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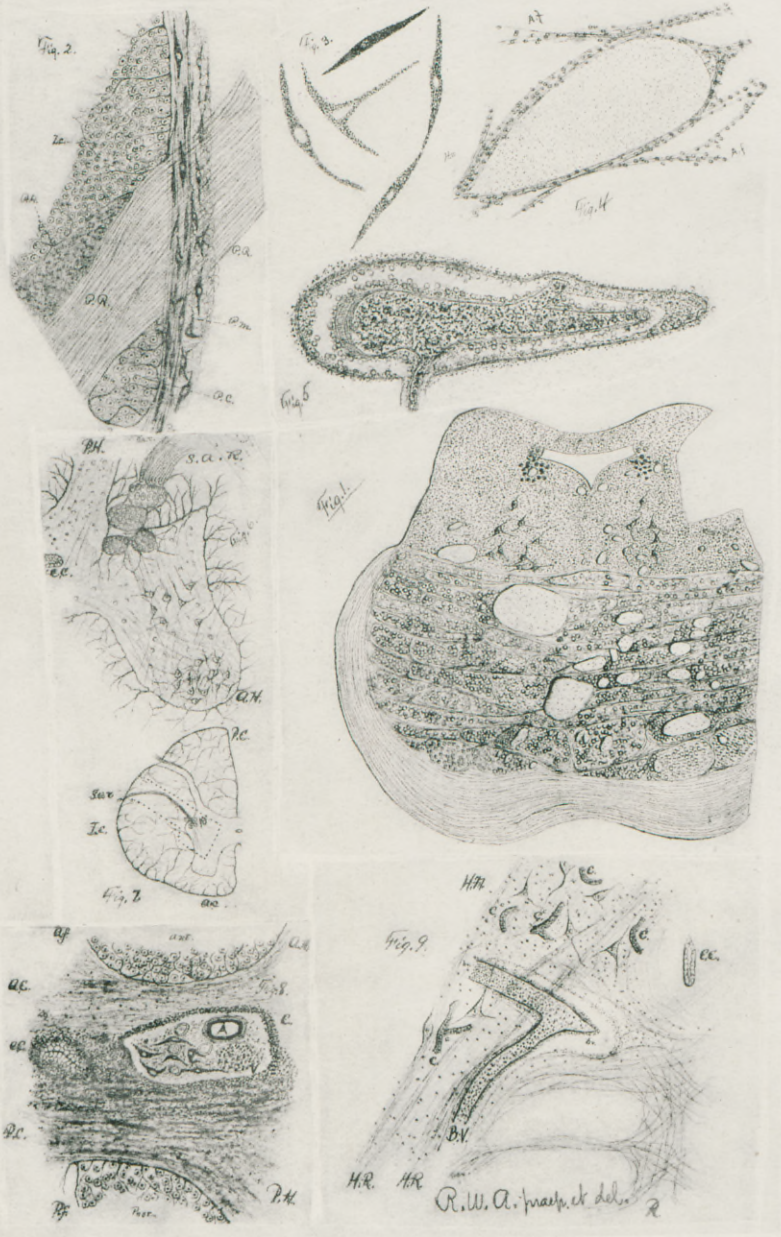
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SOME NEW POINTS ON THE PATHOLOGICAL ANATOMY OF TETANUS.*

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Until within a few years, the pathological anatomy of tetanus was involved in the greatest obscurity.

This did not depend, however, on the lack of post mortem examinations, for their records are numerous, but on the fact that in so few instances the microscope was used to verify gross lesions and to detect minute ones.

Investigators contented themselves by saying "the nerve looked inflamed or the cord congested or softened," and so, wanting sufficient evidence of actual lesions, the profession gradually came to look on tetanus as a purely functional disease.

In the year 1864, appeared J. L. Clarke's report † of the microscopic examinations of the spinal cord in two cases of tetanus, in which he found changes hitherto unknown. He noted a granular exudation in the anterior fissure, general dilatation of the blood-vessels and granular matter in the perivascular spaces.

In 1865 appeared a report of four new cases by the same author.‡ In one of these the medulla oblongata was examined and found normal. The lesions were found in the

* Read before the New York Neurological Society, April 7, 1879.

† *London Lancet*, Sept. 3, 1864.

‡ *Med. Chir. Trans.*, London, Vol. XLVIII, p. 255, 1865.

spinal cord chiefly in the gray matter. They consisted in dilatation of the arteries, hemorrhages, areas of disintegration and cavities containing granular fluid, clear colloid material or débris of nerve-tissue.

