acute rheumatism as an infection, we must be prepared to face the question whether it is a peculiar infection or an attenuated septic infection the result of diverse causes. If we have the treatment of that terrible disease at heart, we cannot be content to rest with the vague assertion that acute rheumatism is an attenuated septicaemia, and, if there is any hope in the serum and vaccine methods, it is clearly of increasing importance that we determine the real position of acute rheumatism. Now, if it is merely an attenuated septicaemia, we must surely admit it may be sometimes pneumococcal, for everything points to that infection as producing a septicaemia. Yet what do we find when, leaving theory, we come to clinical and pathological facts?

In the first place we find that pneumococcal pericarditis is at its maximum in frequency under 4 years of age, whereas rheumatic pericarditis is rare under 5.

Endocarditis is almost invariable in rheumatic pericarditis, most exceptional in the pneumococcal form. Dilatation is invariable in the rheumatic form, it is not considerable or apparently frequent in the pneumococcal.

Empyemata, pneumonia and pleurisy are the rule in the pneumococcal type. Empyemata never occur with rheumatic pericarditis.

Lastly, we have all grades of pneumococcal pericarditis—the acute and virulent haemorrhagic type, the grossly purulent, the fibrino-purulent, and the chronic adhesive.

In rheumatism also we have the virulent, the fibrino-serous, and the chronic adhesive forms.

Throughout the history of pneumococcal pericarditis we notice the tendency to pus formation and the absence of pericardial friction; throughout the rheumatic the tendency to resolution and the presence of friction.

The micrococcus obtained from the pneumococcal pericarditis is a lanceolate, encapsuled, delicate diplococcus; that obtained from the rheumatic form is smaller, not encapsuled, and resistant in its growth, though prone to lose its virulence.

Rheumatic arthritis is exceedingly frequent with rheumatic pericarditis; pneumococcal arthritis, though well recognized, is exceptional.

If there is any meaning in the term "disease," then, I think, we have two different processes here, and if that is so, then we must admit that if acute rheumatism is an attenuated septicaemia, it is not an attenuated pneumococcal septicaemia.

Now let us turn to the rare cases of other forms of purulent pericarditis in childhood, those secondary to the *Staphylococcus aureus* in osteomyelitis for example. In these we find multiple abscesses.

Again, in those septic infections we may find an abscess in one place and clear fluid in other places. For example, pus in a knee-joint and clear fluid in one pleura. Surely, then, in rheumatism when we find clear fluid in one place, we ought to find now and again, if it is an attenuated septic infection, abscesses elsewhere. Such an occurrence is beside my experience, and I hold that acute rheumatism is as special a disease as is a staphylococcal or pneumococcal disease.

PROGNOSIS AND TREATMENT.

The prognosis of suppurative pericarditis in small children is exceedingly grave, and one cannot help feeling that unless a serum can be obtained which will combat these general pneumococcal infections an improvement in our powers of diagnosis will not greatly help our methods of treatment.

Surgical intervention at present offers the best chance of success, and there are a certain number of cases on record of recovery. The best results are to be expected in children older than those with which this lecture has been chiefly concerned, and in which there is a considerable effusion of liquid pus. I feel sure that in many of the cases in infants, even if the pericardium is opened, the relief afforded would be but comparatively slight, on account of the difficulty in the drainage of the tenacious exudation and the multiple associated lesions. Yet pathological investigation amply shows that there is sometimes a remarkable vitality exhibited by children suffering from this form of pericarditis, and that great natural efforts are made for recovery; this encourages us to persevere in aiming at an early diagnosis.

Paracentesis of the pericardium will of course be required

in these cases; and if there is sufficient evidence of a purulent effusion, it is better to proceed at once to open the pericardium after the paracentesis rather than to first explore and then operate at a later date.

There has been a good deal of difference of opinion expressed as to the best site for exploration. Dr. Samuel West has recently advocated the left lower margin of the cardiac dullness—that is, in the region of the impulse.

In uncomplicated cases of pericardial effusions few, I imagine, would doubt the wisdom of this ruling. In the condition under consideration, however, we must be prepared to adapt ourselves to circumstances, and choose the fourth or fifth intercostal space close to the sternum, or the left costo-xiphoid angle, passing the needle inward and upward, behind the lower end of the sternum.

There is no doubt that a needle has been passed into the heart on occasions, and as a rule without ill effects, nevertheless it is an anxious occurrence.

Lastly, inflammatory changes in the anterior mediastinum and great thickening of the pericardium itself make it sometimes difficult to tell when the pericardial cavity has been opened, and it is not surprising that now and again a pericardial cavity has been thought to be opened and a drainage tube introduced into the mediastinal anterior tissues.

SOME CONSIDERATIONS REGARDING PNEUMONIA IN CHILDREN.*

DRAWN FROM AN ANALYSIS OF 500 CASES.

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THAT pneumonia in children is a very interesting, a very common, and a very fatal disease will, I venture to think, be generally admitted. During the period I have had charge of a ward in the Edinburgh Sick Children's Hospital 3,300 cases of all varieties of disease have been treated, and of these over 500, or nearly one-sixth, have been cases of pneumonia, clearly proving, if proof were needed, how largely it bulks amongst the serious diseases of the children of the poor, necessitating treatment in hospital. Much has been written on the subject, and it has been so thoroughly discussed in all its phases, and the field so carefully gleaned, that I fear my contribution will not add very materially to what is already common knowledge regarding the disease. Still, I consider that when one has had a large experience it is desirable that that experience should be critically digested and summarized, and it is more with the object of ascertaining what I myself have learned than with the view of contributing any new facts of material importance that I have ventured to write this paper. I was confronted at the threshold of my inquiry with the consideration of the method to be adopted in the grouping and classification of the cases—a question bristling with many difficulties and pitfalls, whether we approach it from a bacteriological, a clinical, or a pathological standpoint.

