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ANALYSIS OF A SERIES OF 330 CASES OF WHOOP-  
ING COUGH

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ANALYSIS OF 330 CASES OF PERTUSSIS  
TREATED IN THE GOLDSMITHS' WARD OF  
THE GREAT ORMOND STREET HOSPITAL  
FOR SICK CHILDREN BETWEEN JULY 1900  
AND MARCH 1903.

Before commencing the subject matter of this Thesis, I may explain that a Ward of 16 beds has been allotted in this Hospital for the treatment of Whooping Cough, with its more serious complications. It was first utilised for its present purpose in July 1900, and between that date and March 1903 330 odd cases have been treated. A unique opportunity has thus been presented to one who is holding a Resident appointment in this Hospital of closely studying the disease, of following out its course, and of observing its more frequent complications. Treatment both of the specific trouble and of symptoms has

been carried out in a most conscientious manner, a chart being kept which shows at a glance the effect of a particular drug or therapeutic remedy on the number or severity of the whooping and coughing spasms.

It may here be pointed out that as there are only two Hospitals in London into which cases of Whooping Cough are admitted as in patients, the cases which come under our observation are naturally very severe ones, and in the greater number of instances are at the time of their admission suffering from some complication, most commonly broncho-pneumonia, and therefore the mortality statistics of the ward do not represent the true mortality in Pertussis. A certain number of cases are relatively mild cases in which the whooping cough is simply, as it were, an accident in the course of some other disease. This has an important bearing on the frequency with which we

find complications, and also on the effect of treatment.

It would be advisable at this juncture, perhaps, to define Whooping Cough, and give a short summary of its main features.

Pertussis, then, is a specific malady which presents as its outstanding feature the following: A paroxysmal cough, which is immediately preceded by a deep inspiration, and succeeded by a series of short explosive coughs, culminating in a long-drawn-out inspiratory effort made through a narrow chink in the glottis, giving the peculiar sound which is called the "whoop", and which perhaps is the most characteristic phenomenon present, being practically diagnostic. These coughing and whooping spasms recur at varying intervals, and are usually relieved by the ejection of the stomach contents, or by the expectoration of mucus from the air passages.



Whooping Cough may be defined for all practical purposes as a disease of two stages. Firstly, there is that of catarrh and fever, which usually lasts about a week or ten days. This is a stage we rarely see in the Hospital, as the patients are only brought for treatment when the characteristic whoop has developed. We occasionally come across them when they are transferred from other wards. The patient as a rule has some slight pyrexia, and a somewhat hoarse dry cough, with a peculiar timbre, which has been termed "ringing". The child is usually pretty well during the day, with a fairly good appetite: in other cases there may be some fretfulness and loss of appetite. One gets nothing more definite in the shape of physical signs than a few catarrhal sounds in the lungs, on auscultation. As the catarrhal stage advances, the cough becomes more severe, noisy, and paroxysmal,

with nocturnal exacerbations, and the face puffy, with the eyes suffused.

The whoop in the majority of instances appears at the end of the second week, or somewhat later. The onset of a paroxysm while the child is lying in bed is usually very sudden, and feeling some embarrassment it quickly assumed the sitting posture, and then comes the series of expiratory puffs. If the child is up and about it often becomes restless for some seconds before, and has a very definite premonition of the coming storm, running to the mother or nurse for some support. The expiratory part is short, and is followed by a short whoop, to be quickly succeeded by a longer series of similar short expiratory efforts and a second and longer whoop, when the paroxysm may be over, or more may succeed until the child is fairly exhausted. The paroxysm ends with the ejection of a quantity of mucus and food, often streaked

with blood. The facial features after the paroxysms are well established assume a swollen or puffy character, with a considerable amount of duskiness. The eyes are likewise dusky and suffused, an appearance due to numerous minute ecchymoses, or to congestion of the smaller capillaries. The chest in a simple uncomplicated case at this stage has little to reveal beyond a few rhonchi, high or low pitched, or some medium or coarse râles. If the stethoscope be applied while the coughing spasm is on, one hears practically nothing. Vocal resonance is markedly diminished, due to the prolonged nature of the expiratory effort driving most of the air from the lungs, thus abolishing the conducting medium, and during the long-drawn inspiratory effort which marks the "whoop", the entrance of air is so slow and in such small amounts through the chink of the rima glottidis, that one has borne to one only the faintest of vesicular

murmurs. This is rather a peculiar feature at first sight, as one would have perhaps expected marked increase in vocal resonance, or even bronchophony from the boisterous nature of the paroxysms.

The spasmodic stage of whooping cough has no definite duration, and varies greatly in intensity, as a study of the charts in the ward will show. Some paroxysms are accompanied by a whoop, some are not. Very young children often do not whoop at all, but have severe fits of coughing which culminate in vomiting, and in them one must rely on facial physiognomy as an aid to one's diagnosis. Children, again, who are very ill with bronchopneumonia do not whoop at all, and again in the declining stage, quite a habit spasm is developed in this respect, for often, even months after its cessation, the characteristic cough returns again on the patient being affected with some slight catarrh.



As regards the whoop, it appears to be the natural consequence of the paroxysmal cough, and is facilitated to a large extent by the flexibility of the laryngeal cartilages in young life. The essential of the disease is the rapid series of expiratory coughs. Whooping cough is very liable to catarrhal and febrile relapses.

Now as to its infectious nature. That it is infectious is well-known, but from the fact that only the severe cases are admitted it is rare to have more than two children of one family in at the same time. It has only happened four or five times at the most that children from the same family have been in-patients at the same time in this ward. In 87 instances out of the 330 cases recorded were there other children affected with the complaint at home at the same time: in 55 out of the 84 cases there was one other child in the family affected or who had

just died from whooping cough with some lung complication prior to the admission of the patient into the Hospital: in 25 cases out of the 84 there were two members of a family affected in addition to the one in Hospital: in 5 cases there were 3 affected: in 1 case 4 children, and in 1 case 6 children affected, showing that in the over-crowded and badly-ventilated houses of the poorer classes there is a very infectious stage to this malady, that stage being probably the somewhat early catarrhal period. In the well-ventilated wards of a Hospital, on the other hand, where ample cubic space is provided per head, and even in the houses of the middle and upper classes, where an intelligent appreciation of the value of ventilation is seen, the infectivity is not quite so great, for we find in the case of Hospital wards that although cases are sent to us from the general wards of the Hospital, cases which have been in for

some time and have then developed undoubted whooping cough, yet we have not had more than one case ever sent from one ward. If the disease spreads in the way measles or diphtheria spreads, we should have had more cases sent in from one ward. Again, the only instances in which runs of cases have been admitted have been those from Convalescent Homes or bedridden children. Another point worthy of mention in this respect is that while our Nursing Staff is separate - that is to say, that the nurses do not nurse in any other ward - yet they have their meals in common with the nurses of the General Hospital, and associate with them in the ordinary way when off duty.. Now, in spite of this, we have had no case of whooping cough arising in the Hospital which we could trace to infection conveyed by anyone in the Hospital itself. On the other hand, it is an extremely common event for those nurses who apparently



have not had whooping cough in childhood to develop a modified form of Pertussis, which takes the form of violent and prolonged spasms of coughing which end either in actual vomiting or nausea without actually causing ejection of food or mucus.

I can speak of such attacks myself from personal experience. I will relate my own. I came on as House Physician to this Hospital at the beginning of December last, and took charge of the whooping cough ward as well as the out-patient whooping cough department. On an average I saw about 30 cases of Pertussis per diem, and these in a small stuffy room, ill-ventilated and somewhat dark. Now to prevent the whooping cough patients mixing with the other out-patients and thus infecting them, it was essential that they should remain in the same room after they were examined and the condition prescribed for. At the end of a very short time the air of the room was overladen with organic exhalations, and was a perfect



incubating temperature. After about six weeks of this experience I developed what I thought was an ordinary cough, which was somewhat hard at first, but soon loosened, and I expectorated a considerable amount of mucopurulent secretion, especially in the mornings. The cough during the day was more or less in abeyance, but at night time and early morning it was certainly more exaggerated. About a fortnight after the onset I noticed the cough was becoming more paroxysmal, and especially was brought on after eating or drinking rather rapidly, and perhaps more especially at night time after just getting into bed, and in the early morning. Morning after morning I was suddenly awakened with a tickling sensation in my pharynx and upper laryngeal area, and a series of paroxysmal coughs followed which either culminated in my vomiting or in coughing up some rather stringy viscid mucus, after which I was relieved, and was

able to sleep again. This lasted in all about 3 weeks, and I eventually got rid of it by going to the seaside and recruiting. The two House Physicians prior to me had somewhat the same experience, one of them developing something very closely resembling a whoop. Neither of them had had whooping cough before, as far as they could tell.

Period of Incubation is usually stated to be from 4 days to a fortnight, but it is undoubtedly a variable period, depending upon atmospheric and individual conditions which influence the germ cultivation. Seeing that the cases are only admitted when the characteristic whoop plus some serious complication has developed, it is somewhat difficult to get reliable statistics from the parents as to the period of onset of the cough, and the more or less exact interval which elapsed between the commencement of

the cough and the advent of the whoop. In some cases the characteristic paroxysms have been in evidence for weeks and months before one sees the patient.

However, in 40 of my cases I have got a definite interval of 7 days between the cough and the whoop, in 26 cases an interval of 14 days, in 10 one of 4 days, in 9 one of 2 - 3 days, in 12 one of 5-6 days, in 10 one of 9 - 10 days, in 8 one of 11 days, in 12 one of 21 - 24 days, in 18 one of 25 - 28 days. In 8 cases the cough and whoop commenced apparently simultaneously. In 30 cases there was no whoop at all, the cough being simply of a paroxysmal nature.

Longer periods than those above quoted are given by some mothers as being the difference in time noticed between the two associated phenomena, but in most cases one cannot accept such evidence, there being doubtless lack or neglect of proper observation. The usual period which elapses is from 7 to 10 days,

varying, however, within fairly moderate limits.

As to the probable period of incubation, I have no evidence of my own to offer, beyond that of my own case, where I developed a cough 1 month after being exposed to the infection in ward and out-patient department, the paroxysmal element being added about a fortnight later.

Age Incidence: 58 cases occurred in children under 1 year, the vast majority falling under the last two or three months of the 12 months. I have one case of 4 weeks, one of 6 weeks, one of 2 months, two of 11 weeks, 7 at  $3\frac{1}{2}$  months, 6 at  $4\frac{1}{2}$  months, and so on, the rest falling under the later months.

74	were	children	between	1	and	2	years	of	age.
61	"	"	"	2	and	3	"	"	"
51	"	"	"	3	and	4	"	"	"
43	"	"	"	4	and	5	"	"	"
24	"	"	"	5	and	6	"	"	"
8	"	"	"	6	and	7	"	"	"
4	"	"	"	7	and	8	"	"	"
3	"	"	"	8	and	9	"	"	"
1	was	a	child	between	9	and	10	"	"
and	1	"	"	"	11	and	12	"	"



Now the Hospital does not admit cases that are only a few weeks old unless some exceptional circumstance or complication arises, and this to a certain extent may explain the comparatively small figures I give for the first few months of life. Again, cases of over 12 years of age are not admitted either, but the liability to the affection is much less marked at this age. It is preeminently a specific affection of the early years of life, chiefly in the first four or five years of life, and it is perhaps rare for children who have been exposed to the infection in early childhood to escape and to get it at or about the end of the first decennium.

Sex: With regard to the Sex Incidence, there is no marked difference, my figures being 156 males, 174 females. There is therefore no special liability noted in the case of either sex.

Aetiology & Pathology: It is a disease which is perhaps more

frequent during the Spring than the other months.

This fact one does not set forth from the record of admissions into the ward, which is practically full all the year round, but from the out-patient figures.

During the winter months prior to Christmas of last year, I saw on an average about 12 to 20 cases daily, but after Christmas the number almost suddenly rose to 30 or more per diem, and these figures one is able to corroborate from a colleague at the Paddington Green Hospital for Children, who before Christmas of this year saw about 120 cases weekly, but since Christmas sees about 200 cases weekly.

The excess of mortality during the winter months is only what one would expect. The disease shows all the characteristics of a germ dissemination, not only from the fact that it occurs in epidemics, but its onset, its whole course, conforms to the acute

specific maladies, and in addition there is the well-recognised fact that one attack confers absolute immunity. It is perhaps more to be likened to Diphtheria than to any of the other acute specific fevers, for at first the habitat of the organism is a localised one, being in all probability the pharynx and the upper air passages. It is here that the organism produces its toxine which later permeates the system, and thereby becoming a blood disease secondarily. The toxine, we may suppose, acts on the respiratory centre, causing an irritative lesion which ends in a nerve discharge, which in its turn produces the series of expiratory coughs. The organism and toxine act locally as well, causing a swelling of the bronchial mucous membrane with bronchial catarrh and bronchitis, also rendering the lung liable to be attacked by the specific organism of pneumonia. The bronchial glands are also affected directly, an acute adenitis being

the outcome.

Several attempts have been made in the Bacteriological Laboratory of this Hospital to isolate the specific organism, but without success. Swabs have been taken from the throat and from the upper air passages, cultures have been made repeatedly from the mucus coughed up from the bronchi and lungs at various stages of the disease, both at the onset and during the progress of a typical case, and in addition in those cases which come to the Post Mortem room, sections have been made of the swollen mucous membrane of the bronchi and larynx, but without anything definite being elicited. Organisms and cocci of all kinds have been isolated, and inoculations into animals carried out. The contagion is capable of transmission by clothes, and also in all probability is inspired from the infected air of rooms, as witness the liability in crowded homes and in schools where



children of susceptible ages are congregated.

Morbid Anatomy: The actual lesions which one can associate with the specific virus are not many. Some observers have spoken of erosions in the larynx, but I can only record 2 cases in which there was some erosion of the glotto-arytenoid folds. The margins of the lungs are emphysematous, and in addition one usually finds a great deal of compensatory emphysema here and there in both lungs. Broncho-pneumonia in the form of multiple gray or leaden-coloured patches, or in many cases having a granular appearance, is perhaps the most frequent morbid accompaniment. Lobar pneumonia is occasionally seen, but is very rare as compared with the lobular variety. The mucous membranes of the trachea and larynx are often infected and ecchymosed sometimes. The bronchial glands are in the majority of cases acutely swollen, and even inflamed. The sublingual ulcer is a very frequent accompaniment

and is more or less diagnostic. The bronchial tubes again contain a mucus or mucopurulent secretion, sometimes tinted or streaked with blood in the very acute or severe cases. The number of children that die of simple Pertussis is not great, so that it is a comparatively rare occurrence to see a necropsy on a case of Pertussis pure and simple. The cerebral lesions which have been described are very inconstant, oedema, ecchymoses in the brain substance, and larger meningeal haemorrhages have been recorded, but it has not been my good fortune to see any of these, although from hemiplegic symptoms seen in one or two cases one must conclude that some such lesion was present in the motor cortical area, or cutting off or pressing on the motor paths.