In 1868 W. H. Dickinson,* examining the cord of a case of traumatic tetanus, found repletion of the blood-vessels, extravasations, and structureless, transparent material about the blood-vessels and in the nerve-tissue in both gray and white matter. This exudation sometimes occupied pre-existing cavities and sometimes seemed to have torn the nerve-tissue assunder. This substance, he says is stained pink by carmine.

In 1869 Ogle,† in a case of traumatic tetanus, found the fissures of the cord, especially the anterior, occupied by an exudation.

In 1871 T. C. Allbutt,‡ in examining the spinal cords of four cases, found congestion of the vessels with thickening of their walls, large and small hemorrhages, granular exudations through the walls of the vessels, stuffing of the central canal with epithelium, yellow degeneration of the motor cells and spots of granular disintegration in different situations.

In 1871–2 Clarke again comes forward reporting§ the microscopic examination of two more cases of tetanus. In one case there was congestion of the cerebral gray matter. There was also a recent, extra-meningeal blood-clot in the anterior part of the cervical and dorsal regions, dilatation of the blood-vessels, exudation of a homogeneous material, chiefly between the posterior cornua, and irregular cavities in the medulla oblongata.

In the second case he found a coagulum surrounding the

* *Med Chir. Trans.*, London, Vol. LI, p. 265, 1868.
† *British and Foreign Med. Chir. Rev.*, April, 1869.
‡ Trans. London Path. Soc., Vol. XXII, p. 27, 1871.
§ St. Geo. Hosp. Reports, Vol. VI, pp. 319 and 336, 1871–2.

theca vertebralis in the dorsal and lumbar regions, dilatation of the blood-vessels and exudation of a transparent, homogeneous material.

In 1872 Michaud * noticed the exudation of plasma from the vessels, and an aggregation of nucleated corpuscles in both commissures all around the central canal.

In 1873 Allbutt † in examining the cords of four cases found the vessels thickened, distended and plugged, hemorrhages, submeningeal and into the cord, softening of the cord, stuffing of the central canal with epithelium, nuclear proliferation of connective tissue and atrophy of the cells in the anterior horns.

Fox ‡ in 1874 announces the finding of congestion of the spinal pia mater, some new cellular material on the inner surface of the dura mater, colloid degeneration of the white and amyloid degeneration of the gray matter of the cord.

In 1875 Richelot,§ quotes Joffroy ‖ as finding in the pons, medulla and cord, vascular distention and hemorrhages.

He quotes Quinquaud ¶ as finding vascular distension in the floor of the fourth ventricle, oil globules and amyloid bodies outside the vessels in the cord.

Liouville (Soc. Biologie 1869), he quotes as describing the lesions in the cervico-dorsal region of the cord, as distension and at places, aneurysinal dilatation of the blood-vessels, hemorrhages and a granular, pigmented condition of the nerve cells; and in the dorsal region a sero-sanguineous effusion about the posterior spinal nerve roots.

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* *Arch. de Phys.*, p. 60, Jan., 1872.

† *British and Foreign Med. Chir. Rev.*, April, 1873, p. 383.

‡ Path. Anatomy of the Nervous Centres. p. 355. 1874.

§ Richelot, Pathogénie, Marche, Terminaisons du Tétanos.

‖ Soc. de Biol., 1870, Mem. p. 14.

¶ Leclerc, Considérations sur le tétanos traumatique, Thése de Paris, 1872.

Aufrecht * in 1878 found lesions confined chiefly to the cervical region of the cord.

They consisted in hyperæmia, some hyaline exudation about the vessels and in the central canal, degeneration and atrophy of the cells in both anterior and posterior horns and some spots of granular disintegration.

Woods † in 1878 records the finding of vascular distention, mostly about the central canal, round bodies in the perivascular spaces and granular disintegrations of the posterior horns.

In the medulla oblongata which he examined, he noted vascular dilatation about the hypoglossal and pneumogastric nuclei and in the floor of the fourth ventricle.

He also gives a sketch of an enlarged blood-vessel near the hypoglossal nucleus.

It were an injustice, even while confining our attention to those authorities who supplemented their investigations by microscopic examinations, to pass over in silence the clinico-pathological investigations of one of our most original men.

Dr. J. Marion Sims as early as 1846 ‡ 1848, § stated it as his conviction that the form of tetanus, known as trismus neonatorum, was caused by the pressure of the occiput, depressed during labor or by dorsal decubitus, on the medulla oblongata. This, to his mind, was conclusively proven by the results of autopsies, where the occiput was found depressed, and by the fact that a vast majority of his cases were cured by a lateral or facial decubitus alone.