Broadly speaking, pneumonia in children may be divided into cases of bronchopneumonia and those of the lobar variety; but owing to the many anatomical and physiological differences in the respiratory system of the child compared with the adult, the discrimination between the two conditions is frequently puzzling and sometimes impossible, and the difficulties assume wider proportions as one's knowledge of the subject increases. Doubtless in the majority of cases the process is absolutely typical of one or other variety, but there are cases, occupying a position on the borderland, which are very difficult to place, and others again of a mixed variety which seem to defy classification, whether we investigate them clinically or pathologically.

Clinically we recognize a case as belonging to the lobar type of pneumonia by its abrupt onset, its lobar distribution, its continuous high temperature, terminating by crisis, and its speedy resolution; while we characterize another as bronchopneumonia by its slower onset frequently following bronchitis, by the marked remissions of temperature which terminates by lysis, and after a prolonged

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course is followed by a protracted convalescence. Were all our cases marked by these broad characteristics there would be no difficulty in discriminating between them; but pneumonia in children assumes so many varieties, and has the habit of developing so many unexpected eccentricities, that we are often sorely puzzled in which group to place individual atypical cases. Thus, in a considerable proportion of cases of bronchopneumonia, the symptoms commence abruptly with a sudden rise of temperature, which assumes a less remittent type than usual, and runs a less prolonged course. Such cases are styled primary bronchopneumonias, and can generally be recognized by terminating by lysis (though by no means always so) and by being accompanied by general bronchitis.

Sometimes, again, we meet with cases of lobar pneumonia, which are characterized by their wandering disposition, creeping slowly from one part to another of the lung, the crisis in consequence being indefinitely delayed, and the temperature not infrequently falling by lysis. Indeed, a certain remittance of temperature and termination by lysis is a not uncommon occurrence in the lobar pneumonias of young children. It is also far from uncommon to find lobar pneumonia accompanied by bronchial catarrh in the same or opposite lung. In hospital practice, again, when a case is first brought under notice late in the disease, when a large patch of pneumonia has developed, and when the early history, as is often the case, is indefinite, we may have difficulty in determining in which category to place it.

Turning from the clinical to the pathological aspect, we do not find the elucidation of the question any simpler. An ordinary lobar pneumonia is easily distinguished by its lobar distribution, its more massive consolidation, consisting of an exudation of cells, chiefly leucocytes, in a fibrinous matrix, and a bronchopneumonia by its more widely disseminated patches of consolidation, the bronchi and the alveoli having their cavities blocked by an exudation consisting mainly of epithelial cells. Having made this simple statement of fact, we by no means dispose of the many discrepancies which the *post-mortem* table brings under our notice. For instance, how frequently do we find cases of bronchopneumonia with a lobar distribution, brought about partly by the confluence of adjoining patches, partly by collapse of the lung, and partly by thickening of the connective tissue surrounding the bronchi and alveoli. It is, moreover, by no means uncommon to meet with a double lobar pneumonia or with a bronchopneumonia confined to a single lung. In my own experience I have occasionally seen on the *post-mortem* table cases displaying a lobar pneumonia in one lung and a bronchopneumonia on the opposite side, and on at least two occasions I have seen the superaddition of grey patches of bronchopneumonia, appearing like a mosaic on a previously hepatized lung. The difficulty of discriminating between the two varieties even extends to the microscopic examination, for we have it on the highest authority that in a case of bronchopneumonia we may have, in addition to the proliferation of epithelial cells, an exudation of fibrin similar to what occurs in lobar pneumonia.

The general impression seems to prevail that in the age of the child we have a valuable aid to the classification of the disease, bronchopneumonia being regarded as the pneumonia of infancy and lobar pneumonia as that of older children, and, though this is true in a general sense, it is far from being a correct interpretation of facts.

While readily admitting the frequency of bronchopneumonia in young infants, and its uncommon occurrence in children over four years, except as the sequela of some infectious disease, I wish to emphasize the extreme frequency of lobar pneumonia in young infants. In the Edinburgh Sick Children's Hospital, where over a third of the cases admitted are under two years of age, I have treated a large number of infants whose ages varied from a few weeks to 2 years who suffered from lobar pneumonia, and the diagnosis has frequently been confirmed by *post-mortem* examination. In view of these many difficulties I was tempted to adopt a classification which would escape the necessity of specifying the type of pneumonia present by simply dividing the cases into the two groups of primary and secondary pneumonias. I felt, however, that this method, however convenient, might imply that I accepted the view that primary bronchopneumonia and lobar pneumonia were practically one and the same condition,

differing only in their form, on account of the anatomical and physiological peculiarities of the child's pulmonary system.

As I wish to dissociate myself entirely from this view, and as there are other disadvantages connected with this classification, I have adopted the usual grouping of the cases under the headings of lobar pneumonia and bronchopneumonia, dividing the latter again into the subdivisions of primary and secondary.

