CEREBRAL SIGNS AND SIMULATIONS IN PNEUMONIA.

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It is probable that the various papers on the different inflammatory diseases of the lungs, to be presented by this Committee, will take into account the cerebral complications and counterfeits often present, as well as other more frequent, if less serious, complications; and perhaps so fully that anything more would be superfluous. Still, the relative frequency and importance of these cerebral manifestations, grown more frequent and more important since the Russian influenza added its baleful quota to the list of human ills, gives good reason for something of repetition. Indeed, when different writers discuss the subdivisions of lung inflammations practically, that is from the standpoint of bedside observation, there will be much of repetition all through, for at the bedside we find few of those distinct entities which the text-books delight to photograph and catalogue, but instead, a mixture of conditions which baffle rather than invite either delineation or classification.

Again, contrary to what was first confidently expected from the disclosures of bacteriology, the discovery of the specific germ of pneumonia, far from conferring upon that disease well-defined and easily known boundaries, actually added to their vagueness and uncertainty, so that what the culture tube and microscope might determine to be a pneumonia might nevertheless be proven by the stethoscope and percussion hammer to be a pleuritis or a pericarditis or, perhaps, all three.

The co-existence of these or other lesions as the result of pneumonic infection is always of great clinical interest, but its discovery is not always of vital importance, judged by its practical application to treatment. When, however, this infection invades cerebral or meningeal structures, or, originating there, invades later the lung, the case is different, for now it is of vital concern to make out the graver condition, to the end that appropriate changes in treatment may be made, and in time.

What determines this invasion of regions anatomically so remote and dissimilar? So far the question does not seem to have been definitely answered. Fränkel, Weichselbaum and others have found the diplococcus of pneumonia within the membranes and ventricles of the brain coincidently with an attack of pneumonia, and also when that disease was not present. Similarly the influenza bacillus of Pfeiffer and the pneumococcus of Friedlander have been found in those situations, and, to further illustrate the neighborly relations in misfortune of the brain and lungs, cerebro-spinal meningitis is often followed by pneumonitis, the specific germ of the former being found in the lungs.

Perhaps, however, it should be said that the common germ of both is found in the lungs after the previous invasion of the membranes of the brain or spinal cord, since it looks the same, whether found in one inflammation or the other. So, also, the same mixed infections may be present when these inflammations coexist, as by any of the pyococci.

Bacteriology has thus thrown a flood of light upon what was formerly looked upon as the strangest co-

incidence of distinct diseases, and reduced to law the most erratic vagaries of morbid processes. Now we expect that with every epidemic of cerebro-spinal fever, some—few or many—cases of lung complications will occur, and, *vice versa*, that a generally prevalent pneumonic fever will have its quota of brain cases, and that each, in epidemic or endemic form, will be haunted by the independent presence of the other.

The existence, then, of these mutual relations being known, it would be naturally inferred that they could be promptly recognized when present, but actual experience shows that this is not always easy to do. With young children especially, some of the most striking symptoms are common to both, nor are they necessarily different in character or degree. This is particularly true of fever, headache, vomiting and delirium, and it will often be true of the pulse, respiration and certain nervous signs, like the contracted or dilated pupil, regional hyperesthesias, and the like. As for temperature, while it would be expected to run higher in meningeal inflammations, it is not uncommon for it to remain relatively low throughout. Thus, the temperature of uncomplicated pneumonia often reaches 105 degrees or higher, while a case complicated by meningitis may show no higher range than 102 or 103. Nor will sudden variations make the discrimination any easier, for a rapid rise from 102 to 105 degrees may, and usually does, come from those still mysterious factors in the production and dissipation of heat supplied by the specific toxinaemia of the pneumonic inflammation, or, again, from the septic effects of pyogenic bacteria. Since the same chronic causes are operative in cerebral complications it follows that a feeble toxinaemia would be attended by a moderate temperature, always excepting the direct irritating effects of an inflammation involving heat centers, or pressure effects of large effusions upon the medulla.

