

Pericarditis has been reported by Racchi, who observed one case of sero-fibrinous pericarditis in the course of a whooping-cough complicated with broncho-pneumonia. The sero-fibrinous liquid contained in the pericardial cavity was injected into three rabbits and determined a convulsive cough in these animals, and they infected others in neighboring cages. Autopsy performed on several showed pericarditis.



## LARYNGEAL COMPLICATIONS.

Catarrhal laryngitis is frequent in whooping-cough, often accompanying the bronchitis. Rare at the *début*, it is mostly observed during the period of maturity. It is seldom intense, and commonly produces only a transient hoarseness. Several authors, struck with the frequency of the congestive and catarrhal alterations of the subglottic region, of the vestibule and of the vocal cords, observed on autopsy, have ascribed an important rôle to the laryngitis in the production of the paroxysms. This rôle is, however, dubious, for the alterations in the larynx, though frequent, are not constant. In several rare cases, reported and studied by R. Blache, the laryngitis of whooping-cough was accompanied with serous infiltration of the cellular tissue of the subglottic region, and gave rise to grave accidents of glottic œdema.

Laryngeal vertigo has been twice observed by Thermes in aged sufferers from whooping-cough.

Of all laryngeal complications which may be observed in the course of whooping-cough, the most serious is glottic spasm. This formidable accident has been studied by Du Castel, who has published seven observations concerning it. Its essential feature is the absence of the sudden and sibilant inspiration. The spasms of cough occur as in the paroxysms of ordinary whooping-cough; but the inspiration which commonly prevents asphyxia is absent in these suffer-

ers, and suspension of inspiration is the cause of asphyxia.

The accesses of suffocation due to spasm of the glottis, commonly supervene in the paroxysms of cough, but they may ensue independently of any convulsive cough (Du Castel).

Glottic spasm may produce rapid and almost sudden death, and to this accident are due almost all the cases of sudden death observed in whooping-cough.

In a certain number of cases the accidents are less grave, then constituting an attenuated form of glottic spasm. In this form, after a momentary arrest, respiration is resumed, and the return to life is announced by several deep and convulsive sibilant inspirations.

Autopsy performed on children who have succumbed to spasm of the glottis reveals the existence of a very pronounced emphysema, at once vesicular and interlobular, and a state of the lungs corresponding to the asphyxia. The pleural ecchymoses described by Tardieu are frequent.

The sudden death which has been at times attributed to excessive bronchial secretion, and at times to pulmonary embolism, is due in the immense majority of cases to the accidents produced by glottic spasm. There is spasm of the constrictor muscles of the glottis, with persistent contraction of the expiratory muscles.

## NERVOUS COMPLICATIONS.

The most frequent, and at the same time the most serious, of the nervous complications of whooping-cough, are presented in the form of accesses of eclampsia. The frequency of the latter in the acute diseases of childhood is well known. This nervous complication is, then, readily explained in whooping-cough, where the spasmodic element plays a rôle of the utmost importance.

Glottic spasm, due to an internal convulsion of the inspiratory muscles and of the constrictor muscles of the glottis, is a variety of internal eclampsia. True eclampsia manifests itself by external convulsions. It attacks particularly very young children—those who have not yet passed their third year—and is observed almost exclusively in grave cases of whooping-cough. This complication is quite rare. In 431 cases of whooping-cough, H. Roger observed it only fifteen times.

Eclampsia occurs under two conditions. Most commonly it supervenes when whooping-cough is complicated with a broncho-pneumonia. It then marks the *début* of the latter. More rarely, it supervenes in the absence of any thoracic complication.

According to Rilliet and Barthez, the convulsions are preceded by several premonitory phenomena, consisting of nervous agitation and drowsiness. They are sometimes announced by cephalalgia (Abercrombie), or may make their *début* suddenly.

The convulsions ordinarily make their appearance during a violent paroxysm. Sometimes, but rarely, they begin in an interval between paroxysms. The sudden *début* without precursory signs takes place only when the convulsions are related to a pulmonary complication.

The convulsions are partial or general. When partial they consist in tonic and clonic convulsions of the muscles of the face or of the members. When general they extend to all the muscles of the body. During the crisis intelligence is momentarily suspended; insensibility is complete. In ordinary cases, the convulsions, at first tonic, become clonic in a second phase. The first crisis may be terminated by death, most frequently caused by glottic spasm (internal convulsion) accompanying the external convulsions. When, as is the rule, the first accesses do not prove fatal, the convulsions are followed by a phase of coma. Presently new convulsions take place, alternating with the coma. This state of excitation and nervous depression generally ends in death in a short space of time (one, two or three days). Children generally succumb during the twenty-four hours following the appearance of the first crisis (Rilliet and Barthez).

The immediate effect of the convulsions is to produce certain modifications of the cough. The paroxysms are not suppressed, but become less frequent. The expiratory spasms are less loud and

numerous. The whistling inspiration ceases to occur.

The convulsions are of extreme gravity in whooping-cough. When they accompany a pulmonary complication such as broncho-pneumonia, they inevitably end in early death. Supervening independently of any complication, the prognosis still remains of the utmost gravity. Six times out of seven they cause death (Rilliet and Barthez). When they do not kill through spasm of the glottis, they kill by the cerebral hyperæmia, evident signs of which are revealed by autopsy.

Eclampsia is due to congestive lesions of the brain, which leave behind traces appreciable with difficulty on anatomical examination. It is not conditioned by organic lesions such as hæmorrhage or softening. When whooping-cough determines organic lesions of the cerebrum, they manifest themselves by the ordinary symptoms of lesions with foci, particularly by hemiplegia (cases of West and Radcliffe Croker; cases of right hemiplegia with paralysis of the left oculo-motor and transient hemianopsia, observed by Silex).

With respect to the peripheral nervous system, Moussous and Moebius have reported acute paralysees during convalescence from whooping-cough. These acute paralysees are conditioned by a generalized acute polyneuritis.

Moebius' case was that of a child three years old in whom the paralysis made its *début* six weeks after

the beginning of the whooping-cough. The paralysis was characterized by difficulty in walking and in maintaining a sitting posture. As in paralyzes due to peripheral neuritis, the rotular reflexes were abolished. The paralysis extends to the muscles of the upper members and those of the neck. The sphincters remain normal. There is no muscular atrophy. Improvement was rapid; three weeks after the *début* of the accidents every trace of paralysis had disappeared, but the tendinous reflexes still remained absent—these reappeared after the lapse of another month.

*Organs of Sense.*—*Amaurosis* has been likewise observed by Alexander, as a consequence of whooping-cough. In his first case—that of a child three years old, terminating fatally—complete blindness supervened without external lesion of the eye. On examination with the ophthalmoscope a slight dilatation of the central vein of the retina was observed. Shortly after the inception of blindness, cerebral symptoms, with convulsions of the right members, developed, and carried off the child at the end of fifteen days.

In a second case, after several precursory symptoms, consisting of chronic headache and dimness of vision, a girl twelve years old, suffering from whooping-cough, became completely blind. In this case the pupils were dilated, immobile, and no longer reacted under the influence of light. Alexander determined

the existence of a double optical neuritis with slight swelling of the two papillæ, whose contours had lost their distinctness. After several days, vision and optical neuritis progressively improved, and the pupils again reacted to light. Nevertheless, in publishing his observations, Alexander did not venture to assert definitive cure.

Alexander explains the supervention of blindness in the first case by a localized cerebral œdema between the quadrigeminal tubercles and the occipital region; in the second case he attributes it to a descending optical neuritis following a meningitis of the base, which is stated to have been cured.

Acute and transitory blindness has been reported by Jacoby.

Whooping-cough may produce deafness by a double mechanism (Falls). Sometimes the deafness supervenes after recovery from the whooping-cough. It is unilateral or double. The external and the middle ear enjoy an indemnity from any appreciable alteration. The deafness is then the consequence of an affection of the labyrinth (Falls). In another series of cases suppurating otitis media prevails, which may finally become complicated with lesions of the internal ear.

In terminating this review of the nervous complications of whooping-cough, we should not overlook its influence on the subsequent development of a certain number of nervous diseases supervening long after recovery from the original malady.

The labors of Pierre Marie on the pathogeny of several affections of the nervous system have revealed the rôle played by prior infection in the development of these diseases. He maintains that insular sclerosis, epilepsy, and cerebral sclerosis are often the consequence of the infectious maladies of infancy.

Whooping-cough is often revealed in the antecedents of the patient suffering from these nervous affections: There is accordingly good ground for taking into account its pathogenic influence on their development.



## COMPLICATIONS OF THE MOUTH.

Ulceration of the frenum of the tongue may be the occasion of severe complications during the secondary infections. In permanent contact with numerous microbes of the mouth this ulceration may be infected by them, and such is the pathogeny of ulcerous stomatitis, simple or accompanied with alveolar osteitis. At times the ulceration serves as a portal for the admission of saprophytes, producing gangrene of the mouth, which has been repeatedly observed during the course of, or convalescence from, whooping-cough. Most commonly these diverse buccal complications are conditioned by the existence of an ulceration of the frenum of the tongue, but this ulceration is not indispensable. An aphthous stomatitis may develop during whooping-cough when there is no ulceration of the frenum, and may cause ulcerous stomatitis or gangrene of the mouth.

The habitual facial congestion and the mechanical embarrassment of the venous circulation greatly promote the production of these complications.

## SECONDARY INFECTIONS.

Among the complications of whooping-cough which we have now studied, many are due to secondary infections. To cite only the most frequent and serious, broncho-pneumonia seems to be manifestly conditioned by a superadded infection.

The secondary infections which remain to be treated are purely accidental and are not peculiar to whooping-cough. The most important is tuberculosis in any one of its forms.

