

From all that has been said, it would appear conclusive that the plague of Athens was not the true bubonic plague, and it simply remains for us now to examine whether or not it was the smallpox which afflicted the capital of Greece.

In both smallpox and the epidemic which afflicted Athens we find a generalized eruption followed by ulceration, hyperemia of the eyes, tongue and pharynx, while the general symptoms are more marked in the respiratory and digestive systems. It is well known that in smallpox the eruption may interest the conjunctiva and the cornea, and may thus give rise to more or less severe ophthalmia, which from the destruction resulting may cause complete cecity. This fortunately rarely occurs in smallpox.

On the other hand, death is not the same in the two diseases which we are considering. This occurred on the seventh or the ninth day in the plague of Athens, while in confluent smallpox the patient rarely dies before the eleventh day, and, generally speaking, it may be said that from the twelfth to the fourteenth day is the usual time of the fatal ending, should it occur in this disease. Occasionally the patient is carried away during the first five or six days of smallpox, but this apparently only occurs when the disease presents an exceptional malignancy or when it assumes an abnormal type.

To still further show the differentiation between the two diseases, we would here append the following conclusions taken from the dissertation of Krauss, which was published in 1831, and had for title "*Disquisitio historico-medica de natura morbi Atheniensium a Thucydide descripti*," an analysis of which has been given by Littré in the second book of the epidemics of the French translation of Hippocrates:

"(1) True smallpox does not transform into ulcers, the pustules remain full of puriform lymph until desiccation occurs. (2) Thucydides does not intimate that the eruption had a critical character; now, in all epidemics, in the greater part of patients afflicted by smallpox, the eruption has this character. (3) Several symptoms which, according to Thucydides, occurred to some patients, for example, gangrene of the feet, the hands or the genital organs, have not been mentioned by any author who has described epidemics of smallpox. (4) In the long interval of time occurring from Thucydides to the sixteenth century of the Christian era, when smallpox is expressly named, one finds, it is true, various eruptions mentioned that some writers have called traces of smallpox; but one does not find a description of smallpox, and from this fact it may be argued that the latter disease did not exist in antiquity. Now in point of fact, since its first appearance in the sixteenth century of the Christian era, it has always preserved the character by which we know it at the present time, and it constitutes a disease of a perfectly constant type. Now, who would dare to reproach the ancient writers to have not taken into account this type which is so distinctly characteristic. (5) The cicatrices which smallpox leaves on the skin have never been mentioned

by any of the ancient writers, and this seems to be an argument of the greatest force."

From all these considerations I believe that we may conclude that the plague of Athens cannot be compared with any of the diseases known at the present time, and, consequently, it should be considered as a special epidemic disease which is now extinct. Without emitting any hypothesis as to the real nature of this scourge, which would be time lost, it is quite admissible to believe with Littré, Hecker, Daremberg and others that it should occupy a special place in the great class of exanthematic fevers.

THE CONDITION OF THE VASOMOTOR NEURONS IN "SHOCK."¹

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INJURIES to peripheral tissues may at times be followed by a condition termed "shock," characterized by a great fall in blood pressure, sinking of the body temperature, weak and often irregular heart beats and lessening of the normal irritability of the nervous system. Such symptoms may also be called forth by the direct mechanical injury of the nerve cells which maintain these several functions, but this should be distinguished from shock. In shock the primary injury lies outside these nerve cells.

The symptoms which characterize shock have often been ascribed to a depression of the vasoconstrictor nervous system. Each of the nerve chains which constitute the vasoconstrictor nervous system has three neurons. The cell body of the first lies in the bulb; its axis cylinder process ends in the spinal cord or in the bulb itself, in physiological contact with the second neuron. The cell body of the second neuron lies in the cord or bulb, but its axis cylinder process leaves the cord and ends in physiological contact with a sympathetic cell. The sympathetic cell is the third neuron. Its axis cylinder process ends in the wall of a blood vessel.

The bulbar cells, or first neurons, affect the blood vessels only through the second and third neurons. Consequently, if stimuli which pass through afferent nerves to the bulbar cells call forth a normal change in the caliber of the blood vessels, the condition of all three neurons must be normal.

The most conspicuous afferent path to the vasoconstrictor center is furnished by the depressor nerve. It has been shown by Porter and Beyer³ that the depressor nerve connects with all the bulbar vasoconstrictor cells alike. The fall in blood pressure occasioned by the stimulation of the central end of the depressor nerve is therefore a quantitative test of the condition of all the vasoconstrictor neurons,

¹ Presented before the Boston Society of Medical Sciences, Oct. 20, 1903.

² Dr. Quinby took part in this research at the instance of the Surgical Department of the Harvard Medical School.