In chronic cases of Pertussis, one is confronted with the very melancholy fact that the tubercle bacillus has found a suitable habitat, and is producing its

own particular lesions in various parts of the body.

Such cases are for the most part hopeless.

In 52 necropsies done on the cases which died in the Hospital out of my series, 30 showed tubercle in one or other of the organs, in 25 instances there being Tuberculosis of one or other of the lungs, chiefly in the form of caseating patches, or of the smaller miliary type. Six of these lung cases had definite visible tubercle of the pleura as a complication:

25, miliary tubercle of liver, 17 miliary tubercle of spleen, 12 miliary tubercle of the kidneys, and in one there was a tubercular ulcer of the bladder. Six cases had tubercular meningitis, chiefly of the basal variety, with or without miliary tubercle in other organs, and in one there was very marked miliary tubercle in the longitudinal and Sylvian fissures.

The bronchial glands were caseating and obviously tubercular in 15 cases of those recorded, the

mesenteric glands in 9 instances. The omentum was only seen to be affected with tubercle in one of my cases, on the other hand there was tubercular peritonitis twice recorded, tubercular gastric ulcer in one instance, and tubercular lesions of the intestines in 9 cases, these being either of the miliary type, or the more advanced tubercular ulcer. Miliary tubercles were discovered in the heart in 4 cases, twice in the pericardium, and once on the left ventricle.

Choroid tubercle was diagnosed during life in two of the patients, and subsequently verified at Post Mortem examination. Now I have spoken of Tubercle as being one of the most frequent sequelae in the sub-acute and chronic cases, and have given certain figures as evidence of my statement, but we know in reality that it is much commoner than it appears from my figures, for in addition to those cases



which die, and in which we have diagnosed almost certain tubercle, such cases not being available for necropsy owing to the parents' objection to such, there are others who by the physical signs and the temperature chart, and perhaps an examination of the sputum or of some abscess from which pus has been taken, have appeared to us to have had some tubercular lesion which, under favourable hygiene and treatment, has yielded and become quiescent, and we are probably right in our supposition.

The broncho-pneumonic process we know to be the commonest complication of Pertussis, and the majority of Tubercular lesions in the lungs have followed on a simple bronchopneumonia, with the exception of those which constitute the miliary variety. I suppose the process of infection which one is able to trace in the post mortem room is as follows:- The bronchial glands being acutely inflamed, a condition which is

well recognised as being present in Pertussis, the tubercle bacillus enters the air passages, is carried by the lymphates to the bronchial glands - the one especially seen affected is that at the bifurcation of the trachea - finds a suitable nidus for its growth and propagation, and in due time the gland tissue is converted into a cheesy or caseous mass. The process, of course, may stop here, and everything become quiescent, but in the majority of instances it is only too well recognised that the gland becomes first adherent to one or other bronchus, and eventually ulcerates into it, discharging the infective tubercular material into the bronchi and bronchioles and air vesicles, thus we get the tubercular process established .

I dwell on this tubercular infection of our Pertussis cases as being the most fatal complication, and the most hopeless naturally.

To proceed. In 22 of the necropsy cases there was a simple non-caseating bronchopneumonia of one or other lungs or both, in 2 there was lobar pneumonia. Three only disclosed an acute pleurisy, while 14 had definite areas of atelectasis, either large or small, and in various situations. In one instance there was congenital atelectasis of the whole of the left lung. There was marked oedema of the lungs in 2 cases, acute capillary bronchitis in one case, and in most of the cases the bronchi and bronchioles were filled with mucopus. A pretracheal abscess with perforation of the anterior laryngeal wall is recorded in one instance. Markedly dilated right heart was the prominent feature in 4 of the cases, and in one of these we found the cerebral membranes infected and petechial haemorrhages in the cortex.

A rare phenomenon was the occurrence of thrombosis of the right mid-meningeal vein and superior longi-

tudinal sinus, and this was present in one case. I should have added to the morbid appearances met with in the lungs that bronchiectasis was a marked feature in 2 cases, and in one of these it is interesting to note that a Meckel's Diverticulum was present.

Three cases had distinct purulent meningitis of the pneumococcic variety, the brain showing pus over vertex and base, and one of these was complicated with an additional purulent peritonitis, the pus from the peritoneum disclosing Fraenkel's diplococcus. Pneumococcic arthritis of the right knee joint and right upper tibiofibular articulation was present in one case only. Acute endocarditis of the mitral valve was a feature of one case, in which there was no rheumatic history. Inguinal hernia was seen in one case. In addition there were, of course, numerous accidental morbid conditions, which do not come within the scope of such a subject as this.



Symptoms & Signs: As a rule the actual diagnosis does not

present much difficulty to us. First of all let us take the Cough. If the cough is of a paroxysmal nature and unattended by abnormal physical signs in chest or pharynx, in all probability such a case is one of Whooping Cough. If the cough is brought on by some emotional disturbance, or as the result of some mechanical stimulation of the back of throat, such as in examining with a spatula placed far back, or by food or drink coming in contact with the

pharynx, and if the cough that follows is paroxysmal, then we may with probable certainty diagnose whooping cough. If in doubt about the nature of the cough,

never omit to examine the throat, and to make the examination thorough, touching the back of the throat or fauces with the spatula before withdrawing it. This

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will in the majority of cases bring out the paroxysmal cough, and will often induce a whoop which has

hitherto been latent or unnoticed. It is usually averred that the cough of whooping cough is more frequent at night. This may be so in the case of children who are treated in their own homes, or who for the most part are out during the day, the parents probably not being near at hand to hear them cough and whoop. Again, it is at evening time that the child feels tired and out of sorts, and resents being washed and put to bed, and we know from experience that the stirring of the emotions, either those of anger or joy, is almost certain to initiate the spasm afresh. It is partly the mental and partly the physical upset, and also the action of cold during the undressing period, or shortly after being put to bed which, acting reflexly, makes the cough more frequent. In the ward, on the other hand, the paroxysms of coughing and whooping are more frequent during the day than at night time. The explanation of this is

to be found probably in the fact that if the children are well enough they are usually sitting up in bed, or are actually up and about the ward playing with one another, and in addition they are somewhat disturbed by being cleaned and fed, and having temperatures taken. It is therefore largely on this account that we get less frequent paroxysms between 8 p.m. and 8 a.m. than between 8 a.m. and 8 p.m., these being the times between which the number of whoops and coughs are charted. Another point to be noted with regard to the frequency of whoops and coughs is that while the number of individual whoops varies greatly - we have had as many as 300 or 400 in the 24 hours - yet even in these severe cases the number of paroxysms of coughing rarely exceeds twenty in 24 hours. In not one case have as many as thirty paroxysms of coughing been noted in the twelve hours. In one case in the ward it was noted that there were 24 whoops to a single paroxysm of coughing: now this

in the record of coughs and whoops would only count one of the former with 24 of the latter, yet the attacks in the case were so severe that the child was forced into the opisthotonos position during the attack.

Expectoration is not an act which is voluntarily performed by young children, but in whooping cough one finds that the paroxysm usually ends in the ejection of food and mucus, the latter being brought up from the air-passages. In the less severe spasms the sputum comes into the mouth and the child promptly swallows it. In the early stage the expectoration is usually thick and viscid, albeit being clear: later, if the bronchitis resolves, one gets a yellowish heavy sputum, and later in the chronic cases one finds a thick glairy muco-pus. The ejected material is often tinged with blood, this either coming from the stomach, from the lungs or larynx, from the pharynx, or



from cracked lips which are quite a common feature in the malady.

The Whoop gave the name to the disease, which was described

by Willés in 1667. Now it must be remembered that

we get cases of whooping cough without any whoop,

there being 30 of the cases which I have recorded in

which no whooping ever appeared, and on the other

hand we may get a whoop without any whooping cough.

One whoop does not mean whooping cough. In very

young infants it is rare to get a whoop, the paroxys-

mal nature of the cough being evidenced by the choki-

ness which accompanies the prolonged coughing, and

the turgidity or cyanosis of the face which mark the

prolonged expiratory effort. After six months of

age whooping is more common, and there have been

several very severe cases in infants under one year.

The whoop appears to be the natural outcome of the

paroxysmal cough. The nearest approach to it we

know of is perhaps the paroxysm induced by the entrance of food into the rima glottidis. Enlarged tonsils, bronchiectasis, pleural effusions, or empyemata, and enlarged bronchial glands, are often accompanied by an occasional whoop, which may obscure the diagnosis of the real disease. Laryngismus Stridulus, which is so constantly associated with rickets, is another phenomenon which is apt to deceive the unwary. There are certain differences between the sounds of laryngismus stridulus and those of whooping cough which it has been our good fortune to be able to compare in several cases which have had whooping cough plus acute rickets, and it has been interesting to note the change in character of the sounds produced during the prolonged inspiratory stridor. To guide us in diagnosing laryngismus from pertussis, we have not only the accompanying signs of rickets, such as facial irritability, tetany, and Trousseau's Sign,

but the history as to how the child was brought up. All that appears necessary for the production of the whoop is some want of coordination in the action of the laryngeal muscles, such as would cause a relative incapacity in the size of orifice leading to the chest cavity, which latter, having been depleted to an extraordinary degree of its air contents, needs to be filled with great rapidity. Von Herff, who has watched the larynx with a laryngoscope during a paroxysm of coughing, thought that a small flake of mucus secreted from the posterior wall of the larynx was the excitant of the spasm. In addition he found that the entire larynx and trachea was in a condition of catarrh, the greatest irritability being in the interarytenoid regions and the under part of the glottis. Others believe that the nasal mucous membrane rather than the lower respiratory tract is the seat of the irritation, and local applications have

been applied to this region, as in asthmatical conditions, cautery of enlarged turbinate bones, or rather of the hypertrophied mucous membrane surrounding them, and the snicking away of spurs from the nasal septum has been vaunted as being infallibly successful.

Others have again made the external auditory canal the seat of irritation in Pertussis, and have syringed the ears with antiseptic and analgesic lotions, but with what success I do not know.

Vomiting as an accompaniment of Pertussis was noted in 90 per cent of our cases! The act is usually initiated by a cough, and is the terminal phenomenon in a paroxysm. This symptom becomes an actual danger in the younger or more weakly children, as the amount of nourishment they retain is so small, with the result that marked wasting follows, which is extremely difficult to combat in unfavourable cases.



A combination of coughing and vomiting is very suggestive of whooping cough, but we must not be too hasty in diagnosing it on these grounds, for in cases of enlarged tonsils, or where there are enlarged bronchial glands, or in bronchiectasis we frequently get that combination.

The character of the vomit varies. It is usually food of a solid or fluid character, and this may be ejected at any period of the digestive stage, for it is well-known that the mere act of deglutition will initiate a coughing spasm, as will also an overloaded stomach. The stomach contents have usually a considerable amount of stringy mucus mixed up with them, and in most instances the air passages add their quota to the ejected gastric material. I have before noted that streaks of blood or even larger amounts may be seen in the vomit, and this may proceed from the stomach actually, and has probably only

occurred while the spasm was on, for I have never noticed ~~altered~~ blood of the coffee ground type, or it may proceed from the pharynx or pharyngeal adenoid tissue, or even from the nasopharynx, or lastly from cracked lips.

Sublingual Ulcer is a most suggestive sign of pertussis.

For the most part it is situated at the fraenum linguae: in a few instances I have noted it more anterior, either single or one on each side of the median raphe of the tongue, and in still other cases one may see single or double ulcers near the tip of the tongue, but distinctly on the under surface. It varies in size and in shape. It may be as much as  $\frac{3}{4}$ " long by  $\frac{1}{2}$ " broad: usually it is much smaller. In shape it may be transversely elliptical, lying right across the fraenum, or it may have its long axis vertical, e.g. all the length of the fraenum. Almost invariably one first sees it as a greyish

white slough, if one can succeed in getting the child to elevate its tongue properly. The multiple ones have also the same appearance. The ulcer is the result of mechanical injury: it used to be thought that the ulcer had an important bearing on the disease, as being one of the foci of disease or one of the original areas where the specific virus elaborated its toxine, but this has long ago been disproved. Now the ulcer is said to be only present in children who have cut their lower incisor teeth, but this is not absolutely correct, as it has been noticed in this Hospital in children who had no teeth at all. The explanation in these later cases is simply that we have the under-surface of the tongue and fraenum markedly stretched over the alveolar margin, and although there is no erupted tooth there we have sufficient stretching to cause laceration, and the constant movement prevents healing. That the ulcer is simply due to mechanical injury was very

evident in one or two of the cases I recorded. In them the two lower central incisors had an oblique axis outwards, and the corresponding ulcers had corresponding positions to the crowns of these two lower incisor teeth. One gets more laceration perhaps when the teeth are separated by an interval, as in addition to the crown acting one finds that the sharp corners come into play with marked effect. A sublingual ulcer is rarely painful, and it rarely bleeds, which is somewhat surprising seeing the amount of jarring in a severe paroxysm. With regard to the frequency with which we meet sublingual ulcers, it was present in 70 out of the 330 cases I have recorded: in 4 of these it was noted that there were two ulcers, but the frequency of multiple ones is greater and the figures I present are probably inaccurate owing to lack of observation. The sublingual ulcer is almost invariably a feature of those cases in which the paroxysm is abnormally severe and prolong-



ed, and since most of the cases admitted to the ward are complicated ones, the majority having broncho-pneumonia superadded, and as it is a well-known fact that the paroxysms become less severe with the onset of a pneumonic process, or the advent of a high temperature, then perhaps the ulcer is not so frequently present in our inpatients as it is in our outpatients.

The figures as to sex incidence are as follows:-

Females 41 - Males 29, thus showing a preponderance of cases in the female sex, but this is not generally borne out by the figures of other observers, so that there is no special susceptibility to the ulcer in the female child as compared with the male. As regards Age, I have none recorded between the very early months of life, but at the 10th. month I have 3 cases of sublingual ulcer. These children, it may be noted, had lower incisor teeth.