Uniformly tuberculosis has been reported as prevailing during or after the whooping-cough, and has been considered as one of its frequent complications; but the pathogenic rôle of whooping-cough long remained uncertain and variously interpreted. It is now admitted that tuberculosis is always the result of a secondary infection—the consequence of a subsequent contagion. In the children's hospitals where the whooping-cough patients are surrounded by tuberculous subjects, the possibility and the reality of contagion are readily comprehended. The conditions of contagion are not lacking; its possibility is obvious. Hospital patients live in an environment of tubercular bacilli. They are debilitated by the whooping-cough and exhausted by the vomiting which impedes normal nutrition. The respiratory organs are irritated, if not inflamed. They present to the tubercular bacillus a soil all prepared for its germination.

Tuberculosis may develop during the course of a whooping-cough in process of evolution; but most commonly its first signs are observed during convalescence.

The state of things generally perceived is as follows: The whooping-cough is declining when the child begins to manifest the first signs of tuberculosis; in most cases this is of the chronic pulmonary type, of which a fever, aggravated in the afternoon, is the first sign. If no bronchitis prevailed before, the cough which then ensues presents characteristics different from those of whooping-cough paroxysms. This cough is dry and not followed by expectoration; it is the characteristic cough of bronchitis. The paroxysms of whooping-cough do not cease completely, but they become less violent, and notably less frequent; the tubercular process, in course of evolution, attenuates their intensity and masks their characteristics, producing upon them the same effects as does broncho-pneumonia. The persistent paroxysms seem to be smothered or suppressed; the sibilant respiration ceases to be heard.

Subsequently the paroxysms disappear, the symptoms of pulmonary tuberculosis at the same time becoming more persistent and accentuated. The child recovers from the whooping-cough while the tuberculosis continues its generally rapid evolution.

Pulmonary tuberculosis with successive foci is not the only form observed. Frequently the tuber-

culosis is manifested in the form of tuberculous broncho-pneumonia. One or several lobes of the lung are invaded at the outset. Fever is then intense and continued. The cough is incessant; asphyxia progresses rapidly. In such instances the tuberculosis develops in the manner of a subacute broncho-pneumonia. On autopsy, several pulmonary lobes are found infiltrated with cheesy masses—not soft, but with the familiar aspect of Roquefort cheese. With these tuberculous masses so common in infancy, and which ally this form of pulmonary tuberculosis with experimental tuberculosis, disseminated tubercles almost always exist in most of the organs, and particularly in the liver.

Whooping-cough is also followed by tuberculosis of the miliary type, with the deposit of tubercles in most of the organs. This form of tuberculosis presents rather the clinical tableau of typhoid fever than that of an affection of the respiratory passages.

Meningeal tuberculosis has been reported by various authors as one of the possible forms of tuberculosis consecutive upon whooping-cough.

The bronchial ganglia, which quite commonly undergo modification in whooping-cough, are frequently the first organs in which the deposition of tubercles is made. In such case the initial phenomena are those of tuberculosis of the tracheo-bronchial ganglia. Limited to these ganglia at the outset, tuberculosis soon invades the lungs or other viscera

and produces death through tuberculous generalization.

Thus far we have regarded tuberculosis as a complication of whooping-cough; the converse may be encountered. Tuberculous patients are occasionally attacked by whooping-cough during the course of phthisis. In such instances, what is the influence of the whooping-cough on the tuberculosis in course of evolution? The tuberculosis does not prevent the production of the paroxysms, but, by attenuating their symptoms, it renders them less characteristic. The two maladies progress, each presenting its own distinctive signs. In these conditions the effect of the whooping-cough is frequently to accelerate the evolution of the tuberculosis.

Other infectious maladies may also complicate whooping-cough, and are due, as is tuberculosis, to a superadded contagion. The morbid association of whooping-cough with the infectious diseases of infancy has been particularly observed in the hospitals, and may be readily explained by the promiscuous conditions which long prevailed in the wards of children's hospitals, where were gathered together pell-mell patients suffering from diverse infectious diseases. In those days it was no unusual thing for patients suffering from whooping-cough, measles, scarlatina, and even diphtheria, to be attended by the same *personnel* and in the same ward.

In reality there is nothing mysterious in the as-

sociation of whooping-cough with other contagious maladies. There is no need of assuming a special affinity of the virus of whooping-cough with that of other infectious maladies. The contagion is a sufficient explanation of these morbid associations. If whooping-cough is quite often complicated at the hospital with another infectious malady, the cause is some disease of long duration necessitating a sojourn of several weeks in an infected environment. Whooping-cough, not confining children to their beds, permits more frequent and more intimate contact with the other patients (H. Roger). The duration of the sojourn at the hospital, and the possibility of contact, sufficiently explain the frequency of secondary contagions.

The most frequent infectious complication of whooping-cough in children's hospitals is measles. Out of 431 cases observed at l'Hôpital des Enfants, H. Roger observed seventy-eight which were complicated with measles.

The morbid association of whooping-cough and measles has long been the subject of reports during epidemics of the first-mentioned. Some authors have been so struck with this phenomenon that they have inferred an intimate relationship between the two maladies. Franck maintained their identity and their common origin. In 1854 Germain Sée set forth the analogies between measles and whooping-cough with respect to their evolution and their special com-

plications, as also their frequent coexistence, and deduced their common origin.

The doctrine of the identity of whooping-cough and measles is sufficiently controverted by clinical observation, and has been definitively refuted by Rilliet and Barthez and by H. Roger.

Measles may precede whooping-cough or may follow it. Most commonly it precedes, but this is due simply to the fact that measles, more frequent than whooping-cough, more rapid in its course, and more serious in its symptoms, contributes a greater number of patients to the hospital.

When whooping-cough is complicated with measles, the ordinary symptoms persist, with the usual attenuation which accompanies any intercurrent febrile state. The paroxysms are quite as numerous, but duller and less violent; during their intervals the children exhibit the loose cough common in measles, which is always accompanied with bronchitis.

Whooping-cough exerts no action on the eruptive phenomena of the malady. The eruption proceeds as usual and may be extremely intense.

The morbid association of whooping-cough with measles is really peculiar in its broncho-pneumonic complications, which are, we may say, the rule, and which impart to this association an extreme gravity. In two cases out of three, whooping-cough associated with measles is complicated with broncho-pneumonia

or capillary bronchitis. The complications may occur at any period in the malady, but most often develop early. In the Geneva epidemic of 1847, Rilliet and Barthez observed the invasion of capillary bronchitis and of broncho-pneumonia on the very day of the *début* of the paroxysmic period—at a time, consequently, when these complications are exceptional in simple whooping-cough. It seems that these two diseases, whose principal action is brought to bear on the respiratory apparatus, combine their effects and facilitate thus the invasion of the lungs and of the bronchi by the pathogenic micro-organisms of broncho-pneumonia.

Whooping-cough may be associated with diphtheria under the same conditions as measles, that is, as the result of a superadded contagion. H. Roger observed this morbid association twenty-four times at the Children's Hospital. In fourteen cases the diphtheria was of the croupal type, and terminated nine times in death; of the five other cases, which were cured, in three tracheotomy was performed. H. Roger thinks that in such cases the influence of the whooping-cough is rather favorable. The paroxysms and the vomiting facilitate the mechanical detachment of the false membranes, and serve the purpose of emetics which formerly enjoyed such a vogue in the treatment of laryngeal diphtheria.

Sanné, who in four cases observed the complication of whooping-cough with diphtheria, saw his pa-



tients die of broncho-pneumonia. He accordingly concluded that whooping-cough, when complicating diphtheria, exerts a pernicious influence on the latter.

When whooping-cough is complicated by diphtheria the paroxysms persist, but the characteristic sibilance is not produced. The paroxysms do not increase the number of the dyspnœic accesses of croup (Sanné).

Scarlatina may also complicate whooping-cough, and this complication was observed by Sanné in eleven cases. According to H. Roger a benign scarlatina has no influence on the symptoms and evolution of whooping-cough.

According to the observations of Sanné, scarlatina may be modified at times by the absence of one of the principal symptoms (the angina was absent in one case), at other times by the addition of abnormal and serious phenomena such as cerebral disturbances. Five out of the eleven patients of Sanné succumbed. The invasion of scarlatina modified the paroxysms of cough, diminishing their number and intensity. The paroxysms returned after the disappearance of the scarlatinous exanthema. The amelioration sometimes persists (Sanné).

Smallpox may occasionally complicate whooping-cough (H. Roger). If not serious, and if the case be one of varioloid, it neither mitigates nor aggravates the whooping-cough.

## FORMS OF WHOOPING-COUGH.

The disease which I have thus far described is whooping-cough of average intensity as it is most frequently observed in children of from three to seven or eight years of age. Whooping-cough, even the non-complicated, is not always thus presented to clinical observation, being subject to variations which depend upon the intensity of the malady and the age of the patients; and there is good ground for studying these variations in accordance with the two factors—intensity, and age of the subject. The differences are at times sufficiently pronounced to constitute special forms of whooping-cough.

With respect to the intensity of the disease, the different cases may be divided into three classes. According to the division of H. Roger, whooping-cough may occur in three forms:

1. Mild whooping-cough.
2. Whooping-cough of mean or average intensity.
3. Intense whooping-cough.

These three forms are differentiated solely by the number and intensity of the paroxysms, and all three proceed from the same pathogenic virus and may mutually engender one another.

The mildest whooping-cough may by contagion give rise to the most dangerous form, just as intense whooping-cough may determine a very benignant attack. The virus is identical. The degrees depend

upon the conditions of receptivity which permit exaltation or attenuation of the morbid virus.

Whooping-cough of mean or average intensity is the most frequently observed in the clinic, and this I have selected as the type of my description.