Headache is often a prominent symptom in pneumonia with or without complication, but I have seen no case where it afforded help in making a discriminating diagnosis. Sometimes the restlessness and violence of the patient will be attributed to intense headache, but it is very rarely that he complains specifically of this, as is often done in cerebro spinal fever. It has seemed to me that other regional neuralgias and hyperesthesias have more diagnostic value than this, but they also have their perplexities, for, occurring in the chest, they may be explained by the presence of the existing inflammation, and it is only when this last is deep-seated in the insensitive region of the interior of the lung, that it can be eliminated as a cause of pain. Occurring in the extremities they are far more significant, and when is superadded tonic or clonic spasm it is made tolerably certain that the membranes of the cord, at least, are involved in the morbid process, although it must not be forgotten that these, too, are sometimes present when, at most, a meningitis can be only suspected.

As an early symptom in pneumonia, vomiting is present in from 30 to 60 per cent. of cases, according to the experience of different observers. Exceptionally it continues throughout the disease. By itself, therefore, it is only in such cases that it could justly arouse the suspicion of meningeal complications. There is a peculiar explosive vomiting, or a

passive regurgitation, without previous or attendant nausea, which is believed to come from a central irritation through the vagus; but this, too, may be seen when the cerebral structures are intact, so far as can be judged by attending symptoms. Correspondingly, the nausea and vomiting characteristic of gastric irritation as commonly observed may be the stomach symptom of a well-marked meningitis.

Certain forms of delirium, particularly the violent kind occurring before the last days of the disease, or the half-stupor which seems to observe without taking cognizance of the sights and sounds of the sick-room, afford a strong presumption of brain trouble, but they are not proof of this, since they occur fairly often without it. But with it the delirium may be quiet and unalarming till it verges to stupor and unconsciousness.

By itself, the pulse does not afford a criterion by which to judge, unless extreme rapidity be replaced by abnormal slowness. This would be fairly pathognomonic of intracranial pressure, unless it could be explained by some cardiac depressant administered in treatment. But it goes without saying that the significance of all symptoms present should be read in full remembrance of remedies employed and their natural results.

A rapid, more or less cordy pulse, characteristic of serous inflammations is apt to be present, but the pulse of resistance that goes with parenchymatous inflammations may not always be easily known from it. In general, it may be said that, with the exception noted, the pulse gives but little aid in making a discriminating diagnosis.

Usually the respiration has greater significance than the signs and symptoms already mentioned. It may be alike rapid and fairly even in both simple and complicated pneumonia, but irregularity, particularly of the Cheyne-Stokes character, and abnormal slowness, or both, point strongly to a crippled medulla, and a sudden cessation of breathing, from a basal pressure, may all too late give the first positive evidence of a brain complication.

For me the pupillary signs are the most positive, excepting, possibly, the muscular, afforded in the whole history of the case. Exceptionally, without cerebral lesion the little patient's eyes may be bright and glistening, intolerant of light, irregular of pupil—which may be contracted or widely dilated, and as some affirm, the eyes may be distinctly squinted; but such an array of signs should point to the brain, and if the pupils be of unequal size, or insensitive to light, or momentarily contracting to dilate widely in the presence of strong light, then, in my opinion, the evidence should be considered as well-nigh complete.

Reverting to muscular signs, it should be stated that clonic spasms early in the sickness are usually not indicative of nervous lesions. They are regarded by the older writers as having a like significance to the chill in older patients. Now, however, it would be more rational to attribute them to the irritating effects of microbic toxins upon the appropriate motor centers. When we remember how mixed the pulmonary infection may be, how Pfeiffer's bacillus of influenza may reinforce Fränkel's or Friedlander's pneumococcus, or both, and how one or more of the pyococci may join this coalition against the health and life of the little patient, it is not strange that this mixed intoxication should have varied and

alarming effects upon the sensitive brain and cord, nor is it strange that they should not always be easy of analysis. Tonic rigidity of the nuchal muscles and those of the back and extremities has long been regarded as significant of meningeal inflammation, and it would be well to regard it as a positive sign. But if to this be added the eye symptoms already spoken of, or the characteristic vomiting without nausea, the diagnosis would admit of no doubt whatever.