19	cases	occurred	in	children	between	1	and	2	years
19	"	"	"	"	"	"	2	and	3
7	"	"	"	"	"	"	3	and	4
12	"	"	"	"	"	"	4	and	5
7	"	"	"	"	"	"	5	and	6
2	"	"	"	"	"	"	6	and	7

and one case at the age of 11 years. It is therefore extremely rare in infants under one year of age, and also very rarely noted after the age of 5, but it must not be concluded from this that the bigger children do not get them, the point is that we do not see so many big children in the ward. From the above observations, then, the ulcers are most common during the first and second years of life, and have, roughly speaking, occurred in 21 per cent of the cases in the ward.

The ulcer is not absolutely pathognomonic of pertussis, as it has been noted by observers to have been present in a case of enlarged bronchial glands, with paroxysmal coughing, and also in a pure case of broncho-pneumonia.

Subconjunctival Haemorrhage is one of the rarer phenomena.

It is not pathognomic either, but extremely suggestive. Out of the 330 cases it was present in 8, 4 being males and 4 females. It was noted as early as the 4th.month in one case, the 6th.month in another and the 10th.month in yet another.

							years
In 1 case it occurred between the ages of	2	and	3				
" 1 " " " " " " "	3	and	4				
" 1 " " " " " "	4	and	5				
" 1 " " " " " "	5	and	6				
" 1 " " " " " "	8	and	9				

In 5 of the cases it was present in both eyes, and in the other three it was noted in the left eye.

The child of 8 years of age had the haemorrhage first in the right eye, and a week later it appeared in the left.

In the child of two years of age there was very marked ecchymosis of the skin of both eyelids as well, the date of onset being 6 days prior to admission into Hospital, when there had been several convulsive seizures as well, which marked the case as a very

serious one. The ecchymosis first appeared in the left upper eyelid, then later in the day in the right upper eyelid, next day there was a subconjunctival haemorrhage in the left eye, and two days later a subconjunctival haemorrhage in the right eye.

The child of 3 years of age had a subconjunctival haemorrhage in the right eye 2 days before admission with bleeding from the right ear on the same day. A small one appeared in <sup>the</sup> left eye 1 day later, which disappeared in 4 days, only to reappear on the site of the old one 2 ~~or~~ 3 days later. This same case had severe haemoptysis, losing about a drachm of bright blood every time almost the cough came on: this lasted for nearly 30 days, the loss being less towards the end of the case.

Three of the cases had sublingual ulcers as well: and another had both epistaxis and haemoptysis, and still another had albumen in the urine.



One point of interest is the position of the subconjunctival haemorrhages when extensive. They usually occupy the equator of the globe of the eyeball, but this equator is not a horizontal one. Dr. Voelcker thinks that the reason of this is that the subconjunctival haemorrhages occur in the parts that are the least supported, and the part which is least supported is naturally the part between the margins of the lids. From this we might expect that the haemorrhages would be along the horizontal equator, but if we remember that in the act of coughing the eyes are turned upwards and outwards, I think we shall see we have an explanation of the fact that the equator will then come to lie obliquely, because the palpebral fissure remains horizontal. When the eye at rest is open, that is to say the eyelids are opened, this equator will be seen to lie not horizontally but obliquely. Another point to remember is that the

haemorrhages are more extensive anteriorly than posteriorly, forming a contrast to the distribution of haemorrhage which we get when we have lesions due to fracture of the base of the skull. The blood effused is bright arterial, and remains so for the most part until absorbed, which took 16 days in 2 of the cases, and from 17 - 20 days in one other.

Haemoptysis, which is not a common feature in lung diseases in children, is nevertheless met with fairly frequently in pertussis, although doubtless it is difficult in many instances to diagnose the origin of the haemorrhage, as bleeding from the stomach, pharynx, larynx, lungs, and nasopharynx, come out of the same orifice, e.g., the mouth.

It has been noted definitely in 23 of my series of cases, in 4 of which there were sublingual ulcers, in 4 epistaxis, and in 2 subconjunctival haemorrhage in

addition, showing that it is in the more severe cases that the bleeding occurs. The blood ejected is bright arterial blood, and must come from the congested vessels of the mucous membrane lining which have been ~~turged~~ and full during the prolonged spasm which has thrown such a strain on the right heart. The haemorrhage, it should be remarked, is arterial or capillary haemorrhage, since it soon ceases after the paroxysm is over, the sputum evacuated after the next few spasms being slightly tinted or tinged with blood as a rule. On the other hand we had a fatal case of haemoptysis in the ward, but this occurred during a paroxysm in a child who had marked pulmonary tuberculosis, the lungs having been extensively destroyed by the disease.

Epistaxis as an occasional sign was recorded in 20 of the cases, and the majority of these on examination were found to have considerable bulk of adenoid tissue in

the nasopharynx. Its frequency in the severer cases is only what one would expect. The bleeding may be anything from the tinging of the nasal mucus with streaks, to the loss of several drachms. It is rare to note the latter. A fallacy to be guarded against is the picking of the nasal orifices, which is a feature of some cases, especially those which eventually develop tubercular signs.

Albuminuria is occasionally present during the paroxysmal stage, and is perhaps to be explained on the ground of the venous congestion which is general at this period of the malady. Albumin in the urine was found on testing in 24 cases of the whole series, but it is probable that it was present in a much larger proportion, and if systematic testing for it had only been carried out we should have been able to record higher figures. Some observers go so far as to say that it is to be found in all cases at one time or



another during the disease.

Haematuria was a marked feature of two cases, and haematuria and pus in another, the latter being an accidental complication from a superadded morbid condition.

A point which is worthy of notice is the influence of Temperature on the frequency and severity of the whoop. In a large number of cases when the temperature rises above say  $100^{\circ}$  F the frequency of the whoop falls, and this is specially the case when the temperature remains at or about the level of  $103^{\circ}$  or  $104^{\circ}$  F., and vice versa when the temperature falls to a point at or about normal the whooping increases in frequency, the increase being noted from the time the temperature shows a continued and decided downward grade. Such a phenomenon is well traced, in cases which show a gradual lysis, although it occurs in cases of rapid crisis as well, and in those cases of fluctuating temperature with evening rises and morn-

ing remissions. It is also an interesting fact that with the advent of acute inflammatory mischief, attacks of broncho-pneumonia or lobar pneumonia, or the superaddition of a tubercular infection, there is an apparent improvement in the frequency of the whoops, and also in their severity.

Bronchial Catarrh of the larger bronchi and medium-sized

bronchioles is present to a greater or less extent in every case of pertussis, and is a point which I should have noted earlier. The auscultatory signs in the early stage of swelling of the mucous membrane with slight viscid catarrhal secretion, are deep and medium-toned rhonchi: later, when the secretion has become more abundant and less viscid, one hears râles and medium crepitations here and there. There is usually a rapid improvement in the auscultatory signs in such cases, if they remain uncomplicated, although as long as the paroxysms last one will get

an occasional rhonchus here and there. There is only a very slight initial pyrexia with such a catarrh, the temperature perhaps running up to 100° F, and an early subsidence to normal is the rule with perhaps occasional relapses.

Complications: Broncho-pneumonia, as being by far the commonest, I have put first. It is the complication which calls for indoor treatment in the ward, and it is for this that most of our cases are admitted.. It is met with in every variety as regards its degree and the position which the disease occupies in the lungs. As a rule it is somewhat widespread. Out of the 330 cases admitted we had 218 in which broncho-pneumonia or lobar pneumonia was, in our opinion, present, or roughly 66 per cent of the cases had that complication. Of course the ordinary incidence of the pneumonic process in pertussis is far

below the above figure, and I merely quote it to show how largely it figures as a serious complication.

The process as before mentioned is usually widespread, there being definite patches in one or several of the lobes of both lungs at the same time, and I give below figures that are intended to demonstrate the frequency of incidence in either lobe of either lung, always remembering, of course, that more than one lobe and probably both lungs are affected at one and the same time.

<u>Right Lung:</u>	The upper lobe had one or more patches in 73 instances	
	" middle " " " " " " " 59 "	
	" lower " " " " " " " 87 "	
<u>Left Lung:</u>	" Upper " " " " " " " 71 "	
	" Lower " " " " " " " 111 "	

The interscapular region was affected in 11 instances.

A patchy diffuse bronchopneumonia was diagnosed in 9 instances; a lobar pneumonia could only be absolutely vouched for in 4 instances.

The bronchopneumonic process, then, preponderated



greatly over the lobar pneumonic process, as one well knows is the case in children when the specific pneumococcus attacks the lungs. Now the virus is apparently the same in both forms, for it has been found recently that in a typical bronchopneumonia which was investigated at St. Mary's Hospital, Fraenkel's diplococcus was isolated and cultivated from the spleen, showing it is just as much a blood disease as the lobar pneumonia, and is due to the same specific organism. In addition clumping experiments have been done, which demonstrated the fact that the serum from a bronchopneumonic patient clumped a pure culture of Fraenkel's diplococcus, and caused degenerative changes to take place in its structure, thus quite proving that both lobar and lobular pneumonia are caused by one and the same organisms. The spread of a pneumonia in the lung always reminds one of the spread of an erisipelatous process in the skin and

subcutaneous tissue, there being a definite advancing margin which infects as it goes. The analogy is certainly strangely similar.

How is broncho-pneumonia to be definitely diagnosed, you will say, when some of the patches are so small?

It is of course absolutely impossible to do so in many instances: one can only conjecture, and even in several cases where one gets definite percussion and auscultatory signs, after some experience one almost prefers to diagnose such cases from the facial appearance, from the rapidity and distress in breathing, and from the temperature chart, together with the cough which is often characteristic. It may be folly to do so, but after daily observation of broncho-pneumonic patients it is surprising how adept one becomes at diagnosing from certain supplementary signs and symptoms which are often only obvious to oneself and are scarcely definable. It must not, however,

be imagined that a chest examination is not made. The chest is always gone through daily as a routine practice, and our diagnosis is simply confirmed on scientific grounds from an accurate analysis of the signs conveyed to one's ear through percussion or auscultation, not forgetting of course that sense of touch figures largely in our percussion examination, and that the trained eye takes in much before the other senses are brought to bear on the case. The broncho-pneumonic patch may then give one a definitely impaired note on percussion or it may not, often not. There may be a considerable amount of lung tissue between it and the chest wall which admits air freely, or be in an emphysematous condition - compensatory emphysema - thus giving one a hyper-resonant note rather. On the other hand the patch may be so small as to be undiagnosable by percussion, and perhaps one may definitely say that in the majority of

instances the note is only comparatively impaired.

On inspecting the chest there will be no marked difference in conformation, but the type of respiration and a deficiency of movement locally will aid one in forming an opinion as to what is going on in the lungs.

The auscultatory signs must be our sheet-anchor when in doubt, and they will vary greatly within wide limits. One may hear very definite bronchial or tubular breathing over the patches, or one may get merely a whiffing respiratory note, or again merely a harsh note with prolongation of expiration, and over those patches which are situated deeply in the lung with emphysematous or almost normal lung tissue over them, you merely get a harsh vesicular note. The accompaniments in a typical case are either fine sticky crepitations heard at the end of inspiration, and especially of a deep inspiration, or one may hear double rhytm crepitations during both inspiration and expiration. These accompaniments may be near or dis-



tant, depending on the fact as to whether the patch is superficially or deeply situated; or if the lung superimposed over a patch be collapsed, the sounds will be very distinct and near. One is rarely able to diagnose anything like the number of patches that do actually occur in a given case, for the patches at first discrete, later approach one another so closely that one hears the same signs at every point practically over both lungs. The most frequent combination of areas for the bronchopneumonia to be present in was the right upper lobe and the left lower lobe, or slightly less frequently both bases posteriorly. The root of the lung is again said to be a favourite spot for bronchopneumonia, though I was not able to verify it in a large number of my cases. The right middle lobe, which gives signs when affected in the region of the right nipple and apex of right axilla, is very prone to infection with the pneumococcus, and unfortunately one finds it is very slow in repairing.

It is very apt to pass into a solid, dense condition, and to be of a somewhat leaden colour, indicating extensive collapse, and on section such a lobe is found often to be studded over with patches of caseous pneumonia, each patch having a dilated bronchial tube in the centre full of muco-pus, or even softening into a cavity. The prognosis, therefore, in a pneumonia of this region must be guarded, as it often leads to a fatal termination.

The right apical pneumonia is also in some peculiar way apt to be more severe than that of the left apex, and a guarded prognosis is again necessary.

It is important to remember that an apparently severe pneumonia, with somewhat extensive consolidation, though localised, will often clear up suddenly in the course of four or five days. Such cases of comparatively extensive localised consolidation are far more favourable than the widespread diffuse variety.

I have already indicated the effect that a bronchopneumonia either mild or severe has on the frequency and severity of the whooping and coughing spasms.

Bronchopneumonia is extremely liable to become chronic in pertussis, and it behoves one to be suspicious of Tubercle superadded in the "hang-fire" cases, although on the other hand one must not be too hasty in diagnosing the latter, as the chronic cases in a most inexplicable manner all of a sudden clear up quite rapidly, and the child gets perfectly well. The signs by which one diagnoses a tubercular lung infection are supposed to be many, but few of them are absolute. Of course if the sputum test be positive, that settles it, but it is notorious that young children rarely expectorate. Again, signs of a cavity are strongly in favour of a rapidly breaking down tubercular focus, although one may get a simple caseous pneumonic patch doing the same. The temperature

is a valuable aid, only if it shows the typical fluctuation that is somewhat characteristic, e.g. the evening rise to  $101^{\circ}$  F., to  $102^{\circ}$  F, or  $103^{\circ}$  F, or even  $104^{\circ}$  F, with a morning remission to normal or subnormal, such a temperature, if it persists, is extremely suggestive. Another sign is emaciation, with a peculiar dry, harsh skin. Children with the tubercular process do not have the night sweats which are so marked a feature of adult tubercle. The lung signs vary very considerably. One case may show marked general impairment to percussion, with numerous fine to medium crepitations which are general. Another case may show only a comparatively impaired note with numerous medium or fine crepitations, which are usually of a clicking character. Again, a third may be even hyper-resonant to percussion, and one may hear in such just a few distant fine or medium clicking crepitations. Such a chest is known as the "silent chest", and often indicates a tubercular process



present. The explanation of such a phenomenon is probably due to the extensive emphysema which is present, and which is termed "compensatory".

I have notes of ten cases which showed some such signs as the above, and which subsequently turned out to be General Tubercle. Of course there were many others which we suspected, and which we were unable to trace owing to the parents having taken them home, or owing to an inability to obtain a necropsy.

The temperature in a bronchopneumonia complicating pertussis does not differ from that of bronchopneumonia pure and simple, and one should regard a lysis as being more favourable than a crisis, for the latter often only turns out to be a pseudo-crisis, the temperature shooting up again and perhaps taking some days to settle eventually, even in some cases remaining up for a week or two; in such one must be on the guard for an Empyema.