Imagine this same whooping-cough with a greater or lesser number of paroxysms and with secondary symptoms correlated to the attenuated or reinforced paroxysm, and you will have the tableau of the other morbid forms of the disease.

Mild whooping-cough is quite rare. In this type the paroxysms are few in number; from twenty to thirty (the ordinary number during the twenty-four hours in whooping-cough of mean intensity), the paroxysms fall to five or six, sometimes more, sometimes less. In certain cases only two or three (and these not severe) occur during the twenty-four hours. Some are only intimated, as it were, being composed only of expiratory spasms without sibilant inspiration. Expectoration is scanty, sometimes *nil*. Vomiting is exceptional; during the intervals the health is good. Bronchitis is absent, and auscultation of the chest reveals no signs of bronchial catarrh; temperature is normal. In this form the complications are exceptional.

The mild form of whooping-cough performs its evolution in a relatively short time, and is always followed by recovery. Each of the periods is abbreviated, the spasmodic period particularly.

In this form whooping-cough is a benignant malady, producing no disturbance of the general health, hence requiring no active medical intervention. Most commonly the children do not keep their room, but continue to go out and to participate in their ordinary pleasures. The very benignity of the disease, necessitating no assiduous surveillance, often results in facilitating its dissemination in the circle of the patient's acquaintance, and with respect to the diffusion of whooping-cough this form is the most dangerous.

Grave or intense whooping-cough is the form of the disease in which the paroxysms attain their maximum frequency and intensity. In this form a considerable number of paroxysms is noted—twenty-four and more during the twenty-four hours. The paroxysms are not only numerous, but prolonged and intense. As each supervenes, the physician seems to behold a veritable access of suffocation. During these paroxysms the face is purple; the injected eyes protrude from their orbits; the extremities are cold. Blood frequently issues from the nostrils and ears, or appears in the form of ecchymoses in the sub-conjunctival cellular tissue.

The vomitings are constant, and are renewed at each access.

The paroxysms follow in such close succession that hæmatisis, interrupted for the time being cannot proceed to a completion during their inter-

vals. Thus, respiration becomes permanently impeded. In certain patients the asphyxia progresses uninterruptedly and ends by causing death, in the absence of any complication.

Intense whooping-cough is always accompanied with grave general phenomena. The temperature is constantly elevated; the pulse is excessively rapid; dyspnoea is extreme. Auscultation reveals the existence of a generalized bronchitis extending to the lesser bronchial ramifications.

This grave form of whooping-cough commonly ends in death. The fatal termination may be due to progressive asphyxia; sometimes it is the consequence of inanition caused by the incessant vomiting, which is renewed at every attempted feeding; or, finally, it may be due to complications—this form being especially subject to broncho-pneumonia or to the convulsions whose prognosis is almost uniformly fatal.

Despite these numerous causes of death, grave whooping-cough may yet result in recovery if the child be particularly robust. When terminating favorably, this form of whooping-cough is always of long duration and subject to numerous relapses during convalescence. It is often attended with a sequel in the form of lesions of emphysema or of bronchial dilatation. It is particularly true of this form of the disease that long after recovery the children retain a predisposition to paroxysmic cough, which reappears on the occasion of an accidental bronchitis or of any malady of the respiratory passages.

*The Influence of Age on the Whooping-Cough.*— Generally speaking, the gravity of whooping-cough is in inverse ratio to the age of the patient; the younger the subject, the more serious the disease. In this respect whooping-cough in children at the breast is the most dangerous of all, as much by reason of the intensity which it commonly assumes during the first months of life as on account of the slight resisting power of its victims.

*In new-born infants* whooping-cough generally makes a rapid *début*. The first period is notably abridged; the paroxysms make their appearance speedily, and are violent and repeated from the outset. Fever is constant. The disease is almost uniformly complicated with broncho-pneumonia, and fatal in its termination.

The difficulty of alimentation aggravates the prognosis of whooping-cough in the new-born. Any attempt at nursing determines a paroxysm; hence the children instinctively refuse to take the breast. In most cases recourse must be had to forced alimentation, and this requires great caution, for the introduction of the sound, or frequently of an ordinary spoon, is enough to provoke a paroxysm.

During the first days of life the prognosis of whooping-cough is absolutely grave, and the infants stricken at the moment of birth almost inevitably succumb.

The whooping-cough of adults is generally be-

nignant, and, according to H. Roger, it is to this form that the description of mild whooping-cough applies. It differs from the disease as exhibited in children by the modifications which the paroxysms undergo, and which are conditioned by the different structure of the larynx. The relative width of the glottic orifice renders the contraction of the glottis incomplete. Hence the paroxysms are composed solely of expiratory spasms, generally separated by an interval and broken by periods of repose. The sibilant inspiration, which is due in infants to the passage of air through the constricted glottic cleft, is not observed. The expectorated matters are slowly expelled; they are not vomited as in infancy. The cough in adults recalls that of tuberculous subjects whose tracheo-bronchial ganglia are hypertrophied.

Adult whooping-cough is not attended with general phenomena, and it is rarely of such gravity as to require confinement to the room. It is almost uniformly accompanied with bronchitis. Hence, in connection with the paroxysms, which are few in number and are modified in their clinical expression, a cough almost always prevails, which is bronchial in character. Complications are altogether exceptional.

Despite its benignity, adult whooping-cough generally persists for a long time; its duration is rarely less than a couple of months.

## PROGNOSIS OF WHOOPING-COUGH.

The gravity of whooping-cough is in direct ratio to the number of paroxysms. When the latter are few in number—not more than ten or twelve during the twenty-four hours—recovery is almost certain, and death exceptional. When they are very numerous, and exceed seventy or eighty during the twenty-four hours, the whooping-cough is extremely grave and commonly results in death. Trousseau declared death to be certain when the paroxysms reached the number of sixty. This prognosis is too rigorous, for H. Roger has cited examples of cure under such circumstances. And yet, when the paroxysms are so frequent, the disease is of the utmost gravity, and results fatally in the great majority of cases.

With respect to the division of the disease into mild and grave cases, it may be stated that one-quarter of the total number are intense, with more than forty to fifty paroxysms during the twenty-four hours. Another quarter is composed of mild cases with the paroxysms numbering less than fifteen during the twenty-four hours. A half of all cases are of mean intensity, the number of paroxysms varying between fifteen and thirty. Whooping-cough of mean intensity is accordingly the most frequent, and is usually followed by recovery; when terminating fatally, death is the sequel of a complication.

Age is a factor of the utmost importance from a



prognostic point of view. In early infancy, under one year of age, whooping-cough is extremely grave, for the reasons which we have already indicated. At this term of life the disease is almost always intense, and its gravity is aggravated by the feebleness of the patients and the difficulty of alimentation.

During the second year whooping-cough is not infrequently fatal. After the third year, the dominating type is milder up to the eighth or ninth year; later on, it becomes benign and is almost always cured.

Environment plays a very important rôle in making prognosis. In the hospital, whooping-cough is much more dangerous than in city practice—in consequence of assembling all the causes of secondary infection, which are so important in the genesis of the complications. In the city the child escapes the secondary infections, and has far more chance of recovery.

Whooping-cough seems to partake of a graver character in certain countries (particularly in northern lands) than in temperate regions; in warm climates its gravity seems to be least. In Greenland, whooping-cough is stated to be one of the principal causes of death (H. Roger).

We have seen that a quarter of all cases of whooping-cough are serious and terminate almost uniformly in death. Furthermore, cases of mean intensity may result fatally in consequence of a com-

plication. We may accordingly conclude that 35 to 40 per cent. of the cases observed in hospital have a fatal termination; and this proportion is in accord with that of H. Roger, who found death to ensue in the ratio of 142 out of 423 cases observed in his department of the Hospital for Sick Children.

The figures cited by this author are interesting, for they show clearly the influence of age on the prognosis.

Out of 424 cases H. Roger observed:

	No. of Cases.	No. of Deaths.
Between birth and 2 years of age....	16	11
Between 2 and 3 years .....	106	64
Up to 3 years of age .....	85	31
Up to 4 years.....	77	35
Up to 5 years.....	63	15
Between 6 and 14 years .....	77	19
From 9 to 13 years.....	..	0

What is the cause of death in whooping-cough?

In rare instances death may be sudden. As Du Castel has shown, it is then due to spasm of the glottis, by convulsion of constrictor muscles of this orifice. This rule is not absolute, however, as sudden death is sometimes attributable to other causes—according to some authorities, to functional disturbance of the pneumogastric.

Sometimes death is caused by progressive asphyxia, and this is observed when the numerous and close-following paroxysms produce permanent em-

barrassment of respiration and render impossible the fulfillment of hæmatosis amid normal conditions.

In exceptional cases the digestive troubles and the incessant vomiting, repeated at every attempt to administer food, produce death through progressive inanition.

Most commonly the fatal termination is due to some complication, and at the head of the mortal complications stands broncho-pneumonia in its various forms.

Broncho-pneumonia is almost always fatal, and progresses rapidly. Frequently enough it becomes chronic, with a prognosis less immediate but equally formidable.

Eclampsia, which is quite rare, is almost absolutely fatal and causes death after a slight delay.

With eclampsia due to encephalic congestion we must associate meningeal or cerebral hæmorrhage, to which death is occasionally due.

The other causes of death involve superadded maladies which are developed by contagion during the whooping-cough. Such is tuberculosis in any of its forms, as it is too frequently observed among children treated in hospitals. This complication rages with special severity amongst weakly, lymphatic or scrofulous infants, predisposed by heredity or by organic debility.

The prognosis of whooping-cough must be considered not alone with respect to the immediate evolu-

tion of the malady, but also with due reference to the future. Whooping-cough may, indeed, determine lesions which evolve slowly and become manifest only after a long interval.