In locating the seat of the disease in the medulla and pons, he by far antedated many of his successors.

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* *Deutsche med. Woch.*, Nos. 14 and 15, 1878. Quoted in *Am. Jr. Med. Sci.*, July, 1878; *Br. Med. Jr.*, May, 1878; and *Chicago Jr. Ment. and Nervous Diseases*, Oct., 1878.

† *London Lancet*, Sept. 7, 1878. p. 326.

‡ *Am. Jr. Med. Sci*, April, 1846.

§ *Am. Jr. Med. Sci.*, July and October, 1848.

An article by Gowers * deserves notice as demonstrating the existence of lesions like those found in tetanus, in the medulla and pons of a person dying of the very similar disease, hydrophobia.

Some facts of great importance in further study will strike one on looking over the literature of tetanus. While some have considered the disease functional and without lesion; while others have paid special attention to the condition of nerves in and leading from the wound; while, as most of the authors above quoted, others have in the spinal cord sought the only lesion; how few have gone one step higher, and in the medulla oblongata and pons varolii sought the characteristic lesion?

No systematic attempt seems to have been made to reconcile the location of the lesions with the prominent symptoms.

In the typical case followed to a fatal termination, the lesions should correspond to the symptoms present before death.

For example, at an autopsy there is found an embolus impacted in the left middle cerebral artery, shutting off the blood-current from the centre for speech, and the motor centre for the right side of the face and body. On ascertaining the history of the case, it is found the patient was suddenly seized with right hemiplegia and aphasia.

Here the location of the lesion explains fully the symptoms.

The lesions of tetanus, however, have never been made to harmonize with the symptoms.

To the end of making clearer, this part especially, of the pathological anatomy of tetanus, the following case † is presented without further preface.

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* Trans. Lond. Path. Soc., 1877.

† The case was under the care of Dr. T. M. Markoe in the New York Hospital, and is used with his permission.

BERTHA B., aged 15 years, domestic, German. About 5 P.M., June 30, 1877, patient fell two stories, receiving a contused and lacerated wound 5 cm. long, extending transversely across the palm-surface of the right wrist. Through this wound protruded the lower extremity of the upper fragment of the fractured radius. There was also a slight contusion of the right ankle and back.

On admission there was considerable pain, hemorrhage and shock.

Reduction of the protruding radius was attempted, under ether, without avail.

July 1st.—The patient complains of great pain in the arm, which is subdued by morphia administered hypodermically.

July 2d.—Under ether, the protruding bone was cut off and reduction accomplished. The soft parts about and in the wound were in a gangrenous state.

July 3d.—Pulse, 88; temperature (axillary), 38.9° C., (102° F.).

July 5th.—At 10 A.M., trismus set in. The teeth at times very firmly clinched, often biting the tongue. At 3 P.M., temperature in axilla, 39.5° C., (103.1° F.). 5.30 P.M., temperature in axilla 40.5° C., (104.9° F.). 5.30 P.M., temperature on right cheek, 39.0° C., (102.2° F.). About this time the patient complained of pain and stiffness in the back of the neck, thirst and fever, and was very desirous of being fanned. Dysphagia or difficulty in swallowlowing now began to manifest itself, and tonic spasms of the sterno-mastoid and trapezius muscles.

A short time after the "*risus sardonicus*" set in. In this case there was no elevation or depression of the angles of the mouth, giving the face a smiling or forbidding look, but there was simply a retraction of the angles of the mouth with some separation of the lips so as to show the teeth.

7.30 P.M., the patient's cheeks are very red and hot, her face is covered with large drops of perspiration. She is still perfectly conscious and complains of heat and pain. She asked to have the wooden wedge, put between the teeth to prevent biting the tongue, removed.

Respiration was now hurried, the alnæ nasi rose and fell, the inspiratory movements were labored and often interrupted by a spasm of the glottis causing impending suffocation. The pulse was rapid and regular, and the extremities cool.

8 P.M., the struggle for breath ceased. The respiration assumed a Cheyne-Stokes character, *i. e.*, intervals of complete absence of respiratory movements alternated with about equal periods of

more or less normal respiration. The apnœic periods became longer, the consciousness was lost, the cheeks paled, the eyes fixed, the jaw fell, the spasms relaxed, and at 8.15 P.M., death ensued.

Temperature 5 min. later in the axilla being 43.3° C., (110° F.), and on the surface of the abdomen 40.5° C., (105° F.).

