

who survive the initial disease is not established; diffuse fibroid degeneration in the media would affect unfavorably the nutrition of the intima and lead to the early development of general arteriosclerosis or to atheromatous plaques and possibly aneurysm. The frequency of palpable arteries after typhoid fever, previously mentioned, would thus be explained. The findings of Flexner in typhoid fever, and of Wiesel in many infections, are practically identical and seem to point to a uniform primary medial degeneration in these diseases and in certain other intoxications not of bacterial origin.

The growth in knowledge of arteritis in the young, from the early gross observations of rare and accidental instances up to recent systematic and refined study, may, it seems, prove of distinct assistance in our comprehension of early developments in adult life. Whatever the arterial sequels of the acute infections may prove to be, their effect must be intensified by the commonly recognized causes, and these factors combined would develop early and abnormal expressions of degeneration, termed presenile, the primary etiology of some of which has never appeared entirely clear and satisfactory.

LACK OF GASTRIC MUCUS (AMYXORRHOEA GASTRICA) AND ITS RELATION TO HYPERACIDITY AND GASTRIC ULCER.

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OF late a number of articles have been published which deal with the physiological and pathological secretion of gastric mucus and have given a fresh impetus to the long-neglected study of these conditions. I wish to report some of my own observations on this subject which will show that the secretion of gastric mucus is a necessary function, and that lack of mucus leads to serious disturbances. For several years I have made notes on the amount of mucus found in stomach contents. Of the interesting results thereby obtained, I shall confine myself to describing a frequent finding, that is, a complete lack of mucus in the gastric contents, a condition which is pathological and which has not been described heretofore.

The reason that this condition has not been recognized is that, as a rule, the contents of a fasting stomach, gained by expression or lavage, are used for the clinical examination of mucus. In this way, however, we can only detect an abnormal increase of mucus. Although it seems that in the stomach mucus is constantly secreted to some extent, its secretion is principally connected with the digestive period. The wash water of a fasting normal stomach does not show any mucus or only small quantities. Whenever we find, in

the fasting stomach, such quantities of mucus as are easily accessible for examination, we are dealing with a pathological increase. Under these conditions it is obvious that we cannot expect to gain much information about a decrease or lack of gastric mucus by examining the fasting stomach. However, I could not fail to recognize this condition of lack of mucus when I began systematically to examine gastric contents for mucus after test meals. In doing so I easily became convinced that stomach contents under normal conditions show a moderate amount of mucus after meals and that lack of mucus must, therefore, be considered abnormal. I have examined gastric contents for mucus in several thousand cases, always proceeding in the same routine way. In each case the contents were examined, macroscopically and microscopically, immediately after their removal from the stomach, one hour after a test breakfast of tea and toast had been given. Without going into statistics, I wish to give some general results derived from these observations.

RECOGNITION OF MUCUS. The presence of mucus is recognized by its most characteristic quality, its coherency. Such is the case with the moderate quantities of mucus which under normal conditions are found thoroughly mixed with the bread after a test breakfast. The bread more or less finely divided, according to the degree of mastication and the degree of digestion by the gastric juice, is held together by the mucus. In pouring the stomach contents from a glass to a filter, it flows out somewhat coherently. This coherency is more pronounced when the mucus is pathologically increased, and yet it is sufficiently distinct with normal quantities of mucus to make the latter clearly visible. When no mucus is present we miss this coherency of the bread mass; the bread particles are distinctly separated, both in cases in which the bread is divided into fine particles and in cases in which it is hardly changed, and is still present in larger pieces. The lack of mucus, however, is most obvious when we find the bread very finely divided and forming a flour-like layer. The absence of mucus in the latter cases is clearly demonstrated by the ease with which these fine bread particles move about, as also by the clear fluid which quickly forms as a separate layer on top of the bread. In cases in which a great deal of mucus is secreted, this separate layer of fluid shows a distinct, slimy character. The less mucus present, the more easily and the more quickly the fluid passes through the filter and the remaining bread mass on the filter shows distinctly, whether it is mixed with mucus or not.