Each case has been carefully and critically examined before being classified, and I have endeavoured as far as possible to eliminate every source of error. The tendency has been on the whole to place doubtful cases amongst the bronchopneumonias. On analysing the cases according to this basis, I find that 147 out of the 500 belong to the lobar variety; 85 of these cases occurred in boys, and 62 in girls, which is, I think, about the usual proportion, being 3 boys to every 2 girls attacked.

An immense majority of the cases occurred during the winter and spring months, the cases during these seasons numbering 101, compared with 46 during the summer and autumn, as shown in the accompanying table:

Winter, 74; spring, 27; summer, 27; autumn, 19.

A comparison of the different ages at which lobar pneumonia occurred gives strong support to the statement I have already made, that it is a disease of very frequent occurrence in young infants. Forty-five of the cases occurred in children under 2 years old, 12 of these being in infants under a year, the youngest case treated being only 3 weeks old. The annexed table gives the numbers treated at different ages:

45 cases under 2 years old (12 being less than 1 year).
42 cases between 2 and 5 years.
60 cases between 5 and 12 years.

An examination of the portion of the lung affected shows the very decided preference of the disease to attack the bases of the lungs, the pneumonia being confined to the bases in 88 out of the 147 cases. In nearly one-half of the cases the left base was affected. This is quite in conformity with the experience of others, though I can offer no reasonable explanation why it should be so. Next in order of frequency comes the right base, closely followed by the right apex, and after a long interval by the left apex. Both lungs were involved eight times, but I make this statement with reservation in the absence of confirmation by *post-mortem* examination, as owing to the readiness with which sounds are communicated in children from one side to the other, and owing to the difference of the percussion note according to the expansion of the lung, I believe that double pneumonia is oftener diagnosed than is perhaps always justifiable. The annexed table gives the portions of lung affected:

	Cases.
Left base	63
Right base	25
Right apex	23
Left apex	11
Both lungs	8
Whole of one lung	10
Site not ascertained	7

I have divided the cases into those manifesting no physical signs, those with a late development of physical signs, and those in which they were well marked. I think it may be accepted as a fact that a small proportion of cases of pneumonia do occur which do not give evidence of its presence by physical signs. I believe that this proportion would be less than it is frequently stated to be if our methods were more accurate and our examinations more thorough and frequent. Probably it is more often an expression of carelessness on the part of the physician than of non-existing physical signs in the lungs of the patient. I have occasionally, when on the point of concluding that no physical signs were present, found a patch of well-marked pneumonia in a spot which I think is often overlooked in the examination of the chest—namely, high up in the axilla. Still, I think, admitting all these qualifications, there remains a small residue of cases in which no physical signs can be detected. Seven of my cases have been classified under this heading. I believe that the explanation of the absence of physical signs is to be found either in the fact that the pneumonia was of the abortive type which never went on to consolidation, or that it was of small dimension, or situated so deep in the substance of the lung that the sounds were altogether

deadened. My impression is that the greater number of these cases are situated either at the apex or at the roots of the lungs.

A late development of physical signs is of frequent occurrence. It is often not until the fourth or fifth day that they become apparent. Seldom do we find them delayed till a later date, although I have seen them manifesting themselves for the first time as late as the seventh or eighth day. I remember one such case giving me considerable anxiety. From the general symptoms and appearance of the patient I was practically certain that the case was one of pneumonia, and waited anxiously for the development of physical signs to confirm my diagnosis. I had, however, to wait till the ninth day—the day after the crisis—for that confirmation, when my anxieties with regard to the case had already been put to rest by the fall of the temperature and the improvement in the general condition of the patient. Cases like the foregoing are very embarrassing when one is being pressed for an opinion and can only give a provisional diagnosis.

I shall not detain you by recounting the physical signs in well-marked cases. My experience leads me always to be very apprehensive when I hear distant faint or absent breathing or breathing of an indeterminate or jerky character accompanied by an almost tympanic percussion note. These physical signs are very typical of an early pneumonia, especially when accompanied by loud, harsh puerile breathing on the opposite side. To those unfamiliar with the examination of the chest in children it often seems as if the latter was the lung about to be affected. The only other two remarks that I wish to make regarding the physical signs are that the crepitant rle, so distinctive of the early stage of pneumonia in the adult, is more frequently absent than present in the child, and that the physical signs disappear with extreme rapidity. I have frequently been astonished to find scarcely any trace of pneumonia left two or three days after the crisis, and in the majority of cases in the course of a week they have almost entirely disappeared, with the exception of a slight impairment of the percussion note. The chief symptoms observed heralding the onset of pneumonia were vomiting, headache, pain in the side, shivering, convulsions, and diarrhoea. Vomiting was by far the most frequent symptom, and occurred in nearly two-thirds of the cases; indeed, in my experience there is no other disease of childhood, with the exception, perhaps, of scarlet fever, which commences so frequently with vomiting as pneumonia. In some cases the vomiting continued for a day or two after the onset. Pain is a common symptom, but is much oftener referred to the abdomen than to the thorax. The combination of vomiting with pain in the abdomen, which causes the abdominal walls to be kept rigid, leads to mistakes in diagnosis. On no fewer than seven occasions have cases, which subsequently proved to be cases of pneumonia, been sent into my ward with the diagnosis of appendicitis. Convulsions do not occur so frequently as an initial symptom as is generally supposed. They were noted marking the onset of pneumonia twelve times, and were generally accompanied by headache, drowsiness, and apathy, being the symptoms associated with the condition known as cerebral pneumonia, and are usually described as being much more frequent when the pneumonia is situated at the apex. My cases only give very slender support to this view, as the apices were affected seven times, and the bases five times when convulsions occurred. It appears to me to be probable that cerebral symptoms occur when large areas of the lung are involved, when the temperature is unusually high, or when the toxemia is specially intense. Convulsions occurring at the commencement of the disease, and taking the place of the rigor in the adult, do not seriously affect the prognosis. Far otherwise is it when convulsions occur late in the disease. My cases go to prove that they almost invariably herald an early and fatal issue. Convulsions rarely occurred in children over two years old. In older children, especially in cases between five and eleven years, cerebral symptoms manifested themselves chiefly by delirium, sometimes of a wild character, at others of a muttering type, and chiefly marked at night. Delirium was accompanied, as a rule, by muscular twitching, picking at the bed clothes, tremor of the tongue, and often by diarrhoea. These symptoms were associated with a widespread disease and a high temperature. In none of the twenty-five cases in which delirium occurred was