Auditory symptoms will sometimes be available when others are absent or few. There may be a hypersensitive hearing, or impairment or loss of this sense. In the few cases I have seen where either of these has been present it has been the former. It should probably have the lesser diagnostic meaning, as it may doubtless occur when neither the center of hearing nor the auditory nerve in any portion of its course is affected.

The middle ear, as an *atrium infectionis*, should not be lost sight of. Especially in the broncho-pneumonia of influenza, though other forms do not always leave the tube and middle ear inviolate, the danger of tympanic inflammation is very great; and, once present, it involves all the risks to adjacent structures which are known to attend such diseases from commoner causes. A resulting thrombo-phlebitis and following abscess might show focal signs according to its location and the extent of pressure.

Focal symptoms might also supervene upon the occurrence of embolism, depending upon the region made anemic by the arterial plug. Remembering the hyperinosis usually present in croupous pneumonia, and that clot is not an infrequent incident, should a local paralysis suddenly occur it would be safe to assume that the corresponding cerebral circulation had been occluded by clot. This is all the more certain if, as usual, it happens before the decline of the inflammation, for the brain sequels of middle ear diseases are of later origin.

In my experience, meningitis is more apt to recur as a complication in epidemic than in epidemic or sporadic pneumonia; but this is limited, being based upon only two instances. In the first, of three cases occurring in one family two died of meningitis, well-marked symptoms presenting after more or less extensive lobar exudation. In another family, living but a few rods away, out of two cases, one died of meningitis. All of these, and some dozen more of simple pneumonia in the same neighborhood, were nearly cotemporaneous. Neighboring towns had few or no cases.

Four or five years ago, also, Neenah had a visitation of mild pneumonia, and out of the cases I saw two had most of the signs characteristic of meningitis, and two or three others many of them. No deaths occurred, but in the two I could not avoid a diagnosis of brain complication.

This experience is much too limited to generalize from, but it will lead me to look for like coincidences in future epidemics, and, if nothing more, to greater alertness in the discovery and comprehension of early cerebral signs.

From what has been said, a further point of practical interest might be inferred, but it will not be amiss to speak of it. Not only the simulations, but the signs likewise, of pneumonic fever, will sometimes mislead; for as it happens not seldom that a distinct limited exudation may be positively made out

one day to be positively and forever lost the next, so it may happen that slight pulmonary sounds may be attended by alarming cerebral signs, and all replaced in a day or two by evidences of the most extensive lobar exudation. In such cases it is well for the attending physician if the consultant see them before the miraculous transformation, if at all, for he will thereby be saved some mortification of spirit, although the latter will miss the glory of finding out, or seeming to find out, what his colleague had blindly overlooked.

IS MEMBRANOUS CROUP DIPHTHERIA?

Read before the Indiana State Medical Society at Indianapolis, May 17, 1894.

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Notwithstanding the investigations which this question has provoked during the last decade, there is still quite a diversity of opinion concerning the identity of these two affections, and considerable time must yet elapse ere a positive answer may be given to the query which forms the title of this paper.

The desire of the writer is to stimulate an interest upon this subject in the Society, and to bring about a thorough discussion upon it, believing, as he does, that a free interchange of ideas and experiences is one method by which an elucidation of the problem may be brought about.

In the past few years the bacteriologist and microscopist have stepped prominently to the foreground, and to their indefatigable labors and their valuable investigations and experimentation are we indebted for the marvelously rapid strides forward which medical science has taken. Our ideas of the etiology of disease have radically changed, and in consequence of our increased knowledge in this direction our methods of preventing and treating disease have become much more effective.