Bronchopneumonia is extremely fatal in children under 2 years of age - the mortality figures will be given later.

Bronchitis: Now a certain amount of catarrh is common to all cases of pertussis, but there was definite bronchitis of a mild or severe nature in 209 cases, most of the cases of pneumonia being complicated by it. The signs present were those usual in such an inflammatory condition of the bronchial tubes, e.g. rhonchi deep and medium toned, with a few sibilant ones, to be followed later by coarse and medium râles. The sputum at first viscid and clear, later becomes mucopurulent when the bronchial epithelium is cast off, with degenerated leucocytes. Bronchitis in a few instances led to acute oedema of the lungs, with a fatal termination. Most of the cases noticed were in rickety children with deformed chests, in which

the lungs had not sufficient room for full play.

Emetics and oxygen relieved for a time only. A

point which helps one to diagnose between a bronchitis

with much secretion in the tubes and a bronchopneu-

monia, is the following:-- In such a bronchitis one

finds the patient more cyanosed as to the face, and

the breathing more distressed than in a case of

moderately mild bronchopneumonia, the colour of face

in the latter being rather of the pale kind, with

perhaps a faint tinge of cyanosis on the cheeks.

Again, in the bronchitis it will be found that the

right heart is more distressed, being often consid-

erably out to the right of the sternum in the 3rd.

and 4th.spaces. It is in such cases that leeches do

a marvellous amount of good, and often tide the pa-

tient over the severe part of the attack.

The temperature chart in bronchitis rarely shows a

rise over  $101^{\circ}$  F or  $102^{\circ}$  F, and this ought to a

certain extent to help us in our diagnosis. Bronchitis is for the most part recovered from, and does not cause us the anxiety that bronchopneumonia does. Capillary bronchitis occurred in 4 cases, and fibrinous in one case. The symptoms were severe, and closely resembled a diffuse patchy bronchopneumonia.

Collapse or Atelectasis is another important complication, as being in young children extensive and frequently causing death, also important from the fact that it predisposes to bronchopneumonia, emphysema, and bronchiectasis. The collapse is brought about in the following manner:- There being a considerable amount of bronchitis with secretion in the bronchial tubes present in some cases, and knowing as we do that by the forcible expiratory efforts during the paroxysm most of the air is driven out of the pulmonary air vesicles, a bar is placed to its return by plugs of mucus secretion, which <sup>act</sup> as ball valves allowing the



air to be expelled, but preventing its return, thus the lung becomes collapsed in various parts. The collapse in its turn leads to inflammatory processes in the lung, with the additional fact that the tubes in that part become dilated. To crown all, some local pleurisy occurs, with subsequent adhesions, which tend to increase the bronchial dilatation. Thus as a result one finds a small laterally flattened chest, and the child perhaps permanently delicate. The diagnosis of collapse of the lung from pneumonic consolidation is very difficult, and one cannot state definitely any means by which one is to be told from the other: but the fact that they suddenly get well without showing those signs of resolution which the pneumonic cases show, and the fact that the dulness clears up quickly, indicates the nature of the case. In young children collapse of the lung is attended with a rise of temperature, usually  $102^{\circ}$  or  $103^{\circ}$  F,

but after the child has been in the ward for 24 or 48 hours the temperature falls, and the number of whoops is lessened. One must be on one's guard, and not diagnose pneumonia simply on account of the temperature and physical signs, for, as I say, collapse gives one identically the same signs. Perhaps one may say that in collapse the tubular breathing is often very intense, and also the snoring respiration produced in the larger bronchi conveyed with marked distinctness through such a patch. The sudden onset of a large area of apparent consolidation over which intense bronchial breathing is heard should make one suspect collapse, and the additional fact that the patient does not seem obviously ill, or at any rate as ill as one would expect from having such a large area of apparent consolidation. A collapse which does not clear makes one suspect tubercle, but a too-hasty diagnosis must be avoided.

I have 59 cases of collapse reported in my series, which were diagnosed during life, and some of which were verified at the necropsy: others were certainly such, owing to the rapid clearing-off of the impaired note areas without the resolution stage that is common to pneumonias.

The positions of the atelectasis and their frequency

I give below:-

In	9	cases	it	occurred	at	the	right	apex
"	5	"	"	"	"	"	in	the
							region	of
							the	right
							nipple.	
"	2	"	"	"	"	"	at	the
							anterior	edge
							of	the
							right	lung.
"	9	"	"	"	"	"	at	the
							right	base
							either	
							anteriorly	or
							posteriorly,	
							chiefly	the
							latter.	
"	1	"	"	"	"	"	in	the
							right	scapular
							region.	
"	11	"	"	"	"	"	at	the
							left	base.
"	5	"	"	"	"	"	around	left
							nipple.	
"	7	"	"	"	"	"	in	the
							left	upper
							lobe,	
							chiefly	anteriorly.
"	2	"	"	"	"	"	at	the
							anterior	border
							of	the
							left	lung.

Bronchiectasis is very apt to cause expectoration, and it

is usually found in those children who are over 4

years of age. I have collected 5 cases in which it

was a noticeable feature: in two of these the

position of the dilatation was at the left base posteriorly, just below the angle of the scapula, and this is undoubtedly one of the commonest situations for it. In diagnosing such a condition, besides the physical signs in the chest, which are chiefly of a bronchitic kind, one will be aided by the presence of the phenomenon known as "whispering pectoriloquy", which was noticed in two of the cases, also by clubbing of the fingers, and by the expectoration of thick, purulent, and possibly offensive pus. This occurs specially in the early morning, on the patient first getting up. One must not wait for the occurrence of offensive expectoration in a child before one diagnoses a condition of bronchiectasis, if the physical signs justify that diagnosis. Bronchiectasis, it should be remembered, occurs often in children who are the subjects of syphilis.



Bronchial Glands: Pertussis is undoubtedly a frequent

source of cheesy glands, with subsequent tuberculosis, not only of the lungs but also of the viscera. A point important to remember is this, that if after an attack of pertussis the patient remains thin and wasted for a long time, and the cough still preserves its paroxysmal character, then one should suspect bronchial glandular enlargement, and should overhaul the case to try and arrive at some such conclusion if possible. We were able positively to diagnose enlarged bronchial glands in 11 cases, such being verified at the necropsy subsequently.

The points one has to rely upon in addition to the wasting of the patient and the chronicity of the paroxysmal cough, are dulness on percussion over the manubrium sterni, and over the inner ends of the first two interspaces. This anteriorly. Posteriorly there will be a dull note in the interscapular fossa behind on both sides, or on one or other side. Again, a

sign which is not mentioned in text books, and which one has almost come to recognise as being almost diagnostic, is the marked sucking-in of the supra-sternal notch during inspiration, with a marked recession of the epigastric angle during the same act of inspiration.

A tubercular family history, with the additional fact that the child has had measles just prior to the pertussis, would undoubtedly make one strongly suspicious of the presence of enlarged glands. Although only diagnosed in 11 instances, their presence was an invariable certainty in all the cases infected with tubercle, as the necropsy afterwards disclosed, and certainly they were enlarged, if not caseous, in numerous other instances.

Empyema we found to be present in 5 of the cases as a post-pneumonic complication. In one of the children it occurred on both sides, commencing in the right lung

over the lower lobe, this being operated on. Not long afterwards a similar occurrence took place in the left chest over the left lower lobe. This was operated upon. The pneumococcus was found practically in a pure culture. One case terminated fatally. The physical signs will be practically identical with those seen in pleurisy, with effusion. The exploring needle will of course decide at once, and recourse should be had to such a procedure, as delay only means irremediable collapse of lung if left too long, with all its attendant dangers. A supplementary aid to differential diagnosis is also afforded us by the blood count.

In Empyema we get, or should get, a definite leucocytosis, and this has been our experience in several of the cases admitted into the hospital with such a condition.

An empyema situated between the lobes of the lung is

sometimes to be reckoned with, the diagnosis in such a case being practically impossible with any degree of certainty. It can only be suspected from the course of the temperature and some chronic signs in the lung which take long in clearing.

Pleurisy with effusion in 4 cases, without effusion in 5, must also be mentioned. There was no obvious difficulty in diagnosis, especially in the former cases when the exploring needle was used. The effusion cases eventually cleared up, and left the Hospital practically well, though a somewhat impaired note still remained from the adherent pleural condition. It is surprising how rapidly a serous effusion will be absorbed if only a drachm or two be removed in the exploring syringe. If left for 12 to 24 hours after exploration before the aspirator is used, it will be found impossible to obtain more than a few drachms



from a pleural cavity which certainly contained many ounces prior to the exploration.

Displacement of the heart calls for early aspiration.

Hydropneumothorax was diagnosed in 2 cases, in one instance following on the rupture of a bronchiectatic cavity in the left lower lobe posteriorly. A tymphanitic percussion note, with the characteristic "bell sound" were prominent features. The second case had the above signs over the right mid lobe, and in this case it followed on a caseating pneumonic cavity.

Pericarditis is recorded in one patient, there being very definite pleuro-pericardial friction. It is fortunately a rare event in whooping cough, and is probably due to an extension of the pleural inflammation when it occurs in the region of the pericardial sac.

Subcutaneous Emphysema, likewise termed "surgical", was noted in 4 cases, two of which terminated fatally.

One was a case of extensive tubercular disease in a child. The child had tubercle in most of the organs, some old intestinal tubercular ulcers, and a perforating ulcer of the stomach, which was also tubercular. Purulent peritonitis followed, which caused death.

In this patient the emphysema extended over the right chest front and back, also extending down to the umbilicus on the right side. A collar of subcutaneous emphysema was formed round the neck. On the next day the left front of the chest, the axilla, and left supraspinous fossa were the seats of the same process, and finally it spread over the lower jaw and cheeks. Per se the emphysema is not a dangerous complication, and rarely embarrasses the patient, but it usually takes some time to clear up, owing to the violent paroxysms in pertussis, which tend rather to keep the condition going.

Another case was in a girl between 2 and 3 years of

age, who had a dull percussion note, with intense tubular breathing for a long period over the left lower lobe. As it showed no sign of clearing, an exploratory needle was inserted into this area, with the result that the following day a surgical emphysema followed, which, however, limited itself to the left thorax front and back, stopping at the neck above and at the mid-line of the body both anteriorly and posteriorly. It cleared up completely in about 5 days, as did likewise the dull area in the lung, which was probably due to collapse. It is important to remember that the physical signs in a chest often change rapidly after a needle puncture, and therefore it is sometimes advisable to have recourse to its use as a therapeutic measure. A third case had the emphysema over the front of thorax, neck and cheeks. The diagnosis is perfectly simple, and could never be missed, the characteristic crackling sensation under

the skin is absolutely typical. Pertussis being a condition in which an abnormal strain is thrown on the air vesicles, it is perhaps surprising that the complication is not more common.

Acute Emphysema, or marked distension of the lung was present in sufficient degree to constitute a marked feature in 6 cases, although as a temporary condition in a slighter form it is present in a very large number. Its diagnosis from the hyper-resonant note and the harsh vesicular breathing with prolonged expiration should be easy. In addition there is cyanosis from the strain which is thrown on the right heart due to the diminished capillary area in the lung from the marked over-distension of the air vesicles, with subsequent atrophy of the capillaries in the walls of the same. The chest will likewise be somewhat barrel-shaped if it has persisted for some



time, and the apices of the lungs will be prominent in the supraclavicular fossae, especially when the patient coughs. The veins in the neck, both the superficial and the jugulars, become engorged to a marked degree during the coughing paroxysm.

Dyspnoea to be quite an alarming symptom was present in 14 cases. In some it was merely a temporary phase occurring after the paroxysm of coughing, and probably due to dilatation of the right heart. In others it was obviously due to the extensive pneumonia or widespread bronchitis.

It is well to remember that the coughing may produce an asphyxiated condition, with temporary loss of consciousness. This happened in one case.

Cessation of Respiration occurred in two of the cases, and artificial respiration had to be employed to resuscitate. There was no question of food being sucked into the larynx here during a coughing spasm, although

that did actually happen in one case, and laryngo-tracheotomy was performed, the child's life being saved in that way. The cessation of respiration may have been due to a temporary hypercongestion of the respiratory centre, or through the onset of a convulsive seizure, which certainly occasioned it in one case.

Laryngismus Stridulus was present, and rather tended to confuse the diagnosis in 6 cases, but as all of the patients were markedly rickety, and had other confirmatory signs of the rachitic process, such as tetany, marked facial irritability, Trousseau's Sign, enlargement of the epiphyses, and open fontanelle, with delayed dentition, and a history of a dietary which could only produce the condition, the phenomenon was easily explained. It was noticed that the laryngismus attacks had no definite relation to the whoop

they either preceded it or they followed it. They did not tend, in our opinion, to precipitate a whooping paroxysm. Another point to be mentioned is this, that in a chronic case the laryngismus seizures affect the child hardly at all, it calmly stridulates with no obvious inconvenience, whereas a whooping attack nearly always causes some discomfort.

A laryngeal stridor from acute laryngitis is likely to be confusing, and one such case is recorded in my series.

Cyanosis chiefly noticed in the face was a prominent feature of 47 cases, its persistence in the face being probably due to the dilatation of the venules, caused by the severity of the paroxysms, and the consequent facial venous congestion. Other cases must be explained by temporary embarrassment of right heart, as in bronchopneumonia and bronchitis. It must be re-

membered that the blueness and general puffiness of the face persists for some time after the attack of pertussis has passed off.

Oedema may also be mentioned in this connection, occurring as it does in a considerable number of the patients who display severe paroxysmal symptoms. The puffiness or oedema does not visibly or very perceptibly pit on pressure: the face is dusky, the eyes suffused, the lips swollen and often having a purplish hue, and not infrequently cracked. The urine, if examined, may or may not contain a trace of albumin, and the puffiness must not be mistaken for renal anasarca.

Enlargement of the Lateral Thoracic Gland, which is usually pathognomonic of some pleural condition, frequently Empyema or a tubercular process, was a feature of two cases. The gland probably received afferent lymphatics from the pleura, although injection experiments



with coloured fluids have failed to demonstrate the relationship. A fallacy to be guarded against in these cases is some skin infection or other, which will produce the same phenomenon. The enlarged gland in one instance was of the size of a large hazel-nut, and there was obviously a pleuritic lesion present in this case.

Tonsils & Adenoids in an exaggerated form complicated the issue in 25 cases, most of them being operated upon, much to the improvement of the coughing and whooping paroxysms, their presence perhaps exerting some reflex influence on the patient, and tending to keep going the paroxysmal cough, which one finds particularly in these patients tends to become chronic. The removal of tonsils and adenoids was the only therapeutic remedy adopted in many cases.