the child under three years old, and in the great majority he was between five and eleven years. In only two of the cases accompanied by this symptom did death occur. Herpes occurred sixteen times, or in about 11 per cent. of the cases; but this percentage cannot be relied upon, as the symptom was probably not always noted when present. It does not seem to accompany pneumonia in infants, as in only two of the cases in which it occurred was the child under six years of age. In no case when it was present did a fatal result supervene. In most of the cases over three years old the temperature remained high until the crisis, which, in the greatest number of cases, occurred on the eighth day. The various days on which the crisis took place are recorded on the annexed table:

On the second day in 2 cases			On the eighth day in 22 cases		
"	third	3	"	ninth	14
"	fourth	6	"	tenth	10
"	fifth	16	"	eleventh	7
"	sixth	15	"	twelfth	3
"	seventh	16	After the twelfth		
					6

Abortive cases generally terminated between the second and fourth days, while in cases of the creeping or wandering variety the crisis was frequently delayed beyond the twelfth day. In the most prolonged case of the series the crisis did not occur until the twentieth day. In children under 3 years the temperature is not so continuously high, and termination by crisis does not so frequently occur as in older children. In my 147 cases 24 terminated by lysis, which is, I think, a small proportion. The highest temperature recorded in any case was 107.5 F., but any temperature above 105 F. seems to add greatly to the gravity of the prognosis, and in the majority of my cases was followed by death.

The crisis was sometimes followed by perspiration and prostration, but in no case did dangerous collapse occur. A post-crisis supervened in 25 per cent. of the cases, and was generally of ephemeral duration, subsiding in about twelve hours. When the post-crisis rise continued for any length of time it generally indicated either the spread of the pneumonia to another lobe or the onset of empyema. The rate of the pulse has not the significance it assumes in the adult. It generally runs from 130 to 150, and may rise to 160 or 170 without seemingly affecting the prognosis. A rate exceeding 180 is, however, generally to be regarded as a grave condition. At first the pressure and tension of the pulse is high, but later it becomes less marked.

I wish to draw attention to a symptom which I have not seen referred to—namely, the extreme slowness and irregularity of the pulse sometimes present during convalescence, indicating the profound toxemic influence of the pneumococcus on the cardiac muscle. On two occasions a pulse-rate of 40 is noted, and it is quite a frequent occurrence to find it between 50 and 60 a minute—certainly a very low pulse-rate for a child. I have never, however, met with any case of sudden heart failure during convalescence in a child—not an uncommon occurrence in the adult. Empyema is the commonest of all complications, and should always be suspected when the temperature rises again after the crisis. It seems, as a rule, to follow pretty closely on the heels of the pneumonia, and I have found pus within two days of the crisis. The pus is generally of a thick, creamy consistence, and contains the pneumococcus in pure culture. It is not always such an easy matter to recognize an empyema in a child as may be supposed, for though the chest may be full of pus we may have loud tubular breathing over the effusion; and as the vocal fremitus in children is always unreliable, we have often to depend upon the exploring needle for our diagnosis. But even this method fails us at times.

I recall one case in which I suspected the presence of pus, and, though I passed a needle on nine different occasions, I was unsuccessful in obtaining it. As the child was rapidly getting weaker, I asked my surgical colleague to open the pleural cavity. As is his custom, when the child was placed on the operating table he inserted a large needle in three or four different places, and getting no pus he asked me if he should continue. So convinced did I feel of the presence of pus that I asked him to proceed with the operation, but anxiously watched the future progress. He removed a piece of rib and incised the pleura without getting any indication of pus. On inserting his finger, however, he discovered and pulled out a large

piece of thick lymph several inches square, and upon its removal a regular gush of pus took place, which put an end to my anxieties.

There seems to be a considerable amount of confusion and misconception regarding the prognosis of pneumonia in children, some authorities maintaining that the mortality is small, varying from 1 to 5 per cent., while others assert that it is a very fatal disease, the mortality being 27 per cent.

