THE SYNDROME OF TOXEMIA

AN EXPRESSION OF GENERAL NERVOUS DISCHARGE THROUGH THE SYMPATHETIC SYSTEM

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The human body under normal conditions has a perfect physiologic balance. One part or organ is in a state of perfect, balanced relationship with every other part or organ. A disarrangement of this equilibrium produces pathologic conditions and dysfunction. These derangements are indicated by symptoms and signs, which, when grouped, give us a picture of a definite disease.

The explanation of well-known symptoms is often wanting, the practitioner being compelled to learn them as a part of the syndrome belonging to a certain disease instead of being able to point out the exact force operating to produce them. An etiologic classification of symptoms leads us to a better understanding of the disease in question and facilitates diagnosis.

In analyzing the symptoms and physical signs in pulmonary tuberculosis, I found that they could all be classified etiologically in three groups: 1, those due to toxemia; 2, those due to reflex action; and, 3, those due to the tuberculous process per se, as in the table.

ETIOLOGIC CLASSIFICATION OF SYMPTOMS AND PHYSICAL SIGNS IN TUBERCULOSIS

Group 1.—Toxemia	Group 2.—Reflex Action	Group 3.—Tuberculous Involvement per se
Malaise	Hoarseness	Frequent and protracted colds
Lack of endurance	Tickling in larynx	Spitting of blood
Loss of strength	Cough	Pleurisy
Nervous instability	Circulatory disturbances	Sputum
Lack of appetite	Digestive disturbances	Temperature
Digestive disturbances	Loss of weight	
Loss of weight	Chest and shoulder pains	
Rapid pulse	Flushing of face	
Night sweats	Apparent anemia	
Temperature		
Anemia		

In this manner we classify some twenty-five or thirty symptoms which belong to tuberculosis in three groups so that each symptom in the individual group is due to a common etiologic factor. Such a classification simplifies diagnosis. It offers an explanation for the fact, which has long been observed, that symptoms are variable, in that the same ones are not always present, and when present, are not always equally prominent.

When studying these three groups of symptoms I was impressed with the fact that there is nothing distinctive of tuberculosis in that group which is due to toxemia. The same symptoms could be due, as well, to an infection of the tonsil, the prostate, the fallopian tube, a toxemia from intestinal stasis or an acute infectious disease. They are, in short, a part of the syndrome of infections in general. I was further impressed with the fact that the second group, those of reflex origin, all point to organs other than the lung, but that they belong to organs which are supplied by the vagus and sympathetic nervous systems, both of which supply the lung. *A further analysis of the group which is due to toxemia shows that these symptoms are identical in distribution and effect with a general discharge of nervous impulses through the sympathetic nervous system.*

To make this clearer it is necessary to recall that the autonomic nervous system supplies impulses to structures which are not controlled by the will. These are the organs supplied by smooth muscle such as the stomach, intestines, blood vessels, ducts of glands; and also certain organs possessing striated muscle fibers such as the heart, the beginning and terminal portions of the alimentary canal, and the generative organs.

The autonomic system is divided into three groups: 1, the cranial and bulbar, which are spoken of as the vagus system; 2, the thoracic and upper lumbar, known as the sympathetic system; and, 3, the lower lumbar and sacral, known as the sacral system.

The vagus system is the system which conserves life. It contracts the pupil, increases salivary secretion, slows the heart beat, causes an increase of gastric and intestinal juices, and furnishes motor power for the gastrointestinal tract. The sacral system controls the emptying of the lower bowel and bladder and presides over the generative functions. The sympathetic sends branches to all organs supplied by the vagus and sacral systems; and where the fibers from these different systems meet there is an antagonistic action between the sympathetic and other two systems. If the sympathetic stimulation is sufficiently strong, it overcomes the vagus and sacral tonus. If this control is only momentary or of short duration, as it is in fear and anger, as shown by Cannon,[1] the general strength of the individual is increased. He possesses for the time being a power greater than normal. On the other hand, if this sympathetic control continues, a general inhibition of function in the organs supplied by the sympathetic system takes place and the individual's powers suffer a diminution; thus, the dry mouth, lack of desire for food, stoppage of digestion and rapid heart's action, which are temporary in the presence of the major emotions, are emphasized and prolonged during toxemia and are expressed as a more or less continuous absence of appetite, coated tongue, retarded digestion, constipation, rapid heart action and tendency to perspiration.