Otorrhoea, either in evidence before the attack of pertussis commenced, or developing during the onset or

during the run of the condition, was present in 22 cases. The discharge was very offensive, and most of the cases became chronic, in spite of vigorous syringing. In one instance there was very marked haemorrhage from the granulations in both ears during a severe paroxysmal period, this phenomenon lasting for a considerable time. Measles was a common precursor in a large majority of the cases. The condition did not appear to exert any influence on the severity or frequency of the whoops, an influence which might reflexly have become an adverse one in the case.

Diarrhoea or Diarrhoea and Vomiting was present in 34

cases so seriously as to be prejudicial to the chances of recovery. The phenomena mentioned were noticed chiefly in those of very tender age, namely, in the first few months of life, or up to the first and second years. The stools were for the most part green, and contained undigested food. The patients went

rapidly downhill, and in spite of vigorous treatment frequently succumbed. This gastro-intestinal catarrh which perhaps arises from the children swallowing the mucus from the air-passages which is infected with the specific virus, and is therefore itself a specific catarrh, is most intractable, and does not respond at all well to any of the well-known remedies, neither does rigid diet restriction help us much. Perhaps the most useful drugs are Carbolic Acid combined with Tr. Camph. O. Creosote and Castor Oil often does some good, as does Bismuth in very large doses. The child has the "hydrocephaloid facies" in the severer cases, when the drainage of fluid from the circulation into the bowel lumen is great. Glucose saline injected into the peritoneal cavity sometimes saves these cases.

Rachitis in various degrees of severity -- I am only recording the most marked cases -- was present in 60 of my

cases. These were the cases that almost without exception showed the severer symptoms, and were apt to get classed in the chronic set. Tubercle found such patients likewise an easy prey, owing to the poor lung expansion in chests that were deformed or distorted. A bronchial catarrh or bronchitis in such patients was very loth to clear up, and in time from other signs and symptoms it was obvious that another process - to wit the tubercular - was at work as well. These patients with the prominent sternum, lateral chest depressions, with the marked beading of the ribs, an acute epigastric angle, marked protuberant abdomen, and a dorsal rachitic curve of the spine, formed typical pictures. To improve chest expansion and to try and rectify the deformity while the bony wall was still pliable, Martin's rubber bandage was applied fairly firmly over the whole of the abdomen, in order to curtail the abdominal element, and en-



courage the costal element in respiration. Some cases showed definite improvement.

Convulsions I purposely refer to after introducing casually the subject of rachitis, for they were more frequently a complication of pertussis in the rachitic than in other patients, and this is not to be wondered at seeing the nervous irritability of the rickety children, as evidenced by such signs as tetany, laryngismus stridulus, facial irritability, and general irritability. The convulsions showed no definite distribution, being for the most part general. It was often noticed that the onset was in the form of facial twitchings, or of jerky movements in either arm only, to become general in a few seconds. The times of onset of the convulsive condition one might classify as follows:-

(1) at the onset of the pertussis attack, more especially when the rise of temperature and whooping first evidenced themselves.

(2) as a penultimate phenomenon in a case hopelessly ill with extensive bronchopneumonia, and with the temperature running up to 106° or 107° F. Convulsions also often closed the scene in such cases.

(3) On a fresh patch of bronchopneumonia developing.

(4) At any time in a case of rickets affected with pertussis, constipated bowels, teething, or some perfectly neutral catarrh being sufficient to bring about the seizure.

In addition to the above, one must mention that important class of cases which have meningitis as the outstanding complication, these being the patients which show a general infection with the tubercle bacillus. The meningitis, on the other hand, may be a pneumococcic infection, and of the purulent variety. In such patients convulsions are naturally to be expected, and they are frequent phenomena. Convulsions occurred at one period or another of pertussis in 38 cases, a considerable number of which

had a well-marked rachitic process present.

The mortality in the convulsion cases is high, being so often the mortality of the bronchopneumonic complication.

Opisthotonos not having a cerebral <sup>original</sup> origin, but solely and wholly due to the severity and length of the coughing and whooping paroxysm, was present in one case.

Left Hemiplegia was a late complication in one instance, and at the necropsy a massive tuberculide in the region of the basal ganglia was found.

Another case of paralysis occurred in a female child of  $1\frac{1}{2}$  years, who had whooped for 3 months prior to admission. Briefly, the chief points in the case are as follows:- 2 weeks after the onset of pertussis the child had a convulsive seizure, from which it recovered all right: three days later had a second convulsive attack, which affected the whole body, remained unconscious more or less for 3 or 4 days. At

the end of that time she was said to have recovered quite well. She now got measles and bronchopneumonia and a few days later there followed paralysis of right arm, with twitchings of left arm and right leg, followed by paralysis of the left leg. The doctor who saw her then diagnosed meningitis. She had a right otorrhoea, and there was a tubercular history on the mother's side. She was in hospital for 87 days, and was considerably relieved on discharge, though the paralysis still remained - a crossed paralysis.

Aphasia I must mention as having occurred in one case, the history being as follows:- A female child, 6 years of age, with an onset of pertussis and bronchopneumonia 2 months prior to admission. She whooped severely at first, chiefly at night-time, and there was some vomiting, the aphasia developing quite suddenly. The mother noticed one morning that she could not speak, but conveyed her wishes by signs.



She was quite healthy prior to this, with the exception of a fit at the age of 3 years, and measles 5 months before the onset of the pertussis. There was no family history of tubercle or syphilis~~is~~. She was in hospital for 158 days, and improved markedly, being able to phonate distinctly and repeat sentences after one: towards the end of her stay in the ward. The legs and arms, which at first were paretic, also improved gradually.

It would perhaps not be out of place at this juncture to briefly discuss the condition of the knee-jerks in pertussis. They were carefully gone into in practically every case which was admitted, and in the majority of instances they were found to be markedly sluggish, one being sometimes inclined to state them absent, but on carefully tapping the patellar tendon with the legs hanging loosely over the edge of the bed in such cases it was possible to get a faint response.

I imagine the phenomenon is explained on the ground of the pneumonic process present in a large number of the cases, as from frequent observation of acute pneumonia<sup>cases</sup> one has found the knee-jerk to be extremely sluggish. The plantar response was usually flexor, as is normally the case, except in young infants, who had the normal extensor response of the great toe.

Hernia in 5 cases inguinal and 1 umbilical came on after the onset of the pertussis, and was therefore directly due to the enormous strain thrown on to the abdominal wall during the severer paroxysms.

Prolapsus recti was a complication in 4 cases. The strain imposed on the abdominal and pelvic viscera combined with constipation was the determining factor in each case. The amount of prolapse varied: in one case there was 4 inches, and the mucous membrane was very much excoriated. The adoption of the horizontal or the sitting posture during the paroxysm, together

with strapping of the buttocks and attention to the bowel functions, soon rectified the condition.

Rectal Haemorrhage occurred in 2 cases, in one where no prolapsus at all existed. It was not alarming in amount.

Temporary Apical Cardiac Murmurs were heard in 3 cases immediately after the paroxysms. They were systolic in time, and disappeared very quickly after the paroxysm subsided. They are probably more often present if only looked for. They have no particular significance.

Acute Cardiac Dilatation was the cause of death in one case of pertussis.

Abscesses, ischiorectal in 5 cases, multiple in 3 instances, retropharyngeal in 1 case, and pretracheal in 1 case, the latter perforating the anterior wall of the larynx, and although intubation was performed, death resulted.

These abscesses, although not incidental to whooping cough, are the cause of rises of temperature which cannot be accounted for until the seat of such abscesses is determined.

Diphtheria was a complication in 10 instances.

Purpura occurred in 4 cases, and was usually a grave symptom, indicating a final phase of the case.

Herpes, either nasalis or zonal, occurred in 10 instances, there being a pneumonic process to account for it.

Hyperpyrexia was a feature of five cases, the temperature going up to 107° F. There was a fatal issue in each instance.

Enteric Fever was a serious complication to cope with in the case of one patient, but he eventually recovered completely. Ischiorectal and multiple abscesses developed in this patient, and delayed convalescence.

Tubercle as a complication or sequel caused a high mortality rate. One cannot state with anything like accu-



racy the number of cases in which it existed, but one may judge of the probabilities from the actuality as seen in the post mortem records. There were 30 necropsies as before mentioned under the heading of morbid anatomy, that showed definite tubercular lesions either of lungs, pleura, bronchial, and mesenteric glands, liver, spleen, kidneys, intestines, meninges, bladder, stomach, pericardium, etc., and there were numerous other cases in which it undoubtedly existed, but in which permission for a post mortem was withheld. Again, in others it had with almost certainty been present and had become quiescent, such patients going home and being lost sight of. Pertussis, like measles, renders the patients susceptible to the tubercular process, and perhaps the most frequent course of entry is through the bronchial glands which are just in a fit condition to receive the microbe, its gland structure being acutely swollen, and thereby affording an excellent culture medium for

Koch's bacillus. Its further progress has been traced and the probable sequence of events demonstrated.

Differential Diagnosis: There is usually very little difficulty when the whooping stage has been established. The cases most likely to be mistaken are those either of enlarged bronchial glands pressing on the trachea or main bronchi, cases of bronchiectasis, cases of pleural effusions, either serous or purulent, some cases of enlarged tonsils and adenoids, and sometimes a case of laryngismus stridulus. Careful physical examination is essential in order to arrive at a correct solution. I have mentioned, each under its own heading, the chief points in connection with the morbid conditions which are liable to confuse the issue, and recapitulation is superfluous. In the case of bronchial gland enlargement there is one phenomenon which occasionally aids diagnosis, and that is "the retraction murmur" heard over the upper part of the

sternum when the head is retracted. It is systolic in time. In addition, there is a history of wasting long before the cough developed, and there is often the association of lung disease.

I mention a foreign body in the air passages as occasionally causing a paroxysmal cough. Here there may be the history of the child having sucked something in suddenly, while playing with it in the mouth, and the history of an extremely sudden onset, a sporadic case, and not being one in an epidemic, which is the usual thing in pertussis, and lastly the Röntgen Rays may help us.

The catarrhal stage presents no signs or symptoms by which we may definitely diagnose whooping cough. We can only suspect it from the fact that an epidemic is raging in the district, or that there are other cases in the house or near vicinity.

Prognosis: Uncomplicated whooping cough is not a source of great anxiety, but age incidence has a special

importance, for we find that under 1 year of age the mortality is apt to be high. Everything depends on the nature and severity of the complications, and bronchopneumonia and severe bronchitis must be placed in the category of complications that seriously endanger life.

The mortality rate is excessively high in our ward, and on analysis in the acute cases it is found to be due almost solely to bronchopneumonia, with perhaps convulsions as the terminal factor. In general perhaps a third of the cases have complications of a more or less severe character: in this hospital a case is not admitted unless an urgent complication is present, so that complications are the sole recommendation for admission, thus the high mortality. The prognosis is to be more guardedly given in those rachitic cases with retracted costal walls, as the bronchitis, bronchopneumonia and collapse in such are



apt to become chronic, and a tubercular process ensue. Again, the family history must be taken into account: 98 of the patients gave a strong history of tubercle on one or other side, and of these 28 died, the majority with tubercular lesions, others of bronchopneumonia. Of the remainder, 54 were cured, 5 were relieved, 9 were much improved, and 2 remained in statu quo.

Measles seems to have a peculiar influence in rendering patients susceptible to pertussis, and undoubtedly prepares the ground for the tubercle bacillus. A history of measles occurring a few months, weeks, or days before or during the attack of pertussis was elicited in 128 cases, and it was a precursor to the pertussis in 33 of the fatal cases.

In discussing prognosis generally much depends on the season of the year, as naturally one would expect more serious complications during the cold winter and

early spring months, and such is of course the case. Environment is a very important element, as is also reasonable hygiene, plus careful nursing and feeding, especially in those children of tender ages. The children of the lower and poorer classes, where overcrowding, bad ventilation, and unwholesome food are complicating factors, do badly, and even on admitting such cases one finds that the natural stamina is so poor that given a serious complication they go under rapidly.

Mortality Statistics: The contemplation of this part of my subject is melancholy in the extreme. The mortality rate in the ward has been very high, namely 30.9 per cent of all the cases admitted, the number of deaths being 102 out of the 330 cases. Considerably over half the deaths occurred during the first year or year and a half of life, the actual figures being as follows:--

There was	1	death	in the first month of life
"	"	1	" between the 2nd. & 3rd. months.
"	"	1	" " 3rd. & 4th. "
" were	2	deaths	" " 4th. & 5th. "
"	"	2	" " 5th. & 6th. "
"	"	3	" " 6th. & 7th. "
" was	1	death	" " 7th. & 8th. "
" were	3	deaths	" " 8th. & 9th. "
"	"	3	" " 9th. & 10th. "
"	"	5	" " 10th. & 11th. "
"	"	3	" " 11th. & 12th. "
"	"	34	" " 1st. & 2nd. years
"	"	16	" " 2nd. & 3rd. "
"	"	13	" " 3rd. & 4th. "
"	"	11	" " 4th. & 5th. "
" was	1	death	" " 5th. & 6th. "

These figures show at a glance the havoc wrought in the children of the first months and first two years of life. The figures for sex come out almost equally for both sexes, there being 53 males and 49 females.

Acute Pneumonia of the bronchopneumonic type for the most part, accounted for 55 of the deaths, such complications as surgical emphysema, bronchiectasis, pleurisy, purulent meningitis (in 3 or more cases) with very frequently convulsions being merely superadded to the already severe pneumonic complication. It is noteworthy that the bronchopneumonia was especially fatal

in the early months of life, there being only one or two cases that were fatal after the 16th. month or second year at the latest. Convulsions were almost an invariable accompaniment in these fatal cases.

Other causes of death were sarcoma of right kidney, and secondary growths, with pneumonia in one case,

Pneumonia and diphtheria in another, the latter being the determining cause of death. Pneumonia and capillary bronchitis one case, congenital atelectasis pulmonum left lung one case, extensive atelectasis occurring in pertussis 2 cases, morbus cordis and diphtheria one case: septicoemia one case: capillary bronchitis together with diarrhoea and vomiting 1 case; endocarditis 1 case; cardiac failure after a severe paroxysm of coughing one case; marked emphysema and oedema of lungs one case.

Tubercle accounted for 35 deaths, its special incidence being noted between the ages of two and four years.