The presence or absence of mucus should be corroborated by the microscopic findings, which are very characteristic. When gastric mucus is present, the different elements of the stomach contents, that is, starch globules, gluten, yeast bacteria, cells, etc., are bound together by the coherent mucus into smaller or larger clumps. The mucus is recognized by numerous myelin drops which are thoroughly mixed with the above-named elements. Even in places where these ele-

ments are more separate, numerous myelin drops are found and give the whole picture a blurred appearance. Under certain conditions the mucus appears fibrillar or in the form of spirals. When no mucus is present no myelin drops can be seen, the field is absolutely clear everywhere, and the different elements, such as starch globules, gluten, yeast, etc., not kept together by the binding mucus, appear singly and are each sharply outlined—a marked contrast to the picture first described. This characteristic picture of sharply outlined bodies in a clear field is most pronounced in cases of hyperacidity, in which the gluten of the bread has been dissolved and digested, and in which all the starch and yeast globules have thus been liberated. It does not, however, depend on the digestion of the gluten, for in cases of achylia gastrica the gluten itself appears sharply outlined in a clear field, provided no mucus is present.

I have tried to demonstrate the myelin drops more distinctly by the use of sudan, which stains them more or less red, according to the amount of fat which the myelin contains. However, the peculiar dull lustre of the unstained myelin is so characteristic that there is no advantage in staining with sudan. Much more characteristic pictures are obtained by staining the specimen with diluted Lugol's solution. The Lugol stains the starch blue, the yeast yellow, thus showing these bodies in sharp contrast to the rest of the specimen. It does not mix with mucus and, therefore, we find in specimens in which mucus is present only such starch globules stained blue that are free or on the edges of the mucus, while those that are incorporated in the mucus are left unstained. When no mucus is present, the Lugol reaches every single starch globule in the specimen. The distinct appearance of these deeply stained and sharply outlined bodies in a clear field gives a most typical picture, which in itself is characteristic of the absence of mucus. From the combined result of both macroscopic and microscopic examinations, we can thus easily determine the amount of gastric mucus present.¹ Although we have no quantitative method of measuring the amount, experience teaches us to estimate it and thus we find, under normal conditions, a moderate amount of gastric mucus in the breakfast. Under pathological conditions the mucus is either increased, decreased, or absent. The amount of mucus does not go hand in hand with the amount of gastric juice. In certain cases of achylia gastrica we see increased quantities of mucus and in others no mucus

¹ I wish to mention here, that this gastric mucus can be differentiated from mucus which originates from the upper air passages. Mucus which has been swallowed is never so thoroughly and evenly mixed with the stomach contents as the mucus secreted by the stomach and is, therefore as a rule, easily recognized as a foreign body and often identified microscopically by the finding of cells from the throat and pigmented alveolar epithelium, which it carries along. When epithelial cells are found in the gastric mucus they are of a different type (cylindrical); furthermore, they are usually found only in cases of gastritis together with leukocytes. In the gastric mucus which is secreted under normal conditions, as described above, I have rarely seen any cellular elements.

at all. On the other hand, although cases of hyperacidity as a rule show a lack of mucus, we observe cases of hyperacidity with increased mucus (gastritis acida). In fact we meet with various combinations of mucus and gastric juice. I shall not discuss these questions any further, but shall limit my remarks to the lack of mucus.

