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THE PHYSIOLOGIC BASIS OF THE COMMON GASTROINTESTINAL SYNDROMES FOUND IN PULMONARY TUBERCULOSIS.*

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In discussing the relationship between inflammation of the lungs and altered function in the gastrointestinal tract, it is necessary to recall the embryology of these structures; for this gives us a basis for understanding the manner in which they are related through the visceral nerves.

The respiratory system is formed from a diverticulum from the gastrointestinal canal, and therefore carries with it the innervation of the mother structure, the same as the liver, pancreas and body of the bladder, which likewise have the same origin.

This embryological origin gives to the lung the same double innervation as that possessed by the intestinal tract. All smooth musculature and all secreting glands belonging to the lungs and bronchi are activated the same as the stomach and intestines (except the sphincters) by the vagus nerve, which belongs to the parasympathetic division of the vegetative system;

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likewise all, except the sphincters, are inhibited by the sympathetics.

When the pulmonary structures are inflamed, sensory nerves belonging to both of these systems are irritated and result in reflex action. During the stage of toxemia the cells of the entire nervous system also are irritated by the toxins. Toxins express themselves, peripherally, largely through the sympathetic nerves and in this manner produce a general inhibition of action throughout the gastrointestinal tract, decreasing the secretion, and relaxing the muscle of the walls. It may be that there is also an action of the toxins directly upon the muscle cells which interferes with their normal rhythm. The common symptoms on the part of the gastrointestinal tract during toxemia are those of decreased motility and decreased secretion—hypomotility, hypochlorhydria and constipation due to lessened secretion and lessened peristaltic action. This is common in all acute infections accompanied by marked toxemia.

The sensory sympathetics supplying the lung mediate with the spinal nerves in the cervical segments of the cord and produce reflex spasm of the muscles supplied by the motor nerves, and reflex pain in the tissues supplied by the sensory nerves, and, if the process becomes chronic, trophic changes in the skin, subcutaneous tissue and muscles supplied by both motor and sensory nerves.

The sensory fibers of the pulmonary vagus

(parasympathetics) mediate with other fibers of the vagus and with other parasympathetic nerves (7th and 9th cranial nerves) and with the 5th cranial nerve which stands in the same relationship to the sensory fibers of the vagus as the spinal nerves do to the sympathetics.

It is through the vagus (parasympathetics) then that the reflexes take place which affect the gastrointestinal canal when the lung and bronchi are inflamed. This parasympathetic stimulation is the cause of all the common reflex functional disturbances in the gastrointestinal canal which result from pulmonary inflammation.

Before one can understand the reflex symptoms which arise from inflammation in any organ, he must first appreciate the fact that nerve cells in different individuals and in the same individual at different times show different degrees of irritability; and second, that a nerve cell will not discharge and produce action until the stimulus is *adequate*. In other words, nerve cells are able to withstand a certain amount of stimulation without producing action, and this amount varies in different individuals and in the same individual under different circumstances. This accounts for the variability of symptoms as noted in a given disease, a fact which has always made diagnosis difficult. The diagnostician must remember when studying symptoms, that a given stimulus *has a tendency* to produce such and such an action, but that this action may not occur because the stimulus

may not be *adequate* to discharge the nerve cells upon which the action depends. A given stimulus might be able to produce syndromes in several systems of the body, or several syndromes in one system; yet one or more of these might fail to be present.

When the lung is inflamed the sensory fibers of the pulmonary branches of the vagus are irritated, and stimuli are carried to the sensory nucleus of the vagus in the medulla whence they are transferred to other neurons with which they mediate, *viz.*, vegetative fibers of the 7th, 9th, and 10th cranial nerves and somatic fibers of the 5th cranial nerve.

When mediation takes place with the 5th, 7th, and 9th cranial nerves, reflex action results mainly in an increased secretion and an increased irritablilty of the nasal and oral cavities, the pharynx, salivary, and lachrymal glands; in vasomotor disturbance in the cheeks and tongue; in trophic change in the tongue, which at times causes it, when protruded, to turn toward the affected side; and in pain expressed in the sensory neurons of the 5th cranial nerve (headache).

When mediation takes place in other portions of the vagus nerve, reflex action may result in any tissue which this nerve supplies, such as the larynx and pharynx, the bronchi, heart, upper portion of the gastrointestinal canal, liver and gall ducts, and pancreas.