While impulses may be carried directly from the brain and cord to the skeletal muscles, insuring immediate and selective response, and through the vagus and sacral systems through the intervention of a single ganglion, causing response limited to a certain organ, in the case of the sympathetic nervous system numerous ganglia are interposed and the response is shown in widely distributed parts. These ganglia act as modifiers or transformers of the impulse and make its distribution general, which accounts for the fact that so many organs and parts are involved in sympathetic irritation.

Whether a toxemia acts wholly, centrally, on the sympathetic nervous system is open to some question. While the expression is that of general sympathetic discharge, yet we must bear in mind that it is possible that this may be partly due to, or at least partly prolonged by, certain internal secretions which are engendered by, the same stimulation as that which produces the general sympathetic stimulation. Cannon has shown that the emotional states such as anger and fear, while being an expression of general sympathetic discharge, may be kept up by adrenin in the blood. The adrenal glands are supplied through the splanchnics; and impulses which cause a general sympathetic stimulation stimulate these glands also. A minute amount of adrenin poured into the blood stream has the effect of producing a prolongation of the condition which is brought about by direct sympathetic stimulation; thus adrenin

1. Cannon, W. B.: Bodily Changes in Pain, Hunger, Fear and Rage, Appleton, New York, 1915.

will cause a dry mouth, impaired digestion, intestinal stasis and a rapid heart. That toxemia, like the emotional states, acts by stimulating the sympathetics and by prolonging the action through the stimulation of the adrenals seems quite certain. It may also be found on further study that disturbances in other internal secretions may have a part in this general picture; but, even should this prove true, it will not alter the fact that the syndrome of toxemia is an expression of general sympathetic discharge.

SOFT EYEBALL IN DIABETIC COMA *

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Quite accidentally I tried one day the ocular tension in a case of diabetic coma, and was startled to find the eyeball of almost doughy consistency. On looking over the literature of diabetes I found that this symptom was first described by Krause[1] in 1903. He did not observe it in simple diabetes or in cases of acidosis unassociated with coma. In experiments on animals with acetone, diacetic acid and oxybutyric acid he failed to produce hypotonia of the eyeball; and he did find it in a dog rendered diabetic by pancreatectomy. Since Krause's first paper, few articles on the subject have appeared. Heine,[2] an ophthalmologist, refers to it in several publications. The observations of Lea Gite Schütz[3] are the most accurate, for they were made not with the finger but with the tonometer of Schiötz. She found that the tension varied from day to day, but that at the height of the coma it was extremely low or even nil. Few of the systematic writers speak of the symptom. Lépine[4] merely mentions it, referring to the papers of Krause and Heine. Among the many works on ophthalmology that I consulted, only the small monograph of Groenouw,[5] the book of Römer[6] and the article of Heine[7] speak of it.

Hertel[8] made a number of interesting experimental observations bearing on the symptom. He found, for example, that the electric conductivity of the soft eyeball was raised, showing that osmotic changes had taken place. By lowering the molecular concentration of the blood of animals, he succeeded in producing a hypotonia similar to that of diabetic coma in man. The degree of hypotonia could be varied by modifying the amount of substance introduced and the time consumed in its introduction. Various sodium salts, urea and sugar were the agents employed. Ehrmann[9] brought about hypotonia by injections of sodium butyrate and sodium isovalerianate, but in Hertel's opinion the hypotonia is not a specific effect of these salts as the same result can be obtained with sodium chlorid, sodium phosphate, urea and sugar. Chemical examination of the eyes showed that the substances injected had penetrated into the eyeball.