LACK OF MUCUS. In my examinations after a test breakfast, I have noted a complete lack of mucus in a great number of cases, it being more frequent than an increase. The lack of mucus may be due to different causes: there is either an insufficient secretion, a condition which could be named amyorrhoea gastrica, or the secreted mucus is digested, or it is dissolved by the action of bacteria, as we see happen in cases of bronchoblenorrhoea. A certain amount of mucus is possibly digested by an active gastric juice. However, certain considerations speak against the probability, that this complete lack of mucus in the breakfast is entirely due to its being digested.² First among these is the fact, which I have just mentioned, that lack of mucus occurs with varying degrees of acidity and even in cases of achylia gastrica, in which the absence of gastric juice excludes mucus digestion, while on the other hand, an increase of mucus is found in certain cases of hyperacidity with a very active gastric juice (gastritis acida). Another argument is furnished by the following observation: In a number of cases of hyperacidity, which at first showed complete lack of mucus, I found at subsequent examinations, after treatment with silver nitrate, that distinct quantities of mucus were present while the acidity remained unchanged. Therefore, the lack of mucus before the treatment cannot be explained as being due to digestion, otherwise it would be difficult to understand why after the treatment with the same active gastric juice mucus should be present.

No matter what the cause of the lack of mucus, whether it is due to diminished secretion or whether it is dissolved and digested after secretion, I think we are justified in concluding that the lining of the stomach is not well covered with mucus when we find none in the gastric contents after a test breakfast. It is at once obvious what this means. The mucus is the protective agent of the gastric mucosa. In cases in which no gastric juice or gastric juice of low acidity is secreted, it may not be of great importance whether the mucosa is covered by a thick or a thin layer of mucus. This is altogether different in cases with an active gastric juice, especially

² I have said nothing about dissolved mucus, because we are only dealing with mucus which is visible as such both macroscopically and microscopically. Mucus which cannot be recognized as such does not act as mucus any more and, therefore, has lost its proper significance as a protective agent, which we shall see later is one of its chief functions. Furthermore, A. Schmidt (Volkman's Vorträge, N. S., Nr. 202, p. 1131) has justly pointed out that mucus once dissolved in the stomach is readily digested and that the common tests for it, such as the acetic acid test, are unreliable.

when it is of high acidity, because it is the layer of mucus which protects the mucosa against the action of the acid secretion.

LACK OF MUCUS AND HYPERACIDITY. We are all familiar with the various subjective symptoms of gastric irritation (burning, gnawing sensation, more or less severe pains, etc.) and the manifold reflex symptoms which occur in patients suffering from hyperacidity. An analysis of a great many histories has convinced me that all these symptoms are most pronounced in patients whose stomach contents are found entirely free from mucus. That the lack of mucus actually plays a great role here became clear to me when I noted complete absence of mucus in some of those well-known cases which present all the gastric and reflex symptoms of so-called hyperacidity, whose stomach contents, however, showed no excess of acids, but normal or even subnormal figures of acidity. There was always some difficulty in explaining the occurrence of "hyperacidity symptoms" in cases in which there really was no hyperacidity. Most authors (Stockton,³ Steele,⁴ and others) maintain that in such cases a sensory neurosis is the underlying cause of the hyperesthesia of the gastric mucosa. Without underestimating the importance of a general neurosis in the development of painful sensations, I think we have a more satisfactory explanation in some of the cases, if we consider the lack of a sufficient cover of mucus as the principal cause which renders the gastric mucosa so sensitive to secretions of a high or even moderate degree of acidity. I can offer another observation to strengthen this opinion.

ACTION OF SILVER NITRATE ON THE SECRETION OF MUCUS. No treatment removes more quickly all the so-called hyperacidity symptoms than the application, by lavage, of solutions of silver nitrate. I can state this positively on the strength of an experience gained by the treatment of hundreds of cases. When after such treatment and after the patients had been perfectly free from all subjective symptoms the stomach contents were again examined, I have often been greatly surprised to find, instead of the expected lowering of the acidity, that the high figures of hyperacidity had remained unchanged. It has already been pointed out by Baihakoff⁵ that the application of silver nitrate does not necessarily reduce the secretion of the gastric juice. Although I have observed in certain cases a decided lowering of the acidity after treatment with silver nitrate, such lowering was not the rule. I have seen more cases in which the acidity remained high; in fact, in some I found even a higher degree of acidity after the treatment than they had before the treatment. And yet these patients had been freed of their annoying symptoms by the use of silver nitrate and many of them had been promptly relieved from severe pains.