Reflex stimulation, *if adequate,* produces the

action which normally belongs to vagus stimulation in these structures,—an increased tonus in the muscles and an increased glandular secretion.

It is now evident that if stimuli which course centralward over the sensory neurons of the pulmonary vagus are transmitted to the efferent motor neurons of the vagus which supply the gastrointestinal canal, that they will *have a tendency,* reflexly, to cause an increased tonus in the musculature and an increased secretion in the glands. This is what we find clinically when the pulmonary tissue is inflamed in such chronic diseases as pulmonary tuberculosis. The "so-called" functional disturbances on the part of the gastrointestinal canal, which are so common in pulmonary tuberculosis, are nearly all of this type.

Variability characterizes functional disturbances. These symptoms may be present at one time and not at another. Unless one is familiar with this characteristic he cannot fully appreciate the gastrointestinal symptoms in pulmonary tuberculosis.

The common syndromes on the part of the gastrointestinal tract which are indicative of a preponderating vagus stimulation and which often result from reflex stimuli arising in other organs which are the seat of inflammation are nausea, vomiting, hyperchlorhydria, gastric hypermotility, colicky pains, spastic conditions

in the intestines, notably spastic constipation, colitis, diarrhea, and intestinal stasis.

This group of functional disturbances makes up a considerable proportion of the symptoms on the part of the gastrointestinal tract of which patients suffering from early active or chronic semi-quiescent tuberculosis complain, and sends the patient to the gastroenterologist as often, if not more often, than to the specialist in disease of the chest. In fact, the reflex symptoms which are caused by clinical tuberculosis before the advent of marked toxemia, and productive cough, and during the stage of semi-quiescence are practically all expressed reflexly through the vagus in systems other than the lower respiratory; in the larynx as irritation and cough; in the heart as an inhibiting effect producing instability; and in the gastrointestinal canal in the form of the syndromes above mentioned.

These symptoms may be caused by conditions in which the vagus nerve cells are hyperirritable (vagotonia); by conditions which produce a marked stimulation of the vagus or a decreased stimulation of the sympathetics; by direct irritation of the nerve cells lying in the walls of the stomach and intestine; by reflex action in one part of the intestine, the stimulus coming from another part; or reflexly from inflammation in other organs. The most common sources of reflexes coming from without the intestinal

walls, in my experience, are the appendix, gall bladder, lung, and eye.

It can be stated as a rule that functional disturbances on the part of the stomach and intestinal tract are more commonly an expression of reflex action from some other organ than from a disease of the tube itself; and when the syndromes here mentioned are present, the appendix, gall bladder, and lungs should be carefully examined for the presence of disease, and eye strain should be considered. One must not forget, however, that so-called nervous individuals (vagotonics) are also prone to show this same picture of functional disturbance. The expression of "nervousness" in the gastrointestinal canal is predominantly that of increased vagus stimulation. When vagotonics suffer from a pulmonary tuberculosis or a chronically inflamed appendix, or a disease of the gall bladder, then the symptoms on the part of the digestive system which result from vagus stimulation are prone to be very much exaggerated.

In order to understand what functional symptoms on the part of the digestive system are prone to manifest themselves in a patient suffering from tuberculosis, it is necessary first to know the nervous, the physical, and the psychical condition of that patient before he suffered from pulmonary tuberculosis; to know whether he previously suffered from any particultr type of digestive disturbance, and to know what other complications may be present. It is also

necessary to make a distinction between active pulmonary tuberculosis with marked toxemia and the type of disease when toxemia is not prominent.

This is evident from the fact that reflex symptoms do not arise except as the stimulus causing the reflex is sufficient to overcome the action of opposing nerves. When nerve cells are in different degrees of irritability, the strength of the stimulus necessary for their discharge will vary greatly. As long as the force of the sympathetic neurons in the intestinal tract is equal to or approaches the force of the neurons of the vagus nerve (parasympathetics) no disturbance in motility or secretion will take place, and normal function will exist; but just as soon as the force in one system overbalances the other, symptoms will arise.