These discrepancies have doubtless arisen by statistics being drawn on the one hand from hospitals that do not admit infants, and on the other from foundling hospitals where only children under 2 years are treated. In the Edinburgh Sick Children's Hospital, where children of all ages are admitted, we are in a better position to form a just estimate of the true prognosis of the disease. Out of the 147 cases, 15 have died, being a mortality of 10.2 per cent. Under 2 years of age, of the 45 cases treated, 12 have died, a death-rate of 26.6 per cent. Above 2 years, of the 102 cases treated, 3 have died, giving a mortality of 2.9 per cent. My conclusions, therefore, are that below 2 years the prognosis is grave, but above that age it is exceedingly favourable.

Turning now from lobar pneumonia to bronchopneumonia, I find that there were in my series 353 cases of the latter variety of which 120 were primary and 233 secondary. The sexes appear to be equally susceptible, there being 182 boys and 171 girls. As in lobar pneumonia, winter and spring claim the great majority of victims, 225 cases having occurred during these seasons, as against 128 during the summer and autumn.

Of the 120 cases of primary pneumonia, 92 occurred in children under 2 years, 39 of them occurring under 1 year, the youngest case being in a baby 16 days old.

Primary bronchopneumonia commences as a rule suddenly, with high fever, quick breathing, and pain in the side. Vomiting was nearly as common a symptom as in lobar pneumonia, and convulsions and other cerebral symptoms occurred in a not inconsiderable number. In most of the cases the temperature at once assumed a high level, varying between 103° F. and 105° F., but was almost invariably characterized by morning remissions and evening exacerbations, the daily average fluctuations amounting to about 3° F. Although the morning remission was the rule, I have the records of quite a number of cases where the morning temperature regularly assumed a higher level than the evening. The duration of these cases varied from two or three to thirty-five days, but an examination of my records goes to prove that the average duration is very much shorter than that of the secondary forms of bronchopneumonia, and considerably longer than that of the lobar type, being about eleven days. In cases that ended fatally, death occurred on an average on the twelfth day. Relapses were much less frequent than in the secondary bronchopneumonias. The fever usually terminated by lysis, but in 13 of the 120 cases the temperature suddenly subsided by crisis.

The diagnosis of these cases was often difficult; the chief points by which I was guided in differentiating them from lobar pneumonia being (1) the more disseminated character of the lesion in the lung, (2) the remissions in the temperature, (3) the greater amount of cyanosis and dyspnoea, and (4) the more troublesome character of the cough.

Some of these cases of primary bronchopneumonia, especially when they occur in very young infants, are exceedingly puzzling. The temperature suddenly shoots up to 104° or 105°, the child becomes cyanosed and prostrate, his breathing is quick and shallow, and he appears to be suffering from a severe form of toxic poisoning. On examining the lungs we may only hear harsh puerile breathing, with few if any accompaniments, and there may be no change in the percussion note. The infant is dull, listless, and apathetic, and appears to be very seriously ill, when, after lasting one or two, or at the longest three, days, the temperature gradually subsides, the alarming symptoms rapidly disappear, and in a day or two the child is quite well again. I have been in the habit of regarding these cases as abortive cases of bronchopneumonia, chiefly on account of the great cyanosis and distress of the breathing, which is so characteristic of this condition, and but for which they might equally be regarded as abortive cases of lobar pneumonia. I think it is a pity that the

old-fashioned title of acute congestion of the lungs, which so excellently describes this condition, is so seldom used nowadays. It seems to me that a sudden and very intense congestion of the lining membrane of the smaller bronchi and air cells sufficiently explains the alarming symptoms which arise.

The remaining 233 cases, or nearly half of the whole series, were cases of secondary bronchopneumonia. The usual diseases to which these cases were secondary were bronchitis, measles, whooping-cough, influenza, diphtheria, and diarrhoea, and general debilitating conditions, such as atrophy, rickets, and tuberculosis.

An examination of the ages of these cases shows a marked contrast to those of the primary type, 92, or 42 per cent., being over 2 years old. After suffering from one or other of these conditions for periods varying from a week to several months the temperature suddenly runs up, the pulse-respiration ratio becomes markedly altered, the breathing becomes shallow and rapid, respiration ending with an expiratory moan, the child becomes cyanosed, and the cough more frequent and troublesome. These symptoms are, I think, sufficient to warrant us in diagnosing the onset of catarrhal pneumonia, without our being able to discover signs of consolidation in the lung. Such cases are often described as capillary bronchitis; but I have never been able to appreciate where capillary bronchitis ends and catarrhal pneumonia begins. Indeed, I go so far as to affirm that, whenever we are able to diagnose capillary bronchitis, we can confidently state at the same time that the child is also suffering from bronchopneumonia. The range of temperature in these cases during the whole course of the illness is of the remittent type. I was surprised, in analysing my cases, to find that, contrary to my general impression, it was unusual for the temperature to rise above 103° F. In only 99 cases of the series did it assume a higher range, the thermometer recording 104° F. in 59 cases, 105° F. in 30 cases, 106° F. in 8 cases, and 107° F. in only 2 cases. Frequent remissions occurred in most of the cases, followed by exacerbations, marking the spread of the inflammatory process to hitherto unaffected lobules. In the great majority of the cases the temperature fell by lysis. The duration of these cases is exceedingly variable, varying from two to ninety-five days, the fever dropping to normal in the average case about the twenty-third day.