³ Jour. Amer. Med. Assoc., January 11, 1903.

⁴ Ibid., August 18, 1906.

⁵ Archiv f. Verdauungskrankheiten, vol. xii, p. 54.

Such observations certainly prove that hyperacidity cannot be the only cause for the suffering of these patients. That, however, the lack of mucus plays a role here becomes plausible, when I report that I found in such cases with persistent hyperacidity distinct quantities of mucus in the stomach contents after the treatment with silver nitrate, while the examination made before the treatment had revealed no mucus at all. It was the appearance of mucus with an improved condition which first made it clear to me that the lack of mucus before the treatment was abnormal, and might account to some extent for the suffering.

The increased secretion of mucus by the use of silver nitrate is easily understood, when one reads the description of Pawlow's⁶ experiments. Pawlow demonstrated, on dogs with a gastric fistula, that mucus is secreted in very large and at times in enormous quantities when a 10 per cent. silver nitrate solution is brought into the small stomach. He explains this greatly increased secretion of mucus as an effort on the part of the mucosa to combat the deleterious action of the noxious substances on the mucous membrane. My clinical observations corroborate this; they show that when the silver nitrate is used in diluted solutions (1 to 5000 up to 1 to 1000) its power to induce an increased secretion of mucus may be turned to advantage as a therapeutic agent. We thus find an explanation for the well-known beneficial effects of the silver treatment.

Another common treatment is thus satisfactorily explained, that is, the oil treatment. Oil taken before meals spreads quickly over the gastric mucosa and provides it with an artificial protective covering, in cases in which the natural protective layer of mucus is insufficient.

LACK OF MUCUS AND GASTRIC ULCER. An insufficient covering of the mucosa may lead to more serious disturbances than mere discomfort and pain. Experiments have shown that the layer of mucus protects the gastric mucosa against mechanical (Sawriew),⁷ thermic, and chemical insults (Zweig);⁸ we may also assume that the layer of mucus is the best protection of the gastric mucosa against the action and invasion of bacteria, as was demonstrated by F. Müller⁹ for the bronchial tubes and by Walthard¹⁰ for the cervix.

Without sufficient covering of mucus the gastric mucosa becomes more easily injured by the many insults to which it is constantly subjected. When once injured, even though slightly, the damaged part becomes more prone to the invasion of bacteria and to the digestive action of the gastric juice. Again, the thinner the layer of protective mucus the easier will these secondary deleterious insults

⁶ The Work of the Digestive Glands, English translation, London, 1902, p. 168.

⁷ Inaug. Dissert., St. Petersburg, 1901.

⁸ Archiv f. Verdauungskrankheiten, vol. xii, p. 364.

⁹ Sitzungsberichte der Gesellschaft zur Beförderung der ges. Naturwissenschaften zu Mnnburg, 1896, Nr. 6.

¹⁰ Centralblatt f. Bakterien- und Parasitenkunde, vol. xvii, p. 311.

to the injured mucous membrane take effect. Under such conditions the digestive activity of the gastric juice must be particularly pronounced when hyperacidity is present.

It is probable that such occurrences are the primary disturbances in the development of gastric ulcer. The majority of cases of gastric ulcer give a history of a more or less prolonged period of suffering from so-called hyperacidity symptoms. We have seen before that this suffering is just as much due to the lack of mucus as to the increased acidity.

We see now that the lack of mucus plays also a role in bringing about the first lesion of the mucous membrane. That in such cases the first superficial lesion may gradually grow and get deeper becomes clear when we consider the chronic nature of the gastritis which at the same time causes both the lack of mucus and the hyperacidity. The persistency of both these disturbances explains the development of the ulcer and also its eminently chronic character. We should bear this in mind when dealing with a gastric ulcer, and should try to treat all disturbances which cause the development of the ulcer and account for its chronicity. One of these disturbances is the lack of mucus. A treatment which brings about an increase of gastric mucus helps to cure the ulcer.