When the pulmonary parenchyma is inflamed, as it is in tuberculosis, the sensory nerve endings of the pulmonary branches of the vagus nerve are irritated. This stimulus is carried to the medulla, where it is transmitted to motor neurons, which produce reflexes in structures supplied by them. Among the nerves involved are the motor fibers of the vagus which supply the stomach and intestine. This stimulus *has a tendency* to produce action in all the motor neurons to which it is transferred; and when it is *adequate* to overcome all opposing forces acting upon the sympathetics, it produces para-

sympathetic syndromes such as are mentioned below.

Whether a reflex gastric hypersecretion or hypermotility (increased muscle tonus), or a reflex intestinal hypersecretion or hypermotility (increased muscle tonus) will occur depends upon whether the stimulus arising in the inflamed organ and transmitted to the musculature and secreting glands of the stomach and intestine through the vagus is sufficiently strong to overcome the opposing inhibitory action of the sympathetic nerves supplying those tissues. This will depend also, to a large extent, upon the natural characteristics and tendencies of the patient. That the stimulus is adequate in a large proportion of instances is evident from the frequency with which patients afflicted with pulmonary tuberculosis suffer from reflex nausea, vomiting, hyperchlorhydria, spastic constipation, colitis, intestinal stasis, and colicky pains. These syndromes are common in patients who were free from them prior to their clinical tuberculosis and are often rendered more annoying in those instances where they were previously present. I have seen hyperchlorhydria in many instances during the high fever accompanying cavity formation. Not infrequently have I seen a relative bradycardia at the same time. These symptoms can only be interpreted as being a result of reflex stimulation of the vagus from inflammation in the lung, coming as they do during severe toxemia, which

stimulates the sympathetics, depressing gastric secretion, and produces an accelerating influence upon the heart. Nausea and vomiting, which are often present at these times, are also frequently of reflex origin. These reflex relationships I have discussed quite fully in previous communications.[1, 2]

A few words in explanation of the reflex nature of these syndromes may not be out of place. I do not wish to be understood as maintaining that these syndromes when present in pulmonary tuberculosis are always a reflex from the lung, for I realize that there are other causes operating; but I desire to emphasize that the inflammation in the lung produces stimuli which have a tendency to produce them. *Nausea and vomiting,* found in pulmonary tuberculosis, are undoubtedly often of reflex origin; being an expression of increased muscular tonus in the gastric walls. These patients also, at times, show more than ordinary degrees of hunger. Carlson has shown that this phenomenon is due to increased gastric motility.

Colicky pains are very common during the course of pulmonary tuberculosis. Unless there be a mechanical obstruction these are usually due to areas of spasticity in the intestine which interfere with the onward movement of the gas, causing it to accumulate, and distend the gut and cause the pain. When the intestinal musculature is in a state of increased tonus, different areas of the gut show different degrees of

irritability, a condition which favors constriction at intervals and permits of the accumulation of gas with distention of the bowel proximal to the constriction.

Spastic constipation often results from reflexly increased tonus in the musculature of the colon, the stimulus causing the reflex emanating from the lung. This form of constipation is usually accompanied by colicky pains. It is due to the same cause, being an expression of the same increased muscle tonus (hypermotility).

Colitis in tuberculosis has the same reflex cause as the foregoing phenomena. The vagus, when stimulated, not only causes an increased muscle tonus leading to a hypermotility, but also an increased secretion in the intestinal glands.

The reflex type of *intestinal stasis* is caused by a retardation of the intestinal contents in their progress through the canal as a result of spastic conditions in the bowel.

If we substitute inflammation in the appendix and the sensory fibers of the vagus which supply the appendix in one case, and inflammation of the gall bladder and the sensory fibers of the vagus which supply it in the other, for the inflammation of the pulmonary tissue and the sensory fibers of the vagus which supply it, we have the mechanism which explains the gastro-intestinal symptoms in appendicitis and inflammation of the gall bladder. Then, further, if we will substitute the stimuli which arise from

eye strain, and the sensory fibers of the 5th cranial nerve which mediate with the motor neurons of the vagus, for the stimuli arising from inflamed pulmonary tissue and the sensory fibers of the pulmonary vagus, we will have an explanation of the reflex mechanism through which eye strain produces symptoms on the part of the gastrointestinal canal.

Through an understanding of visceral nerves and the relationships which are maintained by the various viscera through them, reflex functional disturbances are stripped of their former mystery and placed on an understandable basis.

REFERENCES.

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[2] Pottenger: Symptoms of Visceral Disease. 1919. C. V. Mosby & Co., St. Louis, Mo.

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