Many of the cases, however, dragged on for many weeks, some of them for months, with a high temperature, interspersed with remissions, relapses, and exacerbations, the child growing daily weaker and more emaciated, creating the suspicion, nay almost the certainty, that the case was tuberculous, when quite unexpectedly the temperature would fall to normal, and the child made an uninterrupted recovery.

Another point to which I would like to draw attention is the fact that in not a few, especially in very young and debilitated infants, extensive pneumonias ran their course with scarcely any rise of temperature. In three atrophic infants the temperature actually remained subnormal throughout the entire course of extensive pneumonia. The pulse and respiration are always increased in frequency, the former varying, as a rule, from 140 to 180, and the latter from 40 to 80; and, as will be gathered from this statement, the pulse-respiration ratio is markedly altered. The alteration of the pulse-respiration ratio I regard as one of the earliest, and also as the most important sign of the onset of pneumonia.

I have made an analysis of the site of the lesion in the lungs in these 353 cases of bronchopneumonia, and find that both lungs were affected 182 times, or in considerably more than half the cases. The right lung alone was affected 83 times, the left lung alone 72 times, and in 16 the site was not stated in the report. The bases were affected five or six times more frequently than the apices.

The only three remarks I shall make with regard to physical signs are: First, that auscultation is a more reliable method of examination than percussion, as in many of the disseminated cases no percussion dullness can be ascertained; secondly, that the pneumonic patches are frequently of such small size that every inch of lung requires to be carefully auscultated to detect them; and thirdly, that, unlike cases of lobar pneumonia, resolution as a rule takes place very much more slowly, and is often a very tedious and long-drawn-out process. One attack of

bronchopneumonia seems to predispose to another, as there is a history of the child having suffered from one or more previous attacks in 49 cases. My experience leads me to give a serious prognosis when I see the cyanosis deepening, the distress and restlessness of the child increasing, the cough and the ability of the child to shed tears ceasing, the pulse becoming faster and weaker, and the child becoming drowsy and lethargic.

Death is generally due to one or other of the following causes: (1) Increasing exhaustion; (2) suffocation and toxæmia, owing to the bronchi becoming blocked and the lungs choked; and (3) to heart failure. Many of the long-drawn-out cases die either from tuberculosis or from fibroid induration of the lungs.

I wish to emphasize three points which have struck me in connexion with cases having a fatal termination: (1) The large number of cases which die very suddenly of heart failure—this occurred in 23 of my cases; (2) the tendency of the temperature to mount higher and higher as death approaches—this occurred 31 times in the 89 cases of death; and (3) the great liability of the child to die during an attack of general convulsions, which termination occurred ten times.

The prognosis of bronchopneumonia varies greatly according to the age and condition of the child, the amount of lung involved, and the nature of the infection, but the prognosis in any individual case is always an uncertain quantity. It is, perhaps, the disease of childhood which best justifies the expression that "while there is life there is hope," as the most unlikely cases, after lasting for months and giving rise to the gloomiest forebodings, often unexpectedly clear up, but, on the other hand, cases which appear to justify a hopeful prognosis sometimes die suddenly and quite unexpectedly of heart failure. The death-rate is always a heavy one. Thus, Keating gives the general death-rate at 49 per cent., and under 3 years at 75 per cent. Holt states the mortality of all cases as 65 per cent.; Morse puts it at 75 per cent., Pepper at from 30 to 50 per cent., and Donkin places it at about 50 per cent.

I am glad to say that an examination of my cases shows a very much smaller death-rate than those quoted. Out of the 353 cases 89 died, giving a mortality of 25.1 per cent., and if we exclude 15 cases which died within twenty-four hours of admission the death-rate is reduced to 20.9 per cent.

An analysis of all the cases of bronchopneumonia treated in the Sick Children's Hospital during the last ten years gives a mortality of 28 per cent. It is evident that a great discrepancy exists between these statistics, and I am at a loss to explain the cause. At first it occurred to me that the very much higher American death-rate might be accounted for by more of the cases being secondary to whooping-cough and measles, or by being complicated by severe forms of summer diarrhoea, but on examining Holt's statistics more closely I see this is not the case, as he gives the mortality of the cases following bronchitis at 65.5 per cent. It certainly cannot be attributed to the better general condition of the cases treated in our hospital, for they are drawn from the poorest classes, and the great majority of the children are underfed, atrophic, or rickety. The only conclusions I can arrive at to account for the disparity are that it is due either to the different climatic conditions or that the infection is of a more virulent type.

I find that the death-rate was considerably less among the primary than among the secondary cases.

The following are the chief points to be considered in estimating the prognosis:

1. The primary disease; those cases which occurred after whooping-cough, measles, and diphtheria were exceedingly fatal, giving a death-rate of 54.4 per cent.
2. The younger the child the worse the prognosis; 64.4 per cent. of the deaths occurred in children under two years.
3. The greater the extent of the lungs involved the less is the chance of recovery. In 73 per cent. of the deaths both lungs were affected.
4. The poorer the previous health of the child the graver the prognosis. I have not made an analysis on this point, but in the great majority of deaths the children were wretchedly nourished and developed.
5. When the child suffers from severe forms of rickets

the prognosis is always grave. In 34 per cent. of the deaths the report states that severe rickets existed.