One of the possible results of whooping-cough is vesicular emphysema, which exists throughout the duration of the malady. It may be, and commonly is, cured. But in some cases it persists, is aggravated from year to year, and finally merges into constitutional emphysema with all its consequences: accesses of asthma, cough, and permanent dyspnœa, terminating in asystolia. A certain number of emphysemas in adult age, attributed to arthritism, are probably due to no other cause than a whooping-cough during infancy, which, though cured long ago, left in its wake a pulmonary lesion which has since been aggravated from year to year.

Chronic dilatation of the bronchi may also have its origin in an ancient whooping-cough.

## DIAGNOSIS OF WHOOPING-COUGH.

The crisis of convulsive cough, constituting the paroxysm, is of so distinctive a character that the observation of one paroxysm of whooping-cough is commonly sufficient to reveal its nature. Doubt is possible only under exceptional circumstances, and the diagnosis of the disease is accordingly limited in most cases to a recognition of the paroxysms.

The difficulty of diagnosis varies considerably, according to the diverse periods of the malady.

During the first period diagnosis is almost impossible, the paroxysm which forms the pathognomonic symptom having not yet appeared. The disease may be suspected, but cannot be affirmed.

At this period the child presents the ordinary signs of a bronchial catarrh of average intensity. The general symptoms are in no wise peculiar, although it has been erroneously affirmed that in the initial catarrh of whooping-cough the fever is more intense and, especially, more persistent than in simple catarrh *à frigore*, and that the dyspnœa is also more marked. As a rule, the general symptoms during the first period of whooping-cough are identical with those observed in simple bronchitis—sometimes, indeed, they are even less grave—and it is rarely possible to establish a precise diagnosis for several days.

And yet, even in the absence of positive signs, an etiological reason will sometimes permit diagnosis of

whooping-cough at its *début*; and that is, positive information of a prior contagion. The malady may be predicted with certain assurance when cough supervenes in a child living in an environment where whooping-cough prevails, provided, of course, the child has not himself undergone a previous attack.

At the close of the first period, the cough, without being yet pathognomonic, assumes certain peculiarities which may put us on our guard. First, it becomes more frequent in the evening and during the night. In certain cases and at certain moments the cough becomes very painful and exhausting, comprising a series of expiratory spasms, like the cough which accompanies the expulsion of foreign bodies from the respiratory passages.

In some cases—and this is a more important fact—the convulsive cough is accompanied from time to time with the suggestion of a sibilant inspiration. Thus constituted, this cough precedes for a short time the true paroxysmic cough, and announces the *début* of the second period.

During the second period, if the physician happens to observe a paroxysm and hears the noise of the expiratory spasm and the sibilant inspiration—if he observes expectoration to follow and terminate the crisis,—the diagnosis becomes peremptory, for no other malady could determine a paroxysm thus characterized.

In truth, the paroxysms of cough sometimes ob-

served in simple bronchitis are never so long or so intense. They are not accompanied with sibilant inspiration and are not followed by expectoration, at least in children.

The expectoration which follows the paroxysm is of the greatest importance in diagnosis. Whooping-cough is, indeed, almost the only malady of infancy which gives rise to expectoration.

The latter is at times very abundant and may cause confusion by suggesting a pulmonary cavity (*vomica*), due to a purulent pleurisy or to a pulmonary abscess discharging into the bronchi. This error will be avoided if we remember that the expectoration due to a *vomica* is composed of greenish and liquid pus, whereas the expectoration of whooping-cough is composed of stringy matter difficult to detach and resembling a gummy emulsion. Moreover, pleural or pulmonary cavities supervene after a malady whose evolution is altogether different, and are accompanied with stethoscopic signs which are not found in whooping-cough.

*Vomicæ* determined by chronic dilatation of the bronchi may also give rise to paroxysms; the origin of the latter may be diagnosed by the characteristics of the expectorated matter, as also by the stethoscopic signs and the evolution of the malady.

The paroxysms due to cavities or *vomicæ* are never so frequent as are the paroxysms of whooping-cough. Rarely do more than three or four occur during the twenty-four hours.

The paroxysms due to tracheo-bronchial adenopathy are more difficult to recognize. We know that these paroxysms are particularly observed in the tuberculous, whose tracheo-bronchial ganglia, having become caseous, are hypertrophied.

All the tumors of the mediastinum may determine paroxysms of cough by a mechanism analogous to that of tracheo-bronchial adenopathy, that is, by compression and irritation of the pneumogastric nerves.

In infancy, tuberculization of the mediastinal ganglia is not rare. Inasmuch as the disease is of slow evolution with evening fever, and as the paroxysms which it determines are often followed by vomiting, the clinical tableau may be very analogous to that of whooping-cough.

Tuberculosis of the tracheo-bronchial ganglia is recognized in that the cough is not manifested solely by paroxysms. In the intervals a dry cough is heard, which does not exist in uncomplicated whooping-cough. Expectoration is *nil*, or if it exists at moments (which is exceptional), it has not the same characteristics as in whooping-cough. If there be expectoration, the bacillus of tuberculosis may be discovered in the expectorated matter. Finally, pulmonary tuberculosis frequently accompanies tuberculosis of the mediastinal ganglia, and gives rise to stethoscopic signs which are readily recognized.

Moreover, the general condition differs consider-



ably in ganglionic tuberculosis. Diarrhœa is frequent; the visage is emaciated, and the appearance is very different from the puffiness observed in whooping-cough. Fever is constant.

In *résumé*: The observation of the paroxysm enables us in the majority of cases to fix its origin. The paroxysms are frequently separated by intervals, and the physician may not witness one during his first visits; hence, in doubtful cases he may endeavor to provoke them by certain means. The child may be made to run, or he may be made to open his mouth, the throat being tickled with the finger or with a feather. A simple means which often succeeds consists in undressing the child and exposing its chest naked to the air of the room. Sometimes, simply stroking or tickling the skin is sufficient to produce a paroxysm. In many instances it is only necessary to administer a mouthful of cold liquid. Rarely do these measures fail; and yet, if the case be mild, they may not provoke a paroxysm. The physician is then obliged to rely on the statements of the parents or attendants. Frequently the description given by parents of experience is sufficiently exact to render possible a precise diagnosis. If the parents state spontaneously that the child expectorates after the paroxysms, this information is of great value. The same applies to epistaxis during or after the paroxysm. Nevertheless it is well to accept with certain reservations the descriptions given by those

attending the patient, and such statements must be controlled by an examination of the signs which reveal whooping-cough.

Among the betraying symptoms the most important are: puffiness of the face, habitual congestion of the veins of the neck and visage, ulceration of the frenum of the tongue in children with teeth. A possible contagion or the modifications of an ordinary cough into paroxysmic cough may put us on the road to a diagnosis. Despite all, a solution of the problem is often impossible during the first days of the second period. Later on it would be very singular if the physician had no opportunity to witness a paroxysm which would put an end to all doubt.

It sometimes happens in an environment where whooping-cough prevails that children enjoying an indemnity from the disease begin to cough in paroxysms after hearing whooping-cough patients cough in this manner. H. Roger states that he observed cases of this kind, in which, however, it was easy to discover the simulation. But such imitative paroxysms no doubt exist, and may be explained without supposing a nervous contagion similar to that observed in the epidemics of tetanus which often attack an entire group of children. We know how susceptible children are to suggestion; frequently their instinct of imitation prompts them to mimic limping, *tics*, or convulsive movements. Paroxysms through imitation enter into this category; are produced by suggestion,

and may pass for paroxysms of whooping-cough. But the diagnosis of these imitative paroxysms is simple, since they develop amid special conditions. The expectoration is absent, and, moreover, on separating the children, the paroxysms rapidly disappear.

The diagnosis of whooping-cough being once established, it remains for the physician to determine its intensity and form, and to assure himself of the existence or absence of complications. The intensity of the whooping-cough may be measured by the number of paroxysms, which the parents should count and observe as they occur. The enumeration of the paroxysms is the only means of determining whether the disease grows worse, rests stationary, or improves. It will also enable the physician to gauge the effect of the treatment instituted.

We have sufficiently studied the complications, and need not return to the signs which characterize them and permit their diagnosis. We simply recall the fact that by following the course of the temperature, examining the thorax each day, and attentively observing the general state, we will be able to anticipate the *début* of most of the complications, and often also to prevent their development.

## PATHOLOGICAL ANATOMY.

Whooping-cough does not determine characteristic organic lesions. Autopsy of the subjects who succumb during the course of this disease, reveals the existence of numerous lesions, but they are of diverse order and may almost always be referred to the complications.

Whooping-cough has no specific lesion comparable to the pustule of smallpox, to the false membrane of diphtheria, to the erysipelatous patches of erysipelas, etc., etc. Accordingly, pathological anatomy must confine itself to a statement of the principal lesions discovered on autopsy, of which the greater number possess a secondary importance. Several, however, are sufficiently frequent to merit consideration as playing a capital rôle.

At all times observers have striven to set forth the lesions which they considered characteristic of whooping-cough. According as they were impressed by the nervous element or by the respiratory element, they sought in the nervous system or in the respiratory apparatus the alterations which might be considered specific.

The nervous system has been incriminated by Desruelles, Copland, Kilian, Breschet, etc.

Desruelles attributed whooping-cough to a lesion of the encephalon, and regarded the malady as a broncho-cephalitis.

Copland placed its seat in the medulla oblongata, which he claimed to have found congested or inflamed.

Sanders, admitting the rôle played by the medulla, deemed it possible to localize with greater precision the initial lesion, and attributed it to hyperæmia of the pneumogastric nerves at their origin.

Kilian likewise maintained that the pneumogastric nerve is constantly modified in whooping-cough. He claimed to have found it congested in fifteen autopsies; asserting also that with hyperæmia of the nerve he found associated inflammation and thickening of the neurilemma. The lesions were not localized at the origin of the nerve; they were extended to the entire trunk, and were particularly visible in the thoracic portion.