We thus understand the effect of the time-honored treatment of gastric ulcer by silver nitrate, which, as we have seen before, causes an increase of mucus. I frequently had good results by applying silver nitrate in cases in which the regular Leube-Ziemssen treatment had accomplished very little.

How important an increased secretion of mucus is for the healing of a gastric ulcer is demonstrated by the clinical experience that the development of a mucous gastritis proves more conclusively than anything else that the ulcer has healed. This clinical experience finds corroboration in Turck's interesting experiments. Turck,¹¹ who succeeded in producing artificial gastric ulcers in the dog, has lately studied the factors concerned in the healing of such ulcers. Among the changes noted during the process of repair he demonstrated a great increase of mycogen cells, in fact to such a degree that even zymogen cells were transformed into mycogen cells.¹² We thus see that this transformation, while it is really a pathological change, acts as a beneficial agent in the healing of an ulcer.

A similar interpretation may be applied to other conditions which are accompanied by an increase of mucus: An increase of mucus, though apparently pathological, may often prove to be nothing but a more active manifestation of a useful function which serves to protect the mucous membrane. A. Schmidt¹³ has explained the

¹¹ Ulcer of the Stomach; Pathogenesis and Pathology, Jour. Amer. Med. Assoc., June, 1906; British Med. Jour., April 20, 1907.

¹² F. D. Turck. Reported at the tenth annual meeting of the American Gastro-enterological Association, Atlantic City, N. J., June 3, 1907.

¹³ Volkmann's Vorträge, N. S., Nr. 202, p. 1122.

profuse secretion of mucus from any mucous membrane as a favorable symptom, because it shows that the upper layers of the mucous membrane are not greatly degenerated. I should like to add that it should be considered a favorable symptom because it proves that the mucous membrane is doing its duty. From this point of view we must then consider the lack of mucus an unfavorable symptom, for we have seen that diminished secretion of mucus and insufficient mucous covering deprive the gastric mucosa of its protection against all harmful influences, and particularly against the digestive activity of its own secretion.

The routine examinations of stomach contents have so far been principally directed toward the secretion of gastric juice and its disturbances. A more careful study of the protective secretion of mucus and its disturbances will greatly help us to understand and to treat gastric diseases.

THE ETIOLOGY AND SYMPTOMATOLOGY OF CEREBROSPINAL MENINGITIS.

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SINCE its first recognition as a distinct disease by Vieusseux, in Geneva, in 1805, to the present day, epidemics of cerebrospinal fever have occurred at varying intervals and of varying severity, in widely separated localities, extending, according to Hirsch, from 45° N. (Montreal) to 30° N. (Mobile) in the Western Hemisphere, and from 63° N. (Sweden and Russia) to 30° N. (Jerusalem, Persia, and Algiers) in the Eastern Hemisphere. An even wider range might be given, however, as the disease has been noted as far South as Java between 5° and 6° S. In the vast majority of instances it has been impossible to trace the origin of one epidemic to another, only a few authentic examples of transfer of the contagion being on record.

The eastern section of the United States has had a rather generous proportion of the world's epidemics, while the British Isles have been comparatively free, though during the past few months the disease has been prevalent in Belfast, Glasgow; and indeed throughout the United Kingdom, to a greater or less degree. I shall have occasion to refer, later, to reports of numerous epidemics, one of the most formidable being that which occurred in Upper Silesia in 1905, 3102 persons being attacked and 1789 dying.

ETIOLOGY. Cerebrospinal fever occurs most commonly in the winter and spring, but may be seen in the summer. It seems probable that overcrowding and bad hygiene, necessitated among the poorer