6. Diarrhoea seriously affects the prognosis. This condition was present in 24 per cent. of the deaths.

7. A temperature of 105° F. adds greatly to the gravity of the case; 60 per cent. of these cases died.

8. A pulse-rate over 180 and a pulse-respiration ratio of 1 to 2 or 1 to 1½ is always serious.

9. The highest mortality was in the cases which had lasted for several weeks.

I know of no disease of childhood where treatment judiciously carried out is more efficacious than in bronchopneumonia. By avoiding, on the one hand, unnecessary interference with Nature's efforts to bring about a natural cure, and by stepping in and giving judicious assistance and help when these natural efforts require stimulation, very much can be done to tide the child safely through the many dangers and sudden emergencies to which he is liable.

The chief aim is to put the child into the best position to withstand the disease, and the indications for treatment are, therefore: First, to endeavour to prevent the digestive organs becoming deranged; secondly, to do all in our power to maintain the strength and vitality of the patient; thirdly, to keep the action of the heart constantly under observation; and, fourthly, to prevent, if possible, the spread of the inflammation to fresh portions of lung.

A supply of pure air is one of the most important considerations, as one of the main causes of death is carbonic acid poisoning. All my cases have been treated in a large airy ward, kept at a uniform temperature, with abundant cubic space and cross-ventilation. I consider the system of treating these cases in the open air undesirable, as I cannot believe that the inhalation of a cold damp raw air acts beneficially upon an acutely-inflamed mucous membrane.

The feeding was carefully attended to and the bowels regulated so as to try and avoid diarrhoea and flatulent distension, which adds so greatly to the respiratory embarrassment. The diet consisted chiefly of milk modified to suit the age and requirements of the child, chicken and mutton broths, and, if the child could digest them, simple milk puddings. Frequent draughts of cold water were always allowed. The clothing consisted of a gamgee tissue jacket and a loose flannel nightdress, so as to allow the movement of the chest walls to be absolutely free and unimpeded.

My practice has been to surround the upper half of the crib with a tent, open in front so as to permit the free access of air. Inside the tent are hung towels wrung out of a solution of 1 part of eucalyptus oil to 5 parts of water. I am convinced that the evaporation of the moisture and the volatilization of the oil has a soothing effect on the inflamed mucous membrane and greatly diminishes the cough, and it is possible that its antiseptic action may tend to prevent the spread of the pneumonia to fresh portions of lung. I instruct the nurse to change the position of the child in its crib frequently, and from time to time to take it up and carry it up and down the ward, to avoid the risk of hypostatic congestion.

I am a believer in the application of lightly-made jacket poultices made of one part of mustard to four or five of linseed meal, and applied three or four times a day for periods of a few minutes at a time. They redden the part, bringing the blood to the surface, act as a stimulant, relieve pain, and seem to be comforting to the child. I generally continue these applications for a week or ten days, or longer if required, at the commencement of an attack of pneumonia. In spite of much that has been said and written to the contrary, I consider that alcohol is the drug upon which we have principally to depend in the treatment of bronchopneumonia. I believe that the great majority of cases require alcoholic stimulation. As a rule it is unwise to commence giving alcohol early in the disease, but it should be reserved until symptoms indicating prostration and lowering of the vital powers manifest themselves. When such symptoms arise alcohol frequently acts like a charm, strengthening and steadying the pulse, lessening the cyanosis, reducing the temperature, increasing the vitality, and, most valuable of all, procuring a much needed sleep. A few years ago, on the advice of a friend whose opinion I valued highly, I discontinued for some time the adminis-

tration of alcohol—an experiment I have never failed to regret, as my results were not nearly so satisfactory during that period. I give whisky in doses of 15, 30, or 40 minims, according to the age and condition of the child, every two or four hours, and once its administration is commenced I generally find it necessary to continue it until convalescence is established. In all cases I give strychnine in minim doses of the liquor every four hours as a routine practice. It acts as a cardiac and respiratory stimulant, as a stomachic and nerve tonic, and it seems to help the contraction of the muscular walls of the bronchi and thus assist the expulsion of mucus. The only cases in which I omit the strychnine are when diarrhoea occurs and when there is marked respiratory stridor.

When signs of advancing cardiac strain make their appearance, evidenced by irregularity of the pulse and increasing cyanosis, to the strychnine I add 1-minim doses of 1885 tincture of strophanthus, finding by experience that strychnine and strophanthus better maintain the blood pressure than digitalis, and they have no injurious effect on gastric digestion. I am a firm believer in the efficacy of the inhalation of oxygen, and have frequently been amazed at the improvement in the child's condition which followed its administration, the breathing and colour improving and the somnolence and cyanosis becoming much less marked. I regard it as a valuable remedy in the treatment of these symptoms. The only means I advocate for the reduction of high temperature is tepid sponging and the cold pack. When sudden collapse of a portion of the lung sets in, I rely upon free alcoholic stimulation and placing the child in a mustard bath. I have entirely given up the administration of cough mixtures, having seen little good result from them, and consider that they are very liable to upset the child's digestion, which it is so important should be kept undisturbed. When the bronchi are choked with secretion and the child is strong, an emetic of ipecacuhana often gives great relief, and, failing that, I have occasionally seen exceedingly good results from the administration of belladonna.

During convalescence, nourishing food, small quantities of good old port wine, quinine, cod-liver oil, and change of air are the methods of treatment on which I place most reliance.

I conclude this paper as I commenced—by remarking that though I have not wandered very far from the beaten track, I have endeavoured to state my own impressions without referring to the work of others, and to give the results of my own experience for what they are worth.