The soft eyeball in diabetic coma is not due to blood pressure changes; it is also not an agonal phenomenon, for it is not present in persons dying from other causes. Whether the ketone bodies play a part in its production is not definitely established. In acidosis without coma the symptom is not present. As, however, in acidosis with coma the acetone bodies are probably retained in large quantities, they may have a share in the production of the soft eyeball. In one of my cases of diabetic coma, in which ketone bodies were not found in the urine at any time, the soft eyeball was not present. In a recent case of diabetic coma in my care, that of a woman aged 41, with intense acidosis, the intern and I tested the tension repeatedly and found that it varied, as shown by the following records:

Oct. 26, 1915, on admission, the patient is conscious but lethargic; breath exhales a strong fruity odor. The urine contains sugar and diacetic acid in large quantities. Tension of eyeballs practically normal.

October 28, the patient is in coma. The pupils are contracted; the tension is greatly diminished in both eyeballs.

October 29, the patient is profoundly comatose. The tension of the eyeballs is somewhat greater than yesterday, but still markedly below normal.

Krause,[10] who observed it in twenty-two cases of coma, was of the opinion that the soft eyeball was a bad prognostic sign, but Schütz found that in one of her three cases recovery took place. Regardless, however, of prognostic value, the sign is important from the diagnostic standpoint. Heine[7] goes so far as to say that whenever found in a case of coma it proves the coma to be diabetic. Further observations are necessary before such a strong statement can be fully accepted.

As to the constancy of the sign in diabetic coma I cannot speak; in the last three cases of coma under my observation — the only ones in which I tested for it — I found it twice. One would hardly expect it to be present in every case, for constant symptoms are rare not only in diabetes but in all diseases.

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* Read before the Section in Medicine of the College of Physicians of Philadelphia, Nov. 22, 1915.

1. Krause: Ueber ein bisher nicht bekanntes Symptom bei Coma diabeticum, Verhandl. d. 21. Kong. f. inn. Med., Leipzig, 1904, p. 439.
2. Heine: Ueber Augenstörungen im Coma diabeticum, Versamml. d. ophth. Gesellsch., Heidelberg, 1903; Ueber Lipaemia retinalis und Hypotonia bulbi im Coma diabeticum, Klin. Monatsbl. f. Augenh., 1906, ii, 451.
3. Schütz, L. G.: Untersuchungen über den Augendruck beim Coma diabeticum, Inaug. Diss., Strassburg, 1913.
4. Lépine: Diabète Sucré, Paris, 1909, p. 578.
5. Groenouw: Augenleiden bei Diabetes mellitus, Halle, 1907.
6. Römer: Lehrbuch der Augenheilkunde, Berlin, 1910, p. 647.
7. Heine: Lehrbuch der Augenheilkunde, T. Axenfeld, 1915, p. 755.
8. Hertel: München. med. Wchnschr., 1913, p. 1191.
9. Ehrmann, quoted by Hertel (Footnote 8).
10. Krause: Deutsch. med. Wchnschr., 1907, p. 84.

A STRANGULATED EPIGASTRIC HERNIA *

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Any protrusion of tissue through a defect in the linea alba between the ensiform cartilage and the umbilicus is designated an epigastric hernia. Most of these hernias occur in the first 3 inches above the umbilicus, while those near the xyphoid are exceedingly rare. The relative frequency of epigastric hernia when compared with other types varies according to different statistics from 0.19 per cent. (Macready[1]) to 1.37 per cent. (Berger[2]). Coley[3] reports twelve cases from the Hospital for Ruptured and Crippled. These were observed in 3,383 cases of hernia (0.33 per cent.). In the last 1,000 hernia cases in which operation was performed at the Presbyterian Hospital of Chicago, there have been nine epigastric hernias, or 0.9 per cent.

Four types or varieties are usually described. The most common type is the protrusion of a small mass

* From the Surgical Clinic of Rush Medical College.

1. Macready, quoted from Keen's Surgery, 1912, iv, 90.
2. Berger, M. Paul: Traité de Chirurgie, 1892, p. 797.
3. Coley, W. B.: Keen's Surgery, 1913, vi, 588.