³ W. T. Porter and H. G. Beyer: Amer. Journ. of Physiol., 1900, iv, pp. 283-299.

those of the first order, in the bulb, and those of the second and third order through which alone the bulbar neurons can alter the constriction of the blood vessels. If, therefore, the stimulation of the central end of the depressor nerve produces as great a fall in blood pressure during shock as before shock began, the condition of the three vasoconstrictor neurons must be normal even in shock.

Upon this conclusive reasoning rests the method pursued in this investigation. The normal fall of blood pressure produced by stimuli of uniform intensity applied to the central end of the depressor nerve was measured in the rabbit and the cat. In the same animals shock was then brought on and the measurements repeated. Following is an abbreviated but typical protocol:

EXPERIMENT XI, SEPTEMBER 24, 1903.

9 A.M. Rabbit anesthetized with ether.

9.15. Carotid blood pressure 67 mm. Hg. Rectal temperature, 38° C. On stimulation of the depressor nerve the blood pressure fell to 36 mm., a fall of 46%.

9.20 to 9.30. Applied nitric acid to exposed intestines. The blood pressure at first rose slightly and then sank slowly; the rectal temperature also sank steadily. Shock soon progressed so far that the anesthetic was no longer necessary. The gradual sinking of the blood pressure to a point almost 50% below the normal and the effect of stimulation of the depressor are shown in tabular form. At 3.25 P.M. the rectal temperature was 26° C.

Hour.	Blood Pressure, Mm. Hg.	Fall on Stimu- lation of Depressor, Mm. Hg.	Percent of Fall.
3.25 P.M.	53	30	43
3.30	53	30	43
4.50	40	22	45
5.16	35	23	34

At 5.16 P.M., eight hours sixteen minutes after the beginning of the experiment, the rectal temperature was 25° C.

In other experiments the blood pressure, which had fallen in shock, was temporarily raised to normal by the injection of saline solution in the jugular vein, and the depressor immediately stimulated. In such cases the blood pressure fell as many millimeters as it had on stimulation before shock set in.

Exhaustion in the vasoconstrictor neurons cannot therefore be the essential cause of the symptoms termed "shock."

A HITHERTO UNDESCRIBED MEMBRANE OF THE EYE AND ITS SIGNIFICANCE.¹

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WHILE studying the external limiting membrane of the retina in a section of a normal eye, the writer was surprised to find it continue on at

¹ This paper will appear in full with illustrations in the Reports of the Royal London Ophthalmic Hospital.

the margin of the disc and pass into the layer of pigment epithelium, where it could be followed until the gradually increasing pigmentation hid it from view. Other sections of the same eye, bleached by the method of Alfieri and stained in a variety of ways, showed the still more surprising fact that this membrane was present in the pigment layer throughout the fundus. Mallory's phosphotungstic acid hematoxylin seems to be the best stain for the membrane, because in specimens fixed in formaldehyde and in bleached specimens fixed in Zenker's fluid it does not stain the cells to any extent, and the membrane is brought out in sharp contrast.

The appearance presented by this membrane in cross section is that of a delicate line running along near the inner margins of the cells, the latter projecting beyond it in the form of processes of variable length. Occurring at almost regular intervals on the line there are black dots, each of which on close examination is found to be situated at the line of junction of two cells. On careful focusing it can be seen that the line is not always on the same level, evidently passing sometimes behind and sometimes in front of the cells. Rarely it is missed entirely for a cell or two. In oblique sections the true structure of the membrane is at once apparent, the latter appearing then not in the form of a straight line, but as a series of hexagonal loops which are fused together at their sides of contact so as to form a screen into the openings of which the pigment cells project. The little dots resolve themselves into the sides of the loops, which in cross section had pointed more or less directly at the observer.

That this is a true fenestrated membrane, and is not an appearance produced by the contours of the cells, there can be no doubt whatever. In the first place, as just stated, it does not correspond in position to the margins of the cells. Secondly, in phosphotungstic acid hematoxylin followed by acid fuchsin it stains blue, whereas the cells stain pink or not at all, and show no vertical lines between them. Thirdly, in certain places where two neighboring cells have shrunk apart, it remains intact. Fourthly, it can frequently be seen projecting beyond a fragmented portion of the pigment layer.

The membrane in the pigment layer is similar to the membrana limitans externa of the retina in structure and also in staining reactions, both membranes giving the staining reactions of neuroglia. The writer has a few specimens which seem to show a similar membrane in the ependymal epithelium of the brain and spinal cord. Possibly this has already been described by others.

For years the membrana limitans externa has been described as being formed either by the apposition of the outer ends of Müller's fibers, or by lateral interlacing offshoots from them. Greeff, in his elaborate article on the retina, still holds to this view. The basis for this belief seems to rest on the fact that Müller's fibers have been seen to end in contact with the membrana limi-