According to these authors, the essential lesions of whooping-cough consist in alterations of the nervous system, central or peripheral, and the slightly marked lesions are of a congestive nature.

In reality, the lesions of the nervous system are but slightly characteristic, and may be totally absent in a large number of autopsies. Furthermore, when they exist they are faintly marked. Finally, they have not been verified by microscopic examination, which would alone permit our ascribing to them their real importance.

The alleged nervous hyperæmia, without microscopical examination of the nerve, has lost all import-

ance in the actual state of science. In fact, we are aware that the color-changes which characterize the nerve on autopsy are more commonly the phenomena of putrefaction than the results of lesions existing during life.

A large number of authors, attributing a predominant importance to the catarrhal element of whooping-cough, have placed the characteristic lesions in the respiratory passages. According to Marcus, Watt, Gendrin, Niemeyer, Beau, the lesions of the respiratory passages are constant. They are said to occupy by preference the upper respiratory passages, particularly the larynx. The larynx, the trachea, and the bronchi present a considerable hyperæmia, accompanied with catarrh more or less intense. The catarrhal lesions are further claimed to reside in very different places. According to Beau, they are seated almost exclusively at the level of the *supralaryngeal* mucous membrane. The theory of this author may be summarized as follows: The supralaryngeal mucous membrane, invaded by phlegmasia, secretes a more or less abundant mucus, the quantity of which is in proportion to the intensity of the disease. This mucus, descending the walls of the vestibule in small pieces, falls on the glottis. Reaching the mucous membrane which lines the lips of this orifice, it determines the reflex action and the spasmodic contraction which constitute the paroxysm.

Catarrh of the upper respiratory passages fre-

quently exists, and has been observed by Parrot and other observers during life, on making laryngoscopic examination. It may be urged that it is not constant; moreover, it cannot be considered as the characteristic lesion of whooping-cough, for it is in no wise special and may be observed in other forms of laryngo-pharyngitis which are not accompanied with paroxysms.

Indeed, hyperæmia of the mucous membrane of the larynx, of the trachea, and of the bronchi is a common lesion in whooping-cough, and may be found whenever a concomitant bronchitis prevails.

Generally speaking, hyperæmia is frequent in whooping-cough and may be noted in most of the organs—in the head, the kidneys, the liver, and on the mucous membranes of the digestive tube. It is ordinary, and is at times accompanied with swelling of Peyer's patches. This lesion is related to the infectious nature of the malady, and is also the consequence of the impeded venous circulation.

The hyperæmia may proceed to vascular rupture and the production of hæmorrhage. According to Copland, Peyer's patches, at first hyperæmic, may suppurate and ulcerate.

Hyperæmia is the dominant lesion of whooping-cough, as of all infectious maladies.

With general hyperæmia we must associate, by reason of their frequency, a certain number of secondary lesions determined by complications. The

respiratory apparatus is most frequently invaded; bronchitis, emphysema, dilatation of the bronchi, are usual. In the ordinary forms, bronchial dilatation is acute and disappears with the disease. Sometimes, however, it persists, if due to broncho-pneumonia of chronic form.

Broncho-pneumonia is frequently noted and is usually of the spleno-pneumonic type, although all the forms may be observed.

Broncho-pneumonia is sometimes accompanied with pleural inflammation with or without effusion. Hypertrophy of the tracheo-bronchial ganglia is frequent even in whooping-coughs not complicated with broncho-pneumonia; it is so often observed that Guéneau de Mussy has considered it to be the cause of the paroxysms. The tracheo-bronchial adenopathy exhibits varying degrees, being at times mild and again extremely pronounced. Hypertrophy is usually simple. The ganglia are voluminous and red, their tissue being hard without trace of softening or relaxation. Sometimes the ganglia are soft and caseous, and the case then is one of whooping-cough complicated with tuberculosis.

The tracheo-bronchial adenopathy seems to be conditioned by the bronchitis, for it is absent in simple whooping-cough without inflammation of the tracheal and bronchial mucous membrane.

Apart from the respiratory apparatus, the organs present few important lesions. Most of the altera-



tions reported as observed in the liver, in the kidneys and in the other organs proceed from the hyperæmia, and appear to be the consequence of infectious lesions.

In recapitulation: When performing autopsy on a subject who has succumbed to whooping-cough, we commonly find hyperæmia of the laryngeal, tracheal and bronchial mucous membrane. The mucous membrane of the bronchi exhibits a deep red coloration. At times this membrane is thickened and œdematous, secreting an abundant mucus. The little bronchi are dilated and filled with muco-pus. The lungs are almost always augmented in volume, and are emphysematous, particularly at the level of the upper lobes. Most commonly they present the manifest lesions of broncho-pneumonia, with intense congestion more or less generalized. The tracheo-bronchial ganglia are usually augmented in volume. With these broncho-pulmonary lesions there exist signs of generalized venous congestion extending to the entire venous system and manifesting itself in all the organs.

Thus we see that these lesions are in no wise characteristic; they are simply what may be observed in any infectious malady. What is peculiar to whooping-cough is the frequency of broncho-pneumonia and hyperæmia of the respiratory passages. These lesions are not sufficient to form an anatomical diagnosis of whooping-cough.

## NATURE OF WHOOPING-COUGH.

Numerous theories have been formulated respecting the nature of whooping-cough. Their enumeration would be superfluous, for many of them possess only an historical interest. For convenience of study, they may be divided into three groups. According as the writers have been struck by the predominance of this or that element of whooping-cough, the latter has been treated as a neurosis, as a catarrhal affection, or as an infectious malady.

The theory which makes of whooping-cough a nervous affection relies for support on the spasmodic character of the paroxysms and on the nervous lesions which have been observed in various autopsies. To this theory it may be objected that the lesions of the nervous system are inconstant and often are even absent. Their seat and their nature are variable. The nervous phenomena are evident in the disease; but, whatever their importance, they should not cause us to neglect the other elements, and they fail to explain either the evolution, the absence of relapse, or the contagiousness which does not exist in simple neuroses.

The catarrhal element is the dominant feature of whooping-cough to those authors who would have us regard the disease as a simple inflammatory catarrh of the mucous membrane of the air-passages. The theory of Beau and of Gendrin is the most celebrated

of the inflammatory theories, which are open to the same objections as the nervous theory, since they explain neither the evolution nor the contagiousness. In comparing whooping-cough with a laryngeal bronchitis, we fail to explain why whooping-cough is accompanied with special characteristic paroxysms, whereas neither laryngitis nor common bronchitis is accompanied with a comparable spasmodic cough.

To obtain a satisfactory idea of the nature of whooping-cough, it is indispensable that we take into account, first of all, the capital fact that the malady is contagious. The contagiousness of the disease places it indubitably in the group of infectious maladies due to an animated virus capable of propagating itself and of living amid the conditions of the appropriated environment. Thanks to this characteristic, whooping-cough has a place by the side of measles, scarlatina, smallpox, typhoid fever, etc.—diseases to which it is further allied by the fact that the organism which has once been invaded by its virus is preserved from subsequent attack.

Whooping-cough, regarded as an infectious malady, is indeed lacking in specific symptoms analogous to the eruptions of the exanthematous fevers, and to the eruption of the lenticular rose-spots of typhoid fever. The sublingual ulcerations, erroneously considered by Delthil as special to whooping-cough, are in truth only a commonplace symptom without specific character.

The paroxysm is the truly special symptom of whooping-cough, and its importance is such that we may affirm that the virus of the disease becomes peculiarly localized in the respiratory passages, and brings its action to bear, through the intermediation of the mucous membrane of the air-passages, on the nervous ramifications of the bronchi and larynx. The virus, acting by a special process on the respiratory organs, produces functional disturbance which manifests itself clinically by the paroxysmic cough and by the bronchial hypersecretion.

The virus of the disease seems to exert a special action on the nerves, especially on the pneumogastric and its branches. This action is the source of the paroxysmic triad: the spasmodic cough, the bronchial hypersecretion, and the vomiting. The virus of whooping-cough, like that of herpes, brings its action to bear on the nerves. The special microbic infection of the broncho-pulmonary nervous system, the infection by a virus probably microbic, the localization of the action of this virus on the pneumogastric and its branches,—such seems to be the cause of the malady. The complications are superadded infections, with no characteristics special to whooping-cough; they attack the bronchi and lungs by preference, in consequence of the reduced resisting power of the pulmonary nervous system previously influenced by the virus.

Among the theories respecting the nature of

whooping-cough, that of Guéneau de Mussy merits special mention; he would have us regard the special symptoms of the disease as due to hypertrophy of the mediastinal ganglia. The paroxysm, he claims, is caused by irritation of the fibres of the pneumogastric, which are compressed by these ganglia. We may object to this theory on the ground that the hypertrophy is not uniform, and that it exists in other diseases which are not accompanied with the special symptoms of the paroxysm.

## TREATMENT OF WHOOPING-COUGH.

The best treatment of whooping-cough continues to be the symptomatic. There is no specific medication which will dispel the paroxysms or abridge with certainty the duration of the disease.

Medicine is not, however, impotent in the presence of whooping-cough. There are a number of therapeutic agents which enable us to act effectively on certain symptoms, and to influence favorably the course of the malady.

Before proceeding to study the general treatment of the disease and its different symptoms, a few words must be said about its prophylaxis. Whooping-cough being a preventable malady, we must needs know how to prevent it.

*Prophylaxis of Whooping-Cough.*—The contagion of whooping-cough may be imparted directly by a patient to a healthy subject, or indirectly through an intermediary.