I have one solitary case only of acute general tubercle occurring at the age of 8 months. It was present either as an acute general tuberculosis, or as a caseating tubercle in the lungs, with caseation of the bronchial and mesenteric glands. Tubercular meningitis was the last phase in some cases. In most of the mortality cases from this cause, there was tubercle in one or other of the abdominal viscera as well. In 29 out of the 35 deaths there was a strong family history of tubercle, thus proving what an important item in prognosis a tubercular family history bears.

Results attained: Through treatment, or through

"masterly inactivity" in certain cases we claim to have cured 172 patients, roughly half or 52 per cent of the patients. There was marked improvement in 34 cases, some of these being taken out against advice.

In 16 patients we were able to effect some relief, some of these being unfavourable cases from the commencement, and possibly tubercular. Six remained in statu quo, no definite or distinct improvement being evident.

Treatment: I approach this part of the subject with a somewhat apologetic attitude. A fair trial has been given to practically every drug which has been vaunted as a remedy, if not a specific, for pertussis, and I fear that if I were to commence discussing them it would occupy more time than one could spare, and moreover it would serve no useful purpose. I merely, then, dwell on those drugs that we have found of distinct benefit at one time or another, and each in its own particular case. As far as I know no specific has been found for whooping cough, and the therapeutic remedies adopted merely aim at alleviating the severity or lessening the number of the paroxysms. The treatment for the most part is and must be symptomatic,

until an absolute specific is found, and to enable us to do this we must first isolate the specific virus and then utilise the antitoxine serum of an immunised animal.

Antipyrin, either alone or combined with carbonate of ammonia and Vinum Ipecac. has been used in 25 cases. The dose has varied according to circumstances from  $\text{grs.ii}$  every 4 hours to  $\text{grs.v}$  three times a day, or in some cases every 4 hours. I have found distinct benefit accruing in 10 of the patients. I am convinced that one can push the drug even up to the extent of  $\text{grs.x}$  every four or six hours for 3 or 4 days in the older patients without any depressing effect. It is of the greatest service in those cases where the whooping and coughing spasms are severe, and frequent, and where there are only few catarrhal signs in the lung. It should not be used in the bronchopneumonia cases if they are at all severe, owing

to its tendency to exert a depressant action on an already overstrained heart. Combined with Bismuth it is of great value in those cases where vomiting is a marked feature, its beneficial action being often rapidly noticed. The Carbonate of Ammonia which was used with it in 4 cases appears to enhance its value, the former being a stimulant, the combination therefore acting excellently well.

Belladonna was given alone in 11 cases in the form of the Tincture, and in 4 of these it certainly was of the greatest service. In two of the others it seemed to be doing some good for a time, but the effect was only temporary. The physiological effect of the drug was produced in 3 instances, and the patients were very depressed. The stools became large and offensive, the mouth parched, the tongue dry, and the pupils dilated. The doses ranged from M <sup>ss</sup>iii 4-hourly to M: x 4-hourly. I do not believe in admin-



tering large doses to commence, <sup>with</sup> although children are very tolerant of belladonna. It is far better, I think, to feel one's way commencing with M iii every 3 or 4 hours, and then gradually working it up. It is certain that it must eventually be administered in fairly large doses to produce any good effect. There are undoubtedly idiosyncrasies, and one must be on the "qui vive" for such developments. In a hospital one has the patient so much under observation that it is possible to experiment with large doses, a practice fraught with danger if carried out in outpatient work.

Belladonna combined with Bromide of Potassium in 6 grain doses three times a day was tried in 14 cases, and certainly had some influence for betterment of the whooping spasms, although the effect was never very striking. The mixture was ordered in certain cases where other drugs had failed for the most part, and perhaps it is in these that we see a distinct improvement after long trial with other drugs.

Mist. Potassium Bromide consisting of Bromide of Potassium grs.iii, Glycerine M xv, Aq. ad.1 drachm was useful in a few cases, although quite as often it produced no effect whatever. It was usually administered in 2 drachm doses every 4 hours, depending on the child's age. Although tried in 16 cases either as the sole remedy or after other remedies had failed, I can only recall it having done definite good in 5 or 6 cases at the most. It is certainly to be prescribed if a convulsive tendency shows itself, with the paroxysms. A bromide eruption was present in 3 of the cases where the drug had been pushed for some weeks.

Mist. Bromide & Chloral, each drachm containing grs.iii of the former, with grs.iii of the latter, was administered in 10 instances; in 3 there was distinct improvement, in 2 there was some relief, and in the others no effect could be detected. Such a remedy as this one certainly does good in certain cases, and

it simply means that one has to ring the changes so to speak with certain drugs until the one suitable is found, and such an one is this mixture. It should, of course, find its chief employment in a case where the spasmodic element is largest. It will probably do harm where there are grave lung signs, and the same may be said of all the remedies I have already referred to.

A mixture which has as its constituents Pot.Iodid. gr.  $\frac{1}{2}$   
Vin.Ip~~icac~~. M v, Chloral gr. ii and Aq.Chlorof. ad  
i drachm, was given in 8 cases, with distinctly excellent results in 2 cases, some improvement apparently for a time in two, and in the rest a failure. The dose of Chloral Hydrate is increased to gr. iii or iv, and the mixture administered every 4 hours if an appreciable effect is not produced in 3 days. The combination of Iodide and Chloral seemed to be an effectual antispasmodic in certain instances, though

why it should not work equally as well in other instances is a problem which we meet with so often in pertussis cases, one antispasmodic acting like a charm where another equally potent one absolutely fails.

Citrophen was used in 12 cases, in six distinct improvement took place, in one there was slight improvement, and in four there was little or no benefit. In one very obstinate and severe case in which Bromoform for 2 weeks, Creasote for 2 weeks, Antipyrin for one week, and Glycerine of Carbolic Acid for 3 weeks, had all failed to give relief. Citrophen did undoubted good, though it is only fair to remember that this was only after seven weeks stay and treatment in the hospital. The dose of Citrophen used for the most part was grs.  $\text{iii}$  every 6 hours, or grs.  $\text{v}$  three times a day. Citrophen finds its chief use in such cases as are likely to be benefited by antipyrin, and it is somewhat efficacious in patients who suffer from vomiting.



Bromoform has been used in 17 cases. Its use is not un-attended with risk, on account of its oily nature and high specific gravity. We have had one case of an outpatient admitted with symptoms of acute bromoform poisoning, which followed shortly after the administration of the last dose in the bottle. The child became comatose one hour after taking the last dose: the pupils were very small, there was no conjunctival reflex, no knee jerks, the pulse was slow and strong, the lips slightly cyanosed, extremities warm, and the breath smelt strongly of Bromoform. Treatment consisted in lavage of stomach, with the administration of strong coffee and brandy, the child becoming conscious again 3 hours later. The dose given has been either M iii 6-hourly, or M v, 3 or 4 times a day, the result being distinct improvement in 4 cases, slight in 4, and in 9 no definite effect. It has been used as long as 17 days, or up to three weeks in some of the cases.

was detected.

Children are very apt to get Carboluria, so a due watch must be kept on the colour of the urine.

Creosote was administered by itself in 9 cases, with cod-liver oil in 7 instances, with Tr.Camph.Co. in 2 cases and with Ol.Ricini in 5. I found it of signal service in those cases which were complicated with gastrointestinal trouble, and there can be little doubt that Creosote given internally is soon excreted by the air-passages, and consequently must and does in some instances exert a favourable influence on the specific morbid process. Its combination with codliver oil was deemed worthy of trial in the bronchiectatic and tubercular cases, when it is found that the patients usually put on weight, and the physical signs in the lung seemed to improve. Given with Castor Oil in proportion of Creosote M  $\frac{1}{2}$ -1 and Castor Oil M v every 4 hours, it was found invaluable often in the

diarrhoea cases, and of slight benefit in the cases of vomiting.

As a Vapour (Creosote ii drachms in a steam kettle) it was tried in 3 cases, with doubtful effect. An objection to its use as a vapour is the difficulty of confining the effect of the Creosote to a given case. It certainly seemed to allay the excessive irritability in the air passages, but the effect was transient only, and the discomfort produced by the vapour was found to be greater than the benefit derived by the patient.

Creosotal, which is the Carbonate of Creosote, was used in 2 cases, which were obviously tubercular, in one with marked relief.

Quinine, usually given as the Mist.Quinin. of this hospital, which has the following composition: Quinin.Sulph. gr.  $\frac{1}{2}$  Acid.Sulph.dil. M.i, Glycerin Mxv, Aq. ad 1 drachm. This was used in 23 cases, with benefit in

a quarter of the number. The patients often do not like it, owing to the somewhat persistent bitter taste left in the mouth, and one must be careful not to keep it on too long, dyspeptic symptoms appearing very frequently. It has been vaunted as a true specific for pertussis, but it does not act up to its reputation. It not infrequently induces vomiting in patients apart from the spasms of coughing. I think its use is limited to a large extent to help in clearing up a somewhat chronic case with râles, which persist for a long time in the lungs; likewise if administered in small doses and not too often it improves the digestive powers, and indirectly of course the child's nutrition. It should not be used when the paroxysms are severe and frequent.

Heroin which we tried in 6 cases produced the following results. In one patient gr.  $\frac{1}{60}$  given every six hours for six days did no good; it was then increased



to gr.  $\frac{1}{20}$  every 6 hours, with no appreciable result, but on being increased to gr.  $\frac{1}{15}$  every 4 hours it certainly allayed the severity of the paroxysms, but only after it had been given for 15 days. Another patient showed no improvement when gr.  $\frac{1}{60}$  was administered every 6 hours for 2 days, but on increasing it to gr.  $\frac{1}{30}$  every 6 hours marked improvement followed in 4 days. Yet another case did not respond to gr.  $\frac{1}{30}$  6. horis in 3 days, but was cured on increasing it to gr.  $\frac{1}{20}$  <sup>4</sup> horis in 14 days.

The other three patients in whom it was tried did not react to it, so a more extended trial was not given to this particular drug.

It certainly acts with promptitude in some cases in relieving the severity of the paroxysms, but must be pushed to gr.  $\frac{1}{30}$  or gr.  $\frac{1}{20}$  every 4 or 6 hours. I have experienced no bad effects from its use.

Bromoform should be dispensed in a dark bottle, kept in the dark, and the quantity of the mixture in the bottle should not be too great, so that the risk of an overdose of the drug in the later doses is diminished. Directions should be given that the bottle must be well shaken each time before pouring out the draught. If used for long it seems to exert a markedly depressant action on the patient, who becomes limp, and more or less ap<sup>2</sup>athetic. A bromoform rash appeared on the buttocks in one case, being something of the nature of a bromide eruption.

Glycerine of Carbolic Acid by itself in doses of M i or ii

every 4 hours, or again combined with Tr.Camph.Co. M iii to M v was very valuable in certain instances, and especially so where there was marked diarrhoea with green stools. It has been tried in 29 cases with benefit in quite a third, slight improvement in three, while in the others no appreciable difference

Chloride of Ammonium was tried, in the hope that the drug

might cause the secretion to be less viscid, and so easier of exp<sup>u</sup>ulsion. The relief to a paroxysm of coughing which follows the mechanical removal of the viscid mucus from the fauces and mouth suggested the employment, but the results obtained were by no means satisfactory. It was employed in 3 cases with some relief in one. In one patient  $\frac{\text{gr } 1}{24}$  was given hourly, in another it was combined with Nux Vomica, in another with Ipecacuana & Amm. Carbonate.

A mixture containing Antimon. Sulphurat. rub.

gr.  $\frac{1}{6}$ , extract Hyoscyami gr.  $\frac{1}{4}$ , Pulv. Glycyrrhiz

gr.  $\frac{3}{4}$ , Pulv. Gum. Acac. gr.  $2\frac{1}{2}$  Pulv. Sacch. Alb.

ad. gr. x. was tried in five cases, one patient apparently doing well on it, but further experience has not impressed one with its value.

Salol.

Gr. i. 4<sup>tis</sup> horis or grs. iii. three times a day was employed in ten instances, twice combined with mv. of Tr. Camph. Co, once with Bismuth, and once with Cascara. It was found of some service in the cases in which diarrhoea with offensive stools was a marked feature. Carboluria was apt to supervene early.

Mist. Ipecac: and Ammonia, which contained Carbonate of Am-

monia gr.  $\frac{1}{2}$ , Vin. Ipecac. M  $2\frac{1}{2}$ , Glycerine M X,

aq. ad. 3 i was used at one period or another of the illness in 44 cases, usually combined with other more specific treatment as well. It was a useful



remedy in those cases where there was much bronchitis or slight bronchopneumonia. It aided the expulsion of mucus from the air-passage and so indirectly relieved the paroxysms.

Mist. Aether Ammonia has the following composition.

Spirit of Aether  $3\frac{1}{2}$  minims, Aromatic Spirit of Ammonia  $3\frac{1}{2}$  minims, Tr. <sup>Auranti</sup> ~~Amantii~~ M ii. Camphor water to 1 drachm. It was given as a stimulant in 73 instances, most of the cases responding to its use. It has been found invaluable as a temporary remedy in many of the pneumonia patients and it was almost solely reserved for that class of patients. The usual dose is  $\frac{3}{4}$  i every 2, 3, or 4 hours.

Strychnine either hypodermically or by mouth was often requisitioned in the urgent cases. As a remedy for pertussis it has been tried in several instances, but apart from its stimulant effect, one

noted no other result. It was urgently needed in 31 cases and combined with Digitalis for Cardiac failure or incompetence in 10 others.

Thyroid Extract Gr. i. three times a day was given a trial in three patients with no improvement.

Phenacetin. Gr. iii. every four hours we tried in three cases with no response, and in another case it was combined with Heroin Hydrochloride gr. 1/24 and this patient did appear to re-act a little. Phenacetin probably needs to be given in much larger doses to produce a favourable influence, but one does not care to experiment with the larger doses to gain what would prove probably to be a very doubtful success.

Mist. Ipec. Opiata which is Vin: Ipecac. M  $2\frac{1}{2}$ , Sod.

Bicarb. gr. 2. Sp. Aeth. Nit. M  $2\frac{1}{2}$ , Tr. Camph.

Co. M  $2\frac{1}{2}$ , Aq. ad. 3i we used in four cases and it

certainly acts well for a time in relieving the

paroxysms. I find it very useful to give as an

Outpatient Mixture, there being always some improvement reported by the mother on her next visit. This mixture given with Chloral Hyd. Grs. ii or iii every four hours with Pot: Bromid. Gr. iii was employed in four instances with some temporary benefit. I think that is all we can say of this particular form of antispasmodic mixture.

Chloral Hydrate alone gr. iii.4<sup>tis</sup> horis was only tried once; it does not appear to act so well as with the combined Bromide.