THE PHYSIOLOGICAL ANTAGONISM BETWEEN ACONITE AND BELLADONNA.

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COMPLETE physiological antagonism amongst drugs, especially alkaloids, is well known to be extremely rare. The following case, which came recently under my observation, seems worthy of record, in that it illustrates the partial antagonism existing between aconite and belladonna, as well as from the fact that the patient recovered after an enormous overdose of both these drugs taken simultaneously:

J. Y., aged 48, suffering from a subacute attack of pleurisy, was recommended by me to take an expectorant mixture and at the same time was given a 6 oz. bottle of aconite, belladonna, and chloroform liniment for external application to the chest. The liniment was dispensed in a blue bottle, labelled poison, but nevertheless the patient on the following morning swallowed by mistake $\frac{1}{2}$ oz. of the wrong bottle. He immediately recognized his error, but did not realize its seriousness. About four hours after its commission his relatives became alarmed and hurriedly summoned me to attend him.

I saw him about four and a half hours after the poison had been swallowed. Apart from copious libations of warm water, no emetics had been taken, and consequently the full $\frac{1}{2}$ oz. of liniment had been absorbed.

The patient was in a state of active delirium, his pupils so widely dilated that only a fine rim of iris was visible. At times he became exceedingly violent, and struck out at those who were vainly attempting to restrain him. He kept constantly muttering and grasping at objects within reach, totally mistaking their identity. At one time he would become violently maniacal, and a few moments later would subside into fits of unrestrained mirth. The pulse was rapid, about 180, small, and thready, and during a violent paroxysm became undiscernible

at the wrist. The tongue was dry and parched, the lips pale and slightly cyanotic, while sordes accumulated at the angles of the mouth. Malar flushing, a characteristic sign of belladonna poisoning, was, however, absent. On the patient endeavouring to stand, distinct muscular weakness was apparent; his legs appeared to give way and he required to be supported. A fine fibrillary tremor was present in the muscles of both extremities. He talked irrationally, and being offered stimulant, clenched his teeth and refused.

Altogether the patient seemed to be approaching a condition of very grave collapse, and, under the circumstances, I did not feel induced to administer morphine, the usual antidote in belladonna poisoning. The stomach tube was also too late to be of service. I injected 10 m liq. strychn. hyd., and repeated the dose in ten minutes. A saline enema, Oij, with $\frac{3}{4}$ ij brandy, was quickly administered, and a hot mustard poultice applied to the precordia.

These measures seemed to bring about some improvement, as the pulse was reduced in frequency and its volume increased. He, however, continued restless, and at times violent, and I decided to remove him to hospital. Here morphine ($\frac{1}{4}$ grain) was immediately administered, with the result that his violence gradually became subdued. Consciousness returned at about 11 p.m. the same day—exactly twelve hours after the ingestion of the poison. With the exception of retention of urine, which lasted during the next day, and a continued pupillary dilatation lasting for six days, the patient soon completely recovered.

It would appear that the patient took 53.3 grains of aconite root, which is at least twenty-seven times a safe dose. It represents $\frac{1}{4}$ grain aconitine, of which $\frac{1}{8}$ grain has been known to be fatal. He also swallowed 40 m ext. belladonnae liq., which is equal to 0.3 grain of the total alkaloids. This would represent something like thirty times the official dose of atropine. Recoveries, however, have been reported after larger doses of this alkaloid, from $3\frac{1}{2}$ to $5\frac{1}{2}$ grains having been taken without a lethal termination. Of chloroform he took 40 m, about eight times the official dose.

The interest of the case lies in the fact that the lethal effect of a large dose of aconite was abolished by the action of a large dose of belladonna. Muscular weakness, numbing of the extremities, and the tendency to complete collapse were all the purely aconite symptoms to be observed. Salivation, usually present in aconite poisoning, was absent, while the usually contracted pupil was lost in the powerful dilating influence of atropine. Finally, the intensely depressant action of aconite on the central nervous system was counteracted by the more stimulating action of belladonna.

The obvious lesson to be drawn is the great value which should be attached to hypodermic injections of atropine in all cases of aconite poisoning, and one can hardly avoid a word of tribute to the wisdom of the framers of our *Pharmacopoeia* in compounding pharmacological antidotes in many of their formulae.

A NEW APPARATUS FOR CHLOROFORM ANAESTHESIA.

By N. H. ALCOCK, M.D.

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It has been evident for some time past that the open method of chloroform anaesthesia, although almost universally adopted, is attended by the disadvantages that are inseparable from the administration of an active drug in unmeasured quantity. That untoward consequences are not more numerous is a tribute to the skill of the anaesthetist, but even the best intentions are sometimes unequal to the very difficult task of supplementing the deficiencies of an unsatisfactory method, and "accidents" such as are recorded in the newspapers emphasize the necessity of a more exact method of administration.

The first step in such a method is to construct an apparatus that shall deliver a known admixture of chloroform vapour and air. Several machines have been already made by Dubois, Vernon-Harcourt, Waller, Collingwood and others, and the result of the work of these experimenters has been of very great assistance in the present research. I would particularly express my indebtedness to the densimetric method of Waller and Geets, without which the construction and testing of this apparatus would have presented even greater difficulties than were met with.

The apparatus consists of a circular copper vessel 5 in. in diameter and $4\frac{1}{2}$ in. deep, which contains 150 c.cm. of