The prophylactic rules relative to direct contagion are simple. Contact between the healthy and the diseased must be absolutely prevented—and it should be remembered that an extremely brief contact, sometimes for only a few minutes, suffices for the contagion. The duration of the contact is immaterial; a single paroxysm is sufficient. As it is almost certain that the germ is contained in the products of expectoration, the child must be watched during the

paroxysm, and must be made to expectorate into a receptacle containing a disinfectant liquid, such as strong carbolic acid or permanganate of potash in a solution of 2 to 1000.

*Indirect Contagion.*—Indirect contagion is that which occurs through intermediaries.

When whooping-cough attacks a child in a family, may the parents or children, free from the disease, visit other children living in a different house, without fear of bearing with them the germ of the malady? If a physician be treating a whooping-cough subject, must he fear that he will carry the germ to other children whom he is going to visit?

Indirect contagion, effected under these circumstances, is admitted by Joseph Franck and by Rosen. H. Roger cites one case, which I have reported, wherein the indirect contagion was certain.

Indirect contagion through healthy persons is possible, though exceptional. Hence it must be enjoined on those attending a young sufferer from whooping-cough, that they avoid seeing other children. The interdiction is especially necessary when there is a possibility of contaminating new-born infants, for whooping-cough transmitted to the latter is peculiarly dangerous by reason of the tender age of the patients. When dealing with children of the same family, whom the parents refuse to discontinue seeing, when a serious reason such as a disease attacking the separated children renders these visits

proper, it then becomes incumbent upon the parents to take those measures which the physician treating the whooping-cough sufferers should take himself. The clothing must be changed. The face, the hair, the hands must be washed with a sublimate solution of fifty centigrammes to the liter. As for the physician, it will be best for him to provide a garment in the domicile of the patient, which he may put on when approaching the latter and remove on the termination of the visit.

It is well to promenade as much as possible in the fresh air before visiting other whooping-cough patients.

Indirect contagion through the garments and bed clothing has not yet been positively demonstrated. It should, however, be regarded as possible, contagion through healthy persons rendering it very probable. It will therefore be necessary, after the termination of the whooping-cough and before permitting the return of the children who have escaped the disease to the family home, to disinfect the furniture, the clothing, and the apartment.

*Duration of the Contagion.*—Whooping-cough must be regarded as contagious so long as there are paroxysms. During convalescence, if the paroxysms still persist, even at long intervals, the contagion is yet to be feared, though we may admit that it is doubtful and even improbable if the paroxysms of the third period are no longer accompanied with expectoration.



The whooping-cough once cured, should a paroxysmic cough occur on the occasion of a subsequent bronchitis or other acute disease, the contagion is no more to be feared.

*General Treatment of Whooping-Cough.*—The ideal treatment of whooping-cough would destroy the infectious agent or prevent its germination in the economy, without causing the patient to suffer from its employment. This ideal treatment does not exist. There is no specific medication which can be directed with certitude of success against the infectious agent of whooping-cough. Numerous essays have been made along these lines, but up to the present time they have remained fruitless. A large number of therapeutic agents seemed for a certain time to encourage the tentatives of experimenters; but the success obtained proved ephemeral. Many medicaments have been proposed; the majority are now forgotten. Some, however, are capable of rendering real service, and merit enumeration.

Whooping-cough comprises three elements: the infectious, the catarrhal, and the spasmodic. Each of these elements should receive the attention of the physician, and each has its corresponding therapeutic indication.

*Therapy of the Infectious Element.*—*Quinine* has been frequently employed in the treatment of whooping-cough, being at times administered by mouth, and again in hypodermic injections. It has been vaunted

by Campbell and by Bing. Hubner has employed it with twelve children; in three of these the duration of the malady was abridged, and in five the paroxysms were rendered less violent.

The salts of quinine most commonly employed are the sulphate and tannate. The dose is generally one decigramme per year and one centigramme per month of patient's age.

Inasmuch as the ingestion of quinine fatigues the stomach, the hydrochlorate or valerianate may be administered with convenience in the form of a lavement. In adopting this mode of administration, the following may be prescribed:

Valerianate of quinine, in full doses, according to age.  
Infusion of valerian, 100 grammes.

That the lavement may be retained, the physician should see to it that a simple lavement of water is administered a quarter of an hour before the medicinal lavement in order to empty the intestine.

*Salicylate of soda* has been recommended by Hubner, who claims that it shortens the term of the disease and reduces the violence of the paroxysms. This medicament is given in the dose of 50 centigrammes up to one year of age, and of one gramme at from one to four years of age. Beyond this age the dose is two grammes. Salicylate of soda may be administered dissolved in a sweetened infusion of polygala or eucalyptus. Children will take it readily in currant or cherry syrup added to carbonated water.

*Resorcin*, recommended by Moncorvo (of Rio de Janeiro), has been employed by that author in the form of a solution of 1 to 100, painted on the orifice of the larynx.

*Carbolic acid* has been given internally by Sulking, Macdonald, and Simpson. Simpson, who treated 240 patients with this acid in the form of a potion, declares that not a single case terminated fatally. The carbolic acid has been especially employed externally—in powder applied with the pulverizer, or in solution in the form of spray.

*Thyme* has been recommended by Neavius in the form of an infusion (10 grammes of thyme to 70 grammes of water; daily dose, eight to twelve spoonfuls of this infusion). Neavius affirms that with this medicament the whooping-cough has always yielded ground within fifteen days and without the appearance of complications. This success is attributed by him to the antiseptic action of the thymol.

*Thymol* has been employed by Bouchut and by Poulet.

*Boric acid* has been recommended by Halloway, who prescribes the nasal insufflation of 15 centigrammes of boric acid every three hours. Twenty-four patients, two of whom had pneumonia, were treated and cured by this method, the duration of the disease varying from fourteen days to three weeks.

Insufflations of antiseptic powder were administered by Moizard and Cartaz and yielded remarkable

results, which were controlled and confirmed by d'Heilly. Most of the antiseptics have been employed in this form. Michael has employed iodoform, salicylic acid, and boric acid, mixed with inert powders. Fifty patients who applied for treatment from the third to the fourth month made the following showing:

Six cases, eight days of sickness;  
Eight cases, three days;  
Twenty-nine cases were ameliorated at the outset.  
Berriat treated ten cases of whooping-cough in this manner and obtained:

Two cures in fourteen days;  
Six cures in twenty days;  
One cure in twenty-four days;  
One cure in thirty-two days.

This medication appeared to Berriat to exert a manifest influence on the vomiting.

Guerder treated thirty cases of whooping-cough by nasal insufflations of a mixture of boric acid and roasted coffee, repeated twice a day. Seventeen children subjected to this treatment exclusively exhibited a rapid diminution of the paroxysms and were cured in a few days without any complication.

The following mixture is employed by Moizard:

Salicylate of bismuth	} ää. . . . 5 grammes.
Benzoin	
Sulphate of quinine. . . . .	1 gramme.

This mixture, pulverized and administered three

to five times a day, yielded excellent results to Moizard, producing a remarkable diminution of the paroxysms on the very day following the application of the treatment. At the same time the vomiting was completely arrested.

The application of the powder is readily made by means of a bulb-insufflator, or even with a glass tube. In these cases it is convenient to use the insufflator employed for iodoform dressings.

Insufflations made for the purpose of destroying the agent of whooping-cough, whose habitat is supposed to be the nose, may be advantageously accompanied with applications of antiseptic liquids to the mucous membrane of the pharynx and larynx. Inhalations of terebinthin or eucalyptol, and fumigations of sulphurous acid, fulfill this indication.

At various times physicians have resorted to inhalations of oxygen charged with eucalyptol, guaiacol, or creosote, with the apparatus employed for the treatment of pulmonary tuberculosis.

The antiseptic substances employed in inhalations often yield good results. They are inoffensive and rational. It was this idea which prompted the therapeutic experiments of Commenge, who proposed to treat whooping-cough by the emanations from gas factories.

*Treatment of the Catarrhal Element.*—In the treatment of whooping-cough by antiseptics the end in view is the conquest of the very cause of the dis-

ease. This (the only rational) treatment has not yet yielded completely satisfactory results. Moreover, the medication does not seem to influence the catarrhal and inflammatory element which is so often present. This catarrhal element should be combated, for it plays an important rôle in the development of the pulmonary complications which impart gravity to whooping-cough. To overcome this element, two means are at our command: the emetics, and medicaments which act on the bronchial secretions.

The emetics have long been employed in the treatment of whooping-cough. Intelligently administered they yield good results, and constitute so far the best treatment of the catarrhal element. It is not very probable that they modify the bronchial secretion; but it is certain that they favor mechanically the expulsion of the mucosities whose presence in the bronchi is one of the causes of the paroxysm. Given at frequent intervals, the emetics permit the mechanical evacuation of the bronchial mucus and diminish the number and intensity of the paroxysms. That the emetic treatment may be really efficacious, it must be prolonged and repeated. These agents should be given when the stomach is empty, that is, a certain time after meals, preferably in the morning.

It may be objected that if there be repeated vomiting, there is no rational ground for administering emetics. This is an error, for spontaneous vomiting expels the food and gastric mucus—rarely the bronchial mucosities.

The depressing emetics such as antimony and potassium tartrate, sulphate of copper and apomorphine, must be rejected in view of their depressing action.

The emetic universally recommended and adopted is ipecac; and this agent, in order to fulfill the therapeutic purpose in view, should be given in small doses in the form of the syrup. It should be administered preferably every day—or rather every two days—in the morning, on an empty stomach.

For children less than a year old, syrup of ipecac will suffice. These infants are to take two or three teaspoonfuls of the medicament. If necessary, the dose should be augmented until vomiting occurs once or twice.

For older children, up to two years of age, add to the syrup of ipecac one decigramme of powdered ipecac; two decigrammes and three decigrammes for children four years of age and more. In the case of the latter a tablespoonful of syrup of ipecac should replace the teaspoonful given to the younger children.