The Carbolic Spray (1 in 40) was tried in four cases. The Resorcin Spray 2% in two instances. There was some alleviation in the irritability of the cough, but the spray obviously did not reach the seat of trouble.

Tr. Benzoin Co. 3i. to pint of hot water was tried in this as it has been in most lung or throat affections, but without appreciable benefit.

A Menthol and Cocaine paint 2% was used in a case where the cough was exaggerated by the existence of enlarged tonsils with inflamed fauces and pharynx. There was certainly some improvement.

Liquor Cocain Hydrochlor. 5% M ii. to ʒ i of water we thought might be of service where there were considerable vomiting accompanying the paroxysms. It did not come up to our expectations in the one case in which it was employed.

Hydrarg. Perchlor. gr.  $\frac{1}{6000}$  given three times a day did not encourage us to give it a further trial. We tried it in one patient without any result. During convalescence nothing has proved so useful as Codliver Oil and Iron, or Codliver Oil and Malt. These remedies were prescribed in forty-two cases and were of great service in picking up the patients. The dosage adopted was usually ʒ i to ʒ ii three times a day after food.



Linimentum Terebinthinae has proved itself to be a splendid

chest stimulant in those cases where the râles persist for a long time in the lungs and where one does not definitely suspect tubercle. It should be rubbed well into the chest front and back night and morning until a glow or a blush is produced. While on the subject of treatment and before leaving it

I must mention Leturiaux's researches into the etiology and treatment of pertussis, the report of which appeared in the Semaine Méd: 16th July 1902.

I reproduce an Extract of the Paper which was published in "The Practitioner" for October 1902.

Dr. Leturiaux has succeeded in isolating a bacillus which he claims is the cause of the infection.

Assuming that the disease is primarily located in the upper part of the respiratory tract, he made cultivations from the sputum of children suffering from pertussis in the following manner:- The thick

viscous part of the sputum is shaken violently in a tube containing sterilised water. After standing for a short time, the larger fragments of sputum are withdrawn on a platinum loop and subjected to this washing process five or six times more.

Afterwards they are pounded in a mortar with sterilised bouillon and a loopful of this emulsion is shaken up in a tube of liquefied agar. A drop from this tube is then placed in a second tube, which is treated in the same manner and a third tube is inoculated similarly from the second. The three tubes are then poured on to plates and incubated at 37° C. Colonies more or less numerous according to the amount of dilution very quickly appear. The bacillus thus isolated is a short thick rod with rounded ends almost as broad as long and presenting an ovoid appearance. It is apt to occur in groups and is actively motile. It is aerobic and stains well with Carbol-*fuchsin* or by Gram's method.

It grows well in agar, gelatine, serum and bouillon and its appearance is to some extent modified by the medium which is employed, it being more slender and elongated when cultivated on blood serum. The colonies on agar plates are rounded, semi-transparent and pearly in appearance. In stab cultures it presents a marked growth on the surface and slight streaks radiating from the line of puncture, resembling a nail with a large head. On serum and potato the colour of the growth is apt to be more yellowish. Leuriaux has inoculated small animals, chiefly rabbits with cultures of his bacillus in bouillon. Half a cubic centimetre injected into a rabbit's ear causes an abscess in twenty-four hours. A cubic centimetre injected intravenously causes the death of the animal within twenty-four to forty-eight hours. The rabbit soon becomes ill, with rapid respiration and spasmodic contraction of the diaphragm, the posterior limbs

become soon paralysed, then the anterior limbs and the thoracic muscles are affected. Respiration becomes irregular and a tonic convulsion heralds the approach of death. The products of the bacillus cause the same local and general symptoms as the microbe itself, filtered bouillon cultures being equally fatal. It is however the therapeutic results which Leturiaux has obtained from his researches which make his paper so interesting. He has used successfully a serum treatment of whooping cough. His serum is prepared much as Roux prepares diphtheria antitoxin. Horses are injected with gradually increasing doses of filtered bouillon, at first only of 2. c.c's in amount and rising after three months to 150 c.c's. There is considerable re-action to the injections. In about four months when a litre of toxine has been injected, the blood is regarded as being sufficiently anti-



toxic and the animal is bled. Sixty-six cases of pertussis have been treated with this serum. As soon as the diagnosis is certain it should be employed, and the earlier in the disease the method is used, the better is the result obtained. A dose of 5 c.c. is considered sufficient for children up to two years of age. Above that age 10 c.c. may be used with advantage. The dose can be repeated once or twice if much improvement is not manifest; usually however this is not found necessary. If used early in the course of the infection, the spasmodic cough and the vomiting diminish rapidly in the frequency of their occurrence and in their severity, and disappear completely in a few (5 - 8) days. In more advanced cases, though the improvement is very marked, it is naturally not seen so soon. Of the sixty-six cases only five showed no improvement after the use of the serum. LeVriaux

considers that his treatment requires no assistance from the methods. All that he does is to give an emetic of Ipecacuana before starting his injections. This clears the passages of mucus and also assists in removing any swallowed sputum by acting on the bowels. He believes in careful feeding and general hygienic precautions. Altogether it must be admitted his work is well worthy of imitation. We may add here that Vincenzi (Centralb. f. Bakt. March 12, 1902) describes the bacillus which he has isolated from whooping cough sputum. This is also an oval cocco-bacillus and grows well on agar and other media and in its main characteristic resembles the microbe described by Jochmann and Krause. Jochmann stated however that his bacillus only grew on media containing blood. Vincenzi thinks that the fact that his own germ grows on agar may be accounted for by the blood

so often mixed with the sputum is pertussis. But the long and careful washing of the sputum by Leoriaux appears to us to render this explanation unsatisfactory, if, indeed the bacilli are identical". This serum we have tried in three of our patients in this Hospital and I give you the results as follows:-

Patient No. 1 was a female three years of age who was admitted with the history of a cough having developed fourteen days prior to admission, the whoop first being noticed four or five days prior to admission. There was no sickness or diarrhoea. No Tubercle on either side of the family. The other two children in the family quite healthy. The patient was a full-time child, and has had rickets, gastrointestinal catarrh and measles. On admission the chest was noted to be of normal shape, the percussion note over both lungs resonant, the breath

sounds normal but there were numerous subcrepitant râles and rhonchi, most numerous at the bases posteriorly. Pulse rate 128 per min. The abdomen normal; no enlarged glands; knee jerks brisk. Two days later there were some rather sharp crepitations at the right apex. Temperature on admission  $99^{\circ}.4$  F. rose to  $99^{\circ}.8$  F. on the evening of the next day and to  $101^{\circ}$  F. on the evening of the day following.

Three days after admission 10 c.c. of the antitoxine serum were injected into the subcutaneous tissue of the abdominal wall. The temperature that evening was  $101^{\circ}.4$  F. down to  $100^{\circ}$  F. next morning. I reproduce the chart which shows at a glance the subsequent progress of the Case. Two days after injection, there was a heavy deposit of ~~urates~~<sup>urates</sup> in the urine. On the fifth day following the injection the patient was worse, she was a little pale and the lips bluish. The breathing became very rapid



up to 80 per min. the pulse rate being 186.

The adventitious sounds over the right lung had increased. Over the left lung the breath sounds were less loud and the adventitious sounds less numerous. Later in the day some sharp crepitations were heard just above and outside the left nipple and in the left axilla. The next day the child seemed better; however there were numerous crepitations in lower lobe of both lungs posteriorly and along the anterior border. Temperature up again to 102<sup>.4</sup>. Patient died suddenly early on the 7th morning following the injection. The lungs were found to be tubercular and child had acute general tubercle. In studying the chart one should notice to study diminution in the number of the coughing and whooping spasms after the day of injection when they were at their highest viz., nineteen coughing paroxysms with eleven whooping. On the day before

death there were eleven coughing and three whooping paroxysms. Whether the result attained was due to the serum or merely to the progressive lung affection is hard to decide, in all probability the lessening in severity and frequency of the whooping and coughing paroxysms went with the lung trouble plus the temperature.

Patient No. 2 was a female of three years and eleven months. There was a history of a cough off and on for about twelve months, became worse three weeks before admission, the whoop appearing two days before the latter date. No vomiting. The mother is anaemic, paternal grandfather died of phthisis. Three children alive, no miscarriages. The patient was a full-time child. On examination the child was seen to be well nourished, percussion note resonant everywhere, breath sounds normal, no adventitious sounds. Abdomen normal.

No enlarged glands; knee-jerks brisk, plantar reflex. is flexor.

Urine 1037. No albumin or sugar. There were fifteen coughing and ten whooping spasms on the day following admission. On this day 10 c.c. of the serum were injected into the subcutaneous tissue of the left iliac region. There was practically no reaction either locally or generally after the injection of the serum, the chart showing the course of events subsequently. The whooping and coughing apparently did not abate one jot after the anti-toxine injection, and three days after the chest was reported as entirely free of physical signs. The next day however there were a few crackles at the left base. Five days after the injection the paroxysms were reported as being more severe, the child becoming very blue. On the 6th crackling râles and rhonchi were present in considerable number over lower left lobe posteriorly.

Thus the case progressed for fifteen days after the date of injection, the lung signs and symptoms being more exaggerated, and the temperature showing slight fluctuation. On this day a second injection of 5 c.c. was given. The same night there was marked tenderness for some distance round the site of injection. Temperature  $99.4^{\circ}$  F., later  $102.4^{\circ}$  F. Next morning the temperature was  $100.2^{\circ}$  F, the swelling and tenderness being more marked. Two days following the left labium majus became red, swollen and tender. The lungs were now resonant, a few crepitations only being heard. The child was quite bright; temperature normal. For the next fortnight the whooping and coughing spasms although somewhat severe became far less frequent and were practically in abeyance twelve days after the second injection. A relapse now set in with a temperature of  $100^{\circ}$  F. and having no more serum and not being able to procure any although we wrote over to Brussels twice, we had to



continue treatment on the usual lines. The child eventually got quite well, but remained in Hospital for six weeks more owing to a severe vaginitis having arisen of a gonorrhoeal nature, there being an epidemic in the ward at the time (eleven cases in all). This patient showed absolutely no improvement after the first injection, but a decided advance was made for the better after the second, and had we been able to have given a third, we might have brought about a cure earlier.

Patient No. 3 was another female of four years, with a history of a cough for one month and a whoop for five days - much worse at night. No vomiting. The mother is healthy, but father is Phthisical. Two children in the family have died of Tubercle, the other two that are living are also delicate. The patient had measles five months before this date and has been ill ever since. Slight otorrhoea.

On examination it was noticed she was well nourished though rather anaemic. No evidence of rickets. No enlarged glands. Abdomen normal.

Sublingual ulcer present. Urine acid - no albumin or sugar, but a deposit of urates.

Patient seemed fairly well apart from a few sibilant rhonchi scattered over the front of the chest. There were eight coughing and four whooping paroxysms on the second day after admission. On the third day

after admission 10 c.c. of Letriaux's serum were injected into the right iliac region. On the evening of the day following the temperature rose to 99.4 F. and continued to rise with a remission on one day until it reached 102.2 F. on the fourth evening following the injection. The pulse then was 180, and in the chest there were numerous sibilant rhonchi plus crepitations at the left base. The note was resonant everywhere. On the next day there were coarse râles heard at both posterior bases, the site of injection was healthy and the temperature had come down to 100°F. The sixth day following injection found the child better, the reaction having passed off, but there was absolutely no improvement in the coughing. On the seventh day an articular eruption appeared over the right side of abdomen and right thigh, it lasted only a few hours and was probably an antitoxine

erythema. The patient shortly after, this became very ill and there were definite physical signs in both lungs indicating areas of collapse with broncho-pneumonia. The temperature fluctuated markedly with evening rises and morning drops for about six weeks, the child showed considerable wasting and altogether we suspected Tubercle, having in view the strong family history. In addition a gonorrhoeal vaginitis appeared which further complicated matters. Eventually however everything cleared up, the temperature steadied and the child left the Hospital perfectly well. This case was an object lesson to one to avoid jumping too hastily at a diagnosis of tubercular disease, and giving an unfavourable prognosis, whereas it subsequently proved itself to be a chronic broncho-pneumonia with areas of collapse. The serum in this case produced no other effect than to make the child considerably worse. We have unfortunately not been



able to experiment with this serum on other patients as our supply gave out and though we have written on several occasions to Dr. Lenrioux, no notice has been taken of our communications.

On the whole then the results obtained by the use of the serum in this hospital were not sufficiently striking to enable us to term it a success.

The bacillus separated and cultivated by Lenrioux being a motile one, it would be interesting to see whether clumping were brought about in using the serum of a patient who had recently had whooping cough and in whom we know from experience there is an immunity established also, could not such a serum which is certainly antitoxic be used as a therapeutic agent? It is perhaps an impossible suggestion.

Before bringing this subject to a close, I should like as an addendum to record the results of a blood

count made in a few cases of pertussis. The figures may not perhaps convey much information, as unfortunately I have been unable to do a count on a case of uncomplicated pertussis.

I. Blood count, in a somewhat anaemic rickety child, who developed whooping cough definitely seven days after admission. The count was made fourteen days after the whoop developed, and was as follows:-

Haemoglobin	35%
Red Corpuscles	4,378,000.
White	13,000

The child was put on liquor arsenicalis M ii t.d.s. and 1.9 days later the count was:

Haemoglobin	28%
Red cells	3,870,000.
White	9,000

The arsenic was then stopped, and Ferri. Redact. gr. ii t.d.s. was prescribed. Twenty days later the count was:

Haemoglobin	42%
Red cells	4,460,000
White	5,000.

It seems then that in this case there was at first a considerable increase of white cells; whether as a result of the pertussis or of the anaemia is difficult to determine. Later the red cells and haemoglobin became diminished, and this when the child was on arsenic.

On Ferri.Redactum the number of red corpuscles increased to normal practically, the haemoglobin also showing a marked increase in percentage. The white cells presumably dwindled down to their normal number.

II. Female, two years of age, whooped for six months having had pneumonia as well - left base pneumonia with pleurisy and effusion over the right lower lobe. Tuberculosis on both sides of the family.

Child has had measles three times, but not recently.

Red cells	5,275,000	)	
		)	ratio 1 to 220.
White "	24,000	)	

This was done seven months after the whooping cough had ceased and indicates a condition of leucocytosis not lymphocytosis.

III. Female,  $4\frac{8}{12}$  years. Duration of cough ten days, whooped one day after admission and there was present a pneumonia of left lung upper lobe, left axilla and left lower lobe posteriorly, also a left pyopneumothorax due to the rupture of a bronchiectatic cavity at the left base. In this case we had on counting a leucocytosis of 40000, this was after the pyopneumothorax developed.