Through these bacteriologic investigations the discussion has arisen concerning the identity of membranous croup and diphtheria, and although the investigations in this matter may be said to be yet in their infancy, they have proceeded sufficiently far to convince many of the workers that the two diseases are one and the same, viewed from an etiologic standpoint.

As I stated, however, these investigations have but recently been commenced, and in this controversy I fear that we have depended too largely upon the statements of our friends, the bacteriologists, and paid insufficient attention to those men who see disease at the bedside as well as in the laboratory. The opinions of intelligent men who are daily in contact with the clinical manifestations of disease are certainly of great importance.

In treating this subject, I shall commence with the consideration of the etiology of diphtheria, as it is generally accepted at the present time. Without burdening you with a discussion of the predisposing causes, with which you are all familiar, I will proceed at once to the exciting cause which is contagion.

The disease is now known to be transmitted by a specific organism, and this is known as the Klebs-Löffler bacillus, which was first described by Klebs and afterwards more extensively studied by Löffler. The production of the false membrane is the primary

or local result, while the secondary or constitutional symptoms are due to the absorption of the poison generated by the Klebs-Löffler bacillus.

In the examination of the diphtheritic membrane, however, it has not uncommonly occurred that the characteristic bacillus was absent, and only streptococci, staphylococci, and at times other cocci were present.

The streptococcus, staphylococcus, etc., were also found in those cases, wherein the Klebs-Löffler bacillus existed, and in these cases the attacks were almost invariably more severe than when these cocci were absent. This fact has been accounted for by the supposition that these cocci penetrated parts that the Klebs-Löffler bacillus did not reach, it being confined entirely to the membrane.

Baginsky, in a paper recently read before the Berlin Medical Society gave the following statistics: Out of 154 cases of diphtheria he obtained cultures of the Klebs-Löffler bacillus in 118 instances. In the remaining thirty-six cases the streptococcus and staphylococcus alone were found; of this latter class, thirty-two of the cases terminated in complete recovery, unhindered by any complications.

From these observations, Baginsky is strongly inclined to the opinion that there occur two separate and distinct forms of diphtheria; a true diphtheria having for its cause the Klebs-Löffler bacillus; and a pseudo-diphtheria, in which streptococci and other cocci are the etiologic factors. Dr. T. M. Prudden, of New York, publishes an account of his investigations, which comprises twenty-four cases. A vast majority of these cases occurred as a complication or sequel to scarlatina, and in none was he able to demonstrate the presence of the Klebs-Löffler bacillus. Martin publishes an account of the examination of 200 cases, in 79 of which the characteristic bacillus was absent, and in this latter class the mortality was very markedly lower than in those cases characterized by the presence of this bacillus.

Dr. Wm. H. Park, of New York, states that in 127 cases in which the Klebs-Löffler bacillus was found to be present, the mortality was 34.2 per cent. while in 79 cases in which it was absent, the mortality was but 4.3 per cent. Park also says: "The inflammation caused by the streptococci and to a less extent by other cocci, differ greatly. They may be conveniently divided into:

1. Pseudo-membranous angina.
2. Pseudo-membranous laryngitis.
3. Croupous tonsillitis.
4. Follicular tonsillitis.
5. Acute pharyngitis and tonsillitis without exudate.
6. Cases similar to all the preceding, but complicating infectious diseases."

From the above facts I would glean the following points, the correctness of which is accepted by many.

1. That those cases which we have heretofore diagnosed as diphtheria are divided into two classes, one of which constitutes true diphtheria, the etiologic factor of which is the Klebs-Löffler bacillus, while the other class consists of cases of exudative tonsillitis or pharyngitis, caused by the presence of streptococci, staphylococci, etc.
2. That true diphtheria is a disease attended with a very high death rate, the severity of the affection

¹ Annual of the Universal Medical Sciences, 1893, Vol. 1.