By reason of its expectorant properties, *white oxide of antimony* may be given in place of ipecac to children in whom gastric susceptibility is feared. By the method of Trousseau it is administered in a white lincture (*looch*), giving a tablespoonful every two hours. The formula of Trousseau was as follows:

White oxide of antimony.....	up to 1 gramme.
White lincture.....	150 grammes.

Or the oxide of antimony may be administered mixed with cherry-laurel water in the following formula:

White oxide of antimony.....	50 centigrammes.
Cherry-laurel water.....	1 gramme.
Infus'n of eucalyptus or hyssop.	100 grammes.
Syrup of tolu.....	50 grammes.

One tablespoonful every two hours.

Finally, the dose of 50 centigrammes may be divided into ten packets, and each packet given in a small cup of an infusion of polygala or peppermint.

While endeavoring with the aid of emetics to facilitate the expulsion of the mucous matter, the physician must seek to diminish the bronchial secretions with the aid of the medicaments reputed to possess this power. The agents which fulfill this indication are: terebinthin, eucalyptus, *Pinus canadensis*, etc.; any of these may be given to children with advantage in the form of syrup which serves to sweeten a hot infusion.

If the whooping-cough be accompanied with considerable hypersecretion, it will be proper to give creosote, guaiacol, or eucalyptol, two to three drops, as in the following formula:

Creosote.....	2 drops.
Water.....	90 grammes.
Orange-flower water.....	30 grammes.
Lemon essence.....	2 drops.

—Péchohier.



In view, however, of the acrid taste of these medicaments, it will be better to administer them in the form of inhalations.

*Treatment of the Spasmodic Element.*—The third indication to fulfill in the general treatment of whooping-cough consists in combating the spasmodic element, for the purpose of diminishing the number and intensity of the paroxysms.

*Belladonna* was long regarded as the medicament best responding to this indication. Its efficacy appeared so great to Hufeland that he regarded it as the specific of whooping-cough. Trousseau praised its effects and declared that they were almost infallible. Without fully conceding the truth of this opinion, we may consider belladonna as an excellent antispasmodic yielding good results in the treatment of whooping-cough. I have almost always employed it with success.

Belladonna may be administered in pills and in powder, or in the form of tincture, syrup or potion.

The dose of belladonna extract, given in pills, is one-half centigramme up to one year of age; one centigramme up to two years. Beyond this age, two centigrammes are given in two doses.

The following formula may be prescribed:

Belladonna extract.....	10 centigrammes.
Extract of valerian	} ää..... 2 grammes.
Oxide of zinc	

For twenty pills. One, two or three pills, according to age.

The powdered belladonna is administered alone or mixed with some infusion. The dose varies from three to eight centigrammes, according to age, as stated in one of the following formulas:

Powdered belladonna-root... 4 to 8 centigrammes.  
Sugar..... 25 centigrammes.

For one dose.

Extract of belladonna..... 5 to 20 centigrammes.  
Sugar..... 5 grammes.

For ten doses.

Administered in potion, belladonna extract may be prescribed with one of the following formulas:

Extract of belladonna..... 2 to 5 centigrammes.  
Cherry-laurel water..... 1 gramme.  
Camphor-water..... 10 grammes.

One spoonful every two hours.

Tincture of belladonna..... 1 gramme.  
Simple syrup..... 20 grammes.  
Lime-flowers water (*Aqua linden*) 130 grammes.

To be given in spoonfuls.

Extract of belladonna..... 5 centigrammes.  
Extract of conium..... 2 centigrammes.  
Pure tannin..... 30 centigrammes.  
Infusion of senna..... 60 grammes.  
Fennel-water..... 30 grammes.  
Syrup of althæa... 25 grammes.

Give a dessertspoonful every two hours.

—*Bouchut.*

Finally, syrup of belladonna is readily taken in the dose of five to ten grammes either alone or with any aromatic infusion.

Other medicaments may also favorably influence the paroxysms. Opium has been recommended and has yielded good results in the hands of several observers. Its employment does not, however, seem to merit recommendation, in view of the susceptibility of children to this medicament.

Chloral has been essayed repeatedly. According to Hubner, it is capable of abridging the paroxysms and rendering them less violent. This medicament may be prescribed in the form of a syrup containing one gramme of chloral, by the tablespoonful, to be taken several times during the twenty-four hours. Chloral, as I have seen it employed methodically by Joffroy at the Hospital for Sick Children, appears to reduce sensibly the number of the paroxysms. The graphic summaries of the number of paroxysms noted before and after the administration of the medicament show this in a striking manner. Unfortunately, chloral at times gives rise to an abundant eruption of livid red spots covering a large expanse of skin, which necessitates its suppression. Moreover, it is frequently ill-tolerated by the stomach.

Bromide of potassium seems to have no action on the paroxysms; such, at least, is the opinion of Hubner, who obtained no results.

Bromoform has been employed by Stepp. With a dose of three to six drops of this medicament, according to his claims, the vomiting ceases rapidly. The paroxysms become more rare and less intense. Cure follows in the course of two to four weeks.

Chloroform was introduced by H. Roger in the therapy of whooping-cough. This author gave from six to forty drops of it in a gummy potion, to be taken in twenty-four hours. He began with six drops, and added two drops every day. In patients who took the chloroform, the paroxysms of cough generally diminished a third at the end of eight days.

Chloroform has been further employed in inhalations. Schilling has treated sixty-two cases of whooping-cough by inhalations of chloroform and chloroform-water, and claims that he has had no failures.

The hydrochlorate of cocaine has been employed in diverse manners. Wintraub has administered it internally in a potion. Most commonly the medicament has been prescribed for external use. Forster, Emmet-Halt, Labric, Barbillion, and d'Heilly have met with success in its use.

Labric and Barbillion prescribe that the pharynx, larynx, and tonsils be painted with a solution of hydrochlorate of cocaine, 5-per-cent. These applications diminish the number of paroxysms and suppress the vomiting.

Prior recommends painting the larynx with a 15-per-cent. solution of cocaine.

Emmet-Halt employs cocaine in the form of inhalations. He causes the patient to inhale ten drops of a 6-per-cent. solution in chloroform, and claims to obtain an immediate arrest of the paroxysms.

D'Heilly paints the isthmus of the pharynx, and immediately after the laryngeal orifice, with a 5-per-cent. solution. He declares that these applications always diminish the intensity of the paroxysms, perhaps also their number, and suppress the vomiting.

Those who fear this form of application may follow the example of Forster and administer the remedy in the form of a cocaine spray.

The use of analgesin (antipyrin) has been recommended by Genser, Jeffrier, Sonnenberg, Griffiths, etc. This medicament, tested on a vast scale by Legroux and by Dubousquet-Laborderie, at the present time enjoys great favor in the treatment of whooping-cough. Dubousquet-Laborderie claims to have observed its success in nearly 200 out of a total of 300 cases of whooping-cough.

Those writers who laud the properties of analgesin state that, administered from the *début* of the spasmodic period in doses varying from fifty centigrammes to two grammes, this medicament rapidly diminishes the number of the paroxysms; it may even suspend them, at least momentarily, but they recur as soon as the medicament is discontinued.

I have several times had occasion to employ analgesin in the treatment of whooping-cough. The

doses were one gramme (six months to a year); two grammes up to three years; and three grammes beyond three years. Generally the results proved satisfactory. Analgesin diminished the number of paroxysms by a third in most cases. It never suppressed them completely. In two cases of grave whooping-cough with abundant vomiting, it had no effect on the latter.

My experience, comprising fifteen cases, shows that the action of analgesin is real but that it is not superior to that of belladonna. Analgesin, however, is to be recommended, for it is generally well tolerated and is entirely innocuous. It yields good results, but I do not consider it a specific medicament possessing all the value attributed to it by certain authors.

## TREATMENT OF SYMPTOMS.

### GENERAL HYGIENE.

In the preceding chapter we have had in view the general treatment of whooping-cough. We have reviewed the forms of medication whose purpose is to overcome the malady itself and abridge its duration. It remains for us to study the treatment of each period and of each symptom by itself.

During the first period the phenomena of catarrh alone exist; diagnosis is yet impossible, save under certain specially observed conditions. Since the children present only the symptoms of bronchitis, the sole treatment applicable is that of bronchial catarrh. The best treatment of any affection of this kind in children is the expectorant and emetic; and this method should be employed in the first period of whooping-cough. The children should be subjected to the use of emetics (ipecac) every day or every two days. Prescribe at the same time a cough potion intended to calm the irritation of the respiratory organs. This symptomatic treatment is the only rational mode, for we have no means of arresting the evolution of whooping-cough. Even in the first period the disease cannot be checked.

If, for special reasons, it is to be feared that the bronchitis is the *début* of this period of whooping-cough, we can then begin with advantage the anti-

septic treatment by insufflations and inhalations and give internally sulphate of quinine or, preferably, analgesin.

During the first period the child should keep his room, as in any bronchitis.

During the second period the paroxysms dominate the scene. We must then seek to prevent their increase in number, and to reduce their intensity, by the ordinary means of general treatment. Among these means, the insufflation of antiseptic powders, the application of solutions of cocaine, the administration of analgesin, belladonna, and sulphate of quinine, are indicated.

The special treatment of the paroxysm merits particular attention. First of all we must endeavor to remove the most common causes. For this purpose the child is to be guarded against the emotions to which his age is so susceptible. He must be forbidden from indulging in violent play; he must not pass suddenly from a heated room into the fresh air, etc., etc. The paroxysm once begun, the child instinctively assumes the positions which during the crisis facilitate the action of the accessory respiratory muscles. He should be assisted and furnished the necessary points of support.