IV. Male,  $2\frac{3}{12}$  years, onset of cough two months before admission, whoop fourteen days. The blood count was made fifty days after the onset of the pertussis. - there had been a pneumonia of the right apex anteriorly and posteriorly on admission.

Haemoglobin	58%
Red cells	3,700,000
White cells	19,000.



V. Female, fourteen months old with rickets and enlarged spleen - child having developed pertussis eighty-two days prior to the date on which the count was made, so that the serum could at this time be fairly assumed to be antitoxic.

Red cells	2,535,000
Haemoglobin	41.5%
White cells	160,000.

A differential count of 400 white cells gave the following:

Small lymphocytes	65.75)	
	)	74.75
Large lymphocytes	9.0 )	
Polymorphonuclear		24.5
Eosinophiles		<u>0.75</u>
		100.

No myelocytes seen. One nucleated red blood disc was seen to every 100 white cells.

Another count was made twenty-one days later, arsenic m i t.d.s. having been administered for ten days prior to the count. The result proved to be:

Red blood cells	3,275,000	)	
Haemoglobin	29%	)	Ratio of white
White cells	4,500	)	to red cells =
		)	1 to 728.

Differential count.

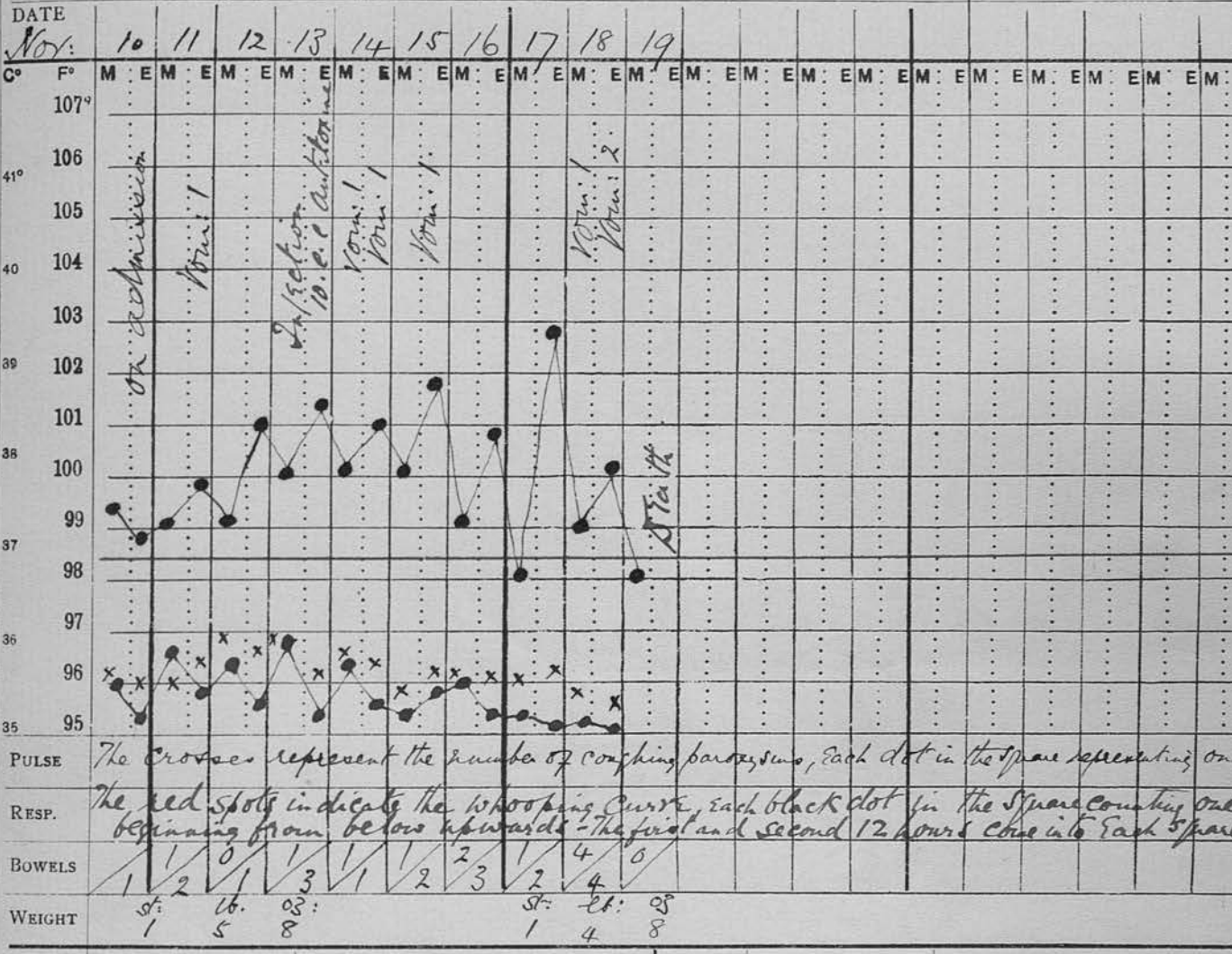
Small lymphocytes	54.	)	
Large lymphocytes	21.5	)	75.5.
Polymorphonuclear		)	23.
Eosinophiles		)	1.5.
			<hr/>
			100.

Varying sizes of young white cells were very marked.

No poikilocytosis. No mast cells or myelocytes seen.

No increase in blood platelets. No nucleated erythrocytes.

No. *Rose Cummings* AGE *3 Years* WARD *Goldsmiths* UNDER CARE OF *Dr. Voelcker*  
 DISEASE *Cas. No. 1.* RESULT

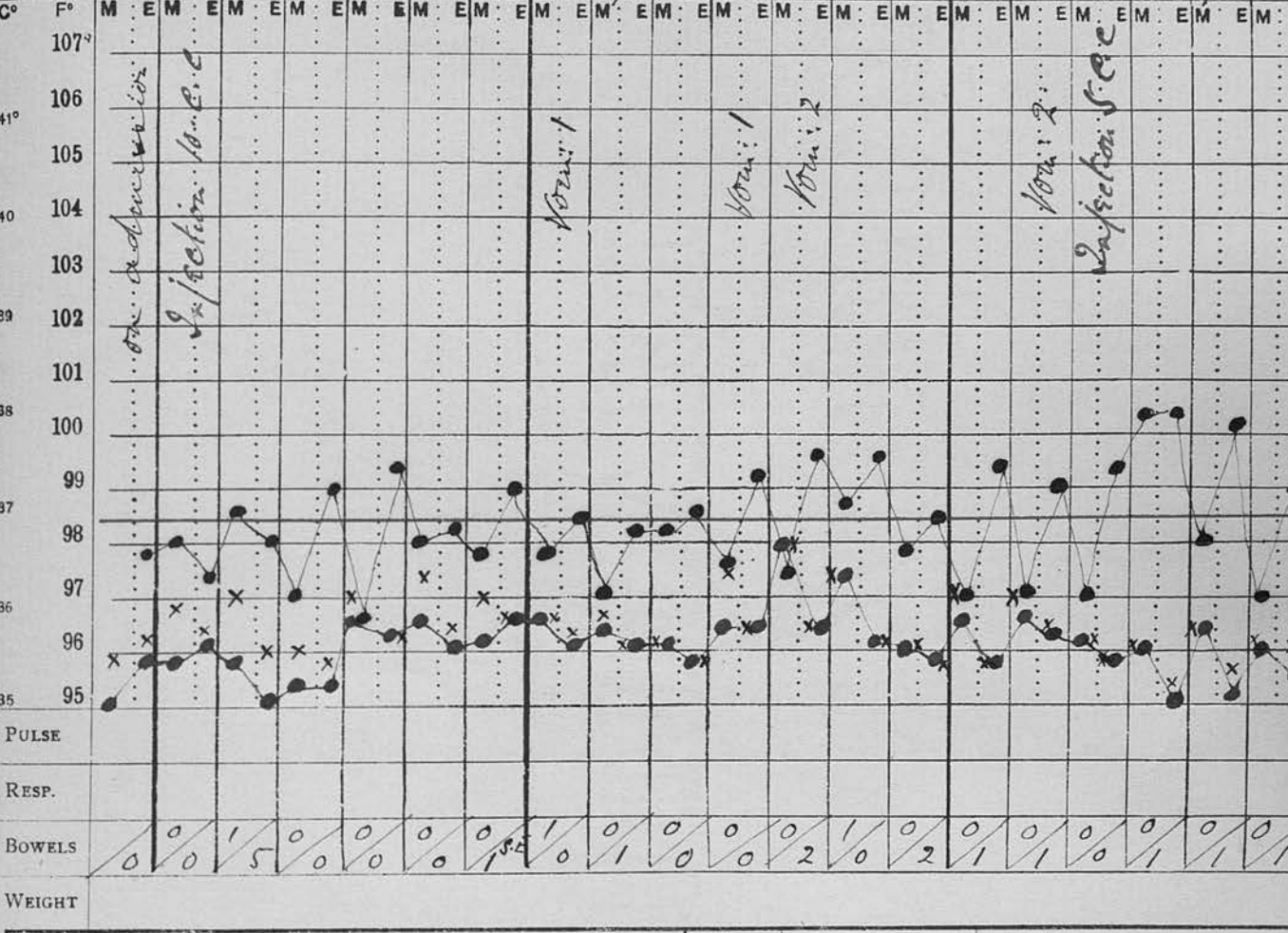


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DATE	DIET	TREATMENT	DATE	DIET	TREATMENT
<i>Nov. 10</i>	<i>Milk Dist Extra milk Brandy 3 ss. Sperdin</i>	<i>Leuridan's Antitoxin 10.c.c infect. Subcut. Vin Splane 3i 12.30 pm</i>			

No. NAME *Jessie Gregory.* AGE *3 3/12 yrs* WARD *Goldsmiths* UNDER CARE OF *Dr. Volcker.*  
 DISEASE *Case No. 2.* RESULT *Dis.*

DATE *Nov.* 19 20 21 22 23 24 25 26 27 28 29 30 1 2 3 4 5 6 7 8



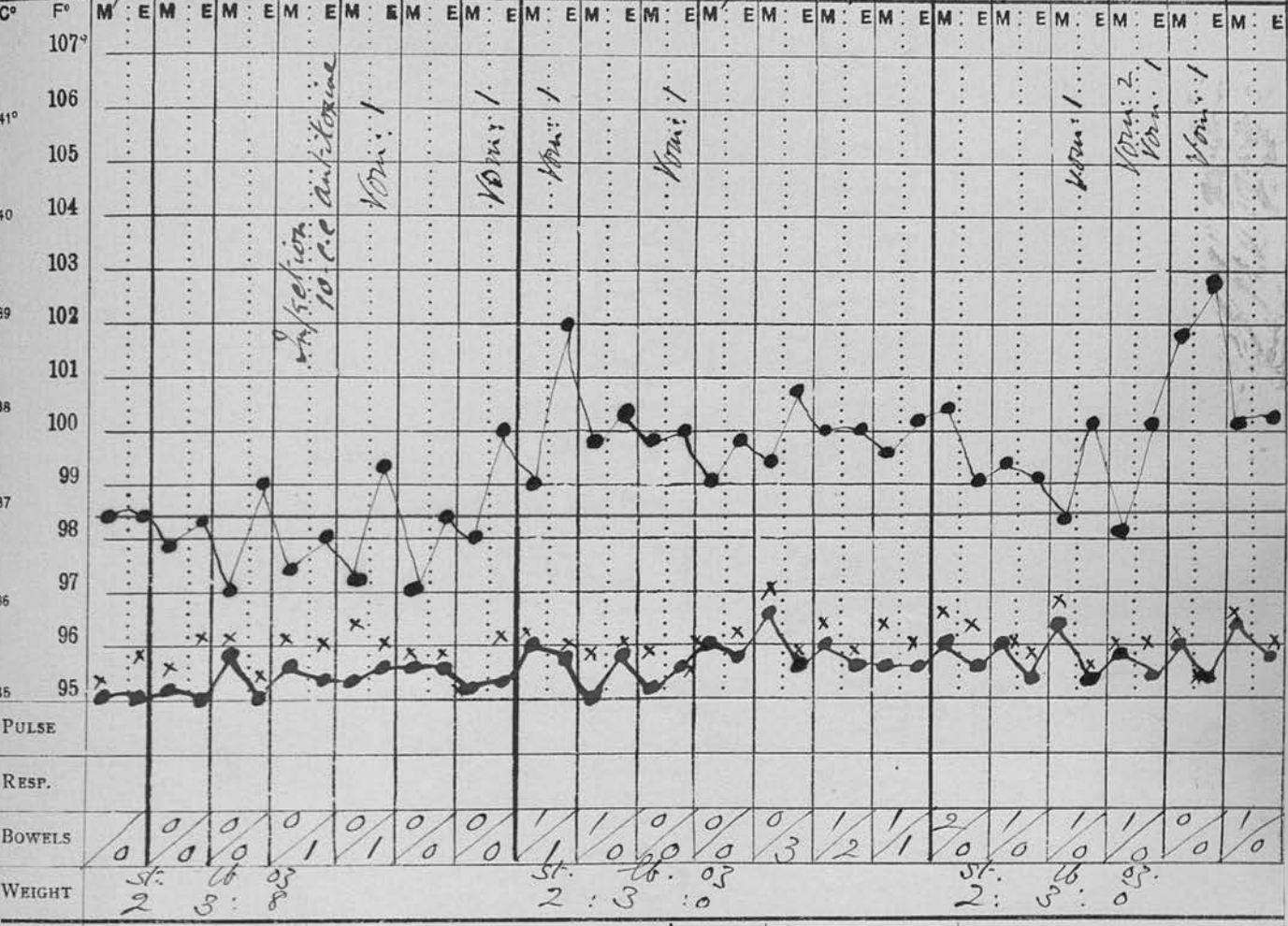
DATE	DIET	TREATMENT	DATE	DIET	TREATMENT
<i>Nov. 19</i>	<i>Full</i>	<i>Leuridan's Antitoxin Serum 10.c.c</i>			
<i>Dec. 5.</i>		<i>Leuridan's antitoxin Serum 5.c.c</i>			



No. *Florence Ethel West.* AGE *4 years* WARD *Coldsmiths* UNDER CARE OF *Dr. Volcker.*

DISEASE *Case No. 3.* RESULT *Sc.*

DATE *Nov.* 17 18 19 20 21 22 23 24 25 26 27 28 29 30 1 2 3 4 5 6



MARGIN TO BE LEFT FOR BINDING.

DATE DIET TREATMENT DATE DIET TREATMENT

*Nov. 20 Full Leuriaux's Antitoxin 10.c.c. injct. Subcut.*