The paroxysm is often prolonged by the difficulty which the children experience in expelling the glaucous matters of expectoration. It is then proper to relieve the mouth and throat of the mucosities with



which they are filled, by means of the finger or of a brush touched against the pharynx.

When the paroxysms are very severe it is well to have the patient inspire a little chloroform or ether. With one child, whose paroxysms were extremely violent, I found it very beneficial to administer in this fashion several drops of iodide of ethyl. The inhalation of ten drops diminished the intensity of the paroxysms.

Sometimes a simple mechanical excitation suffices to diminish the violence of the spasm. Kurt claims to have checked paroxysms of whooping-cough by exciting the mucous membrane of the conjunctiva or of the nasal fossa with a goose-feather, charged or not with a mixture of quinine and sugar or with analgesin. He attributes to a reflex action of the trigeminal on the pneumogastric this effect of the mechanical excitation of the ocular and nasal mucous membranes, producing an arrest of the spasmodic movements characterizing the paroxysm.

The vomitings which so often accompany the paroxysms, and which also occur independently of them, should be combated, by reason of the profound disturbance of nutrition which they may determine. When they are purely mechanical it is well to give the food immediately after the paroxysms, that it may be digested and absorbed ere another paroxysm can follow. The food should be very nourishing, small in volume, and should consist of meat broths (*purées*),

eggs, etc. Thin soups are to be proscribed; liquids are to be administered in small quantity. In a certain number of cases, powdered beef may be employed with advantage. Coffee, according to Guyot's claim, finds an indication against the vomitings. The food seems to be better tolerated when the repast is followed by drinking a small quantity of coffee (a dessertspoonful after each mouthful of food). In short, the repasts should be numerous, but small in quantity. The child should take frequently a few grammes of solid food, preferably after the paroxysm.

When the vomitings are very frequent and supervene after each meal, nutrition languishes, the child becomes enfeebled and wastes away.

Under such circumstances alimentation with the aid of the œsophagal tube often becomes a necessity. The sound should be employed with prudence, and gently introduced, for its introduction may provoke a paroxysm. In connection with the use of this method, we may likewise prescribe nutritive enemata containing eggs, powdered beef rendered alkaline, assimilable peptone, etc.

With all young infants refusing to take the breast, the physician is often obliged to have recourse to alimentation by the sound, to gavage, or to enemata of milk.

May the patients be permitted to go out during the second period of whooping-cough, or are they to keep their rooms? In serious cases of the disease

confinement to the room is absolutely essential. In cases of mean intensity, some physicians permit a promenade, under favorable conditions, that is, in the middle of the day when the temperature is favorable. In my judgment it is better not to authorize this change, for experience has demonstrated that it favors the complications of bronchitis and broncho-pneumonia. In several cases I have assured myself that the number of paroxysms was thus augmented. In mild cases, and when the paroxysms have shown a manifest and long-continued diminution, confinement to the room is less necessary; promenades may then be permitted on condition that they occur amid good conditions and that they be of short duration.

During the third period, there is no longer any question of treatment for the vanishing disease. The problem then becomes: how to combat the debility produced by the disease.

If the whooping-cough has been mild, if the child is vigorous and of sound constitution, a tonic and reparatory regimen is sufficient. In addition, care must be taken to prevent chills, which may produce bronchitis or provoke a relapse or reappearance of the paroxysms.

Before terminating the isolation of the patient and permitting the other children to return to the family home, it is indispensable to prescribe the necessary disinfection of the apartments and clothing.

If whooping-cough has attacked delicate children

predisposed to bronchial catarrh, or lymphatic or scrofulous children, convalescence should be attentively observed. The fortifying agents in all their forms (cod-liver oil, iodide of iron, etc.) are then indicated.

Occasionally the third period becomes abnormally prolonged. The acute period of whooping-cough is finished, but the paroxysms persist at rare intervals and will not finally vanish. At times the bronchial catarrh is even observed to persist, and this may render the whole situation dubious and result in the possible tardy supervention (though rarely) of a complication of broncho-pneumonia. In these forms of persisting whooping-cough, the arsenical preparations in the form of the granules of Dioscorides (one to two a day), of Fowler's solution (dose four to six drops daily), of solution of arsenate of soda (a half-teaspoonful of a solution of five centigrammes arsenate of soda in 200 to 300 grammes of water mixed with milk), frequently exhibit a real efficiency.

Where the persistence of whooping-cough is abnormal, change of residence has been frequently and very properly recommended. This empirical measure is often successful. The indications for a change of air should, however, be set forth with precision. Change of residence is useless and may even prove dangerous during the second period. During the third period it is useful provided it occurs in a rational manner. Thus, it is well to leave the city in

summer and take the whooping-cough convalescent into a healthy country district sheltered from winds and free from humidity. In winter a temperate climate is to be recommended.

Most authors counsel such change of residence, and cite cases of rapid cure occurring amid such conditions. It may accordingly be recommended to the parents, who will seldom fail to propose the measure themselves. The physician should only take heed of the climatic conditions prevailing in the place to which the child is to be sent.

So long as whooping-cough manifests itself by paroxysms, however rare, sojourn at the sea-coast should be forbidden. It is to be permitted only after several weeks have followed the disappearance of the malady.

If a change of domicile is impossible, aërotherapy may be recommended in the form of baths of compressed air. This measure often yields good results. It is useful in the anæmia which may follow the malady, and is, moreover, an excellent treatment for the emphysema which commonly accompanies and follows whooping-cough. This mode of treatment is easy of application with children.

*Treatment of the Complications.*—The most important and serious of the complications (broncho-pneumonia, tuberculosis in all its forms) are the secondary infections. Thanks to an intelligent prophylaxis and a rigorous hygiene, children may be

withdrawn from the danger of these to a certain extent. For this purpose isolation is the essential condition. Readily carried out in the city, isolation is generally efficacious, for the whooping-cough of the city is much more rarely complicated with broncho-pneumonia and tuberculosis than the disease in the hospital.

In the latter, isolation is necessary in the interest of the occupants free from whooping-cough, and of the whooping-cough sufferers who may thus more readily avoid tuberculosis and the superadded infections. Inasmuch as the isolation and treatment of whooping-cough subjects in the same ward does not exclude the danger of broncho-pneumonia, the ideal condition will be realized when each patient is treated in a separate room. This ideal is fulfilled with difficulty, but we may at least demand that every whooping-cough subject attacked with broncho-pneumonia or tuberculosis be immediately separated from the rest. If the principle of isolation be carried out, whooping-cough will be rarely complicated with broncho-pneumonia or tuberculosis, and the prognosis of the disease in the hospital will cease to be as grave as it now is.

Prophylaxis thus applied may supply the whooping-cough subject a shield against microbes coming from other diseases. But we must not forget that certain pathogenic microbes (the pathogenic streptococcus of broncho-pneumonia, for example) normally

inhabit the mouth. It is therefore well to destroy them or diminish their virulence by internal antiseptics, performed by lavage of the mouth with solutions of boric acid or naphthol, by nasal irrigations, or, better still, by the insufflation of antiseptic powder.

Once declared, the broncho-pneumonia of whooping-cough falls within the sphere of its ordinary treatment. The most beneficial general treatment continues to be the tonic treatment by alcohol in large doses. Locally, the congestive symptoms are to be combated by dry cupping and mustard plasters. If the broncho-pneumonia tends to present a localized focus, vesicatories of small dimension, readily renewable, and the conical cautery are indicated. The latter will be found particularly useful in chronic broncho-pneumonia, which is more frequent in whooping-cough than in any other malady.

In some cases, after apprising the parents of the gravity of the situation, we may essay the use of cold baths or the cold pack, repeated several times daily—applying a cloth moistened with cold water. This measure is justified by the intensity of the cutaneous congestion which it determines. It constitutes the most powerful revulsive at our command, and is at the same time an excellent sedative to the nervous system.

Two complications, unhappily almost always fatal, by reason of their immediate gravity demand prompt intervention. These are: the convulsions, and the glottic spasm.

The eclampsia is of extreme gravity, killing one whooping-cough sufferer out of every ten. To combat it we may employ the stupefying agents—inhalations of chloroform, bromide of potassium in the dose of fifty centigrammes to one gramme, chloral given in lavements. Warm baths are also to be prescribed.

Eclampsia is accompanied with hyperæmia of the brain, verified on autopsy, which justifies resort to bleeding. It may therefore be combated from the outset by leeches applied behind the ears and by wet cups to the neck. If the case is ominous, a general bleeding may be essayed, and would be justified by the gravity of the situation.

Though I have had no personal experience with this mode of treatment, I believe that when eclampsia supervenes, cold water applied in the form of repeated baths or of the moist pack will yield good service. The eclampsia being due to hyperæmia of the encephalon, cold water would be an efficacious means of inducing a flow of blood to the periphery.

In spasm of the glottis, the accidents commonly follow one another with such rapidity that death occurs before we have time to come to the child's rescue. Whenever, for any reason, this accident is to be dreaded, the persons attending the children should be apprised of what they are to do in case the accident occurs.

When spasm of the glottis is incomplete it is



sometimes terminated by causing the patient to respire several drops of acetic acid or ammonia-water, the attendant meanwhile performing energetic friction of the body with vinegar.

When the spasm is more intense and prolonged, it is well to perform artificial respiration by raising and lowering the arms and by exciting the region of the diaphragm in every possible way—by faradization if it be available. Mayor's hammer, applied to the thorax and abdomen, is an efficacious means, according to the experience of Du Castel, of regulating the respiratory movements.

Du Castel and his master Labric absolutely discourage the employment of the measure recommended by Rosen of Rosenstein, which consists in provoking vomiting by introducing the finger into the child's mouth.

When all means have proved futile, if death be imminent, tracheotomy is the supreme resource.