AUTOPSY 13 HOURS AFTER DEATH.

Temperature in axilla 33.5° C., (92.3° F.), and on abdomen 31.25° C., (88° F.).

The rigor mortis was wanting in the parts tetanized before death.

The cerebral sinuses were filled with blood.

The brain was superficially finely injected on the superior surface of the occipital lobes. There was a vertebral artery only on the left side.

The lungs were œdematous.

Thin, semi-organized clots were adherent to the mitral and tricuspid valves.

The spleen was enlarged and softened. An examination of the right forearm showed rupture of the tendons of the palmaris longus and the outer part of the flexor carpi ulnaris, while some of the deep and superficial flexor tendons were partly ruptured.

The median nerve was stretched and contused, and was of a green color. There was partial rupture of the internal lateral ligament of the wrist-joint tearing off the styloid process of the ulna. There was a "green stick" fracture of the ulna about 5 cm. above its lower extremity and a compound fracture of the radius about 2 cm. from its lower articular surface.

A great many signs of blood poisoning were found in the organs which are not mentioned.

The pons, medulla oblongata and upper cervical cord, together with the right median nerve were removed and put in Müller's fluid for preservation and hardening.

For the detection of lesions, the pons, medulla and cord having been hardened in Müller's fluid and alcohol, were placed in a microtome, and horizontal transverse sections made at short distances all the way from the upper part of the cervical enlargement of the cord to the upper border of the pons. These sections stained by carmine and put through Clarke's process, *i. e.*, absolute alcohol and oil of cloves, were mounted in Canada balsam.

In studying the present case, it will be well to take up first the general lesions and their nature, and then go on to a more minute study of the location and significance of these lesions.

In examining the sections, the first deviation from the normal structure was noticed in the pia mater. It was thickened and vascular. On its free surface there was a considerable recent exudation product, composed of fibrine and cells, and everywhere in its meshes, especially around the blood-vessels, there were a great number of very large cells, (see Fig. 2), some fusiform and some multipolar, very coarsely granular, pigmented and with large oval nuclei. These cells are still better brought out by teasing a little pia mater when they appear as dark, granular, spindle-shaped cells, some 1 mm. long, whose large oval nucleus without a nucleolus appears like a vacuole. (See Fig. 3).

Carmine did not affect these cells at all.

The next lesions found were hyperæmia and thrombosis. In many places the larger vessels seemed filled to repletion, (see Fig. 5), while at some points arteries, veins and capillaries were so universally over-distended with blood elements, as to suggest the possibility of an embolic or thrombotic process. (See Fig. 5). The vessel walls did not seem, as some have described them, thickened, but on the other hand thinned, from continued over-distention.

There was seen at certain spots enormous dilatation of the

perivascular spaces, some of which were partly filled by a small-celled, granular exudation from the vessel, to whose adventitia it was greatly adherent. The walls of these spaces presented a dense infiltrated margin. (See Fig. 5).

The next lesion consisted in cavities of various sizes in the nerve matter, some empty, others filled by a transparent, colloid material containing some small granules, but no cells or nerve debris. The small empty cavities may have been perivascular spaces from which the contained vessel had dropped out, but to the walls of the larger ones might be seen still adherent, some of the colloid material. The walls of these cavities have an organized look, not a ragged disintegrated edge. (See Fig. 4).

The connective-tissue bands were enlarged, and the increase in the number of fixed connective-tissue cells was shown by the nuclear proliferation.

The central canal of the cord was choked with desquammated epithelium, and there was a granular degeneration of some nerve cells.

The first three sets of lesion named will be designated the meningeal, vascular and cavernous, respectively. Having enumerated in general the lesions found in the present case, it will be seen that few of them present the charm of novelty, but it will be the aim of the present paper to elicit some new facts as to the hitherto unknown location of the already known lesions.

The sections are now taken in order from below upwards, and the character and distribution of the lesions will be more minutely studied. The meningeal lesion is confined to the upper part of the cervical cord, ceasing at the lower part of the medulla.

It is especially manifested about the nerve roots, both anterior, posterior, (see Fig. 2), and spinal accessory. In some places the inflamed membrane surrounds and

seems to constrict the roots as they emerge, and in others the new cells have pierced the nerve-bundle.

In sections of the upper cervical cord, few lesions but those of the pia mater were seen. On closer inspection however, changes in the nerve-tissue were noticed which will be better understood by a brief demonstration of the parts. The only respect in which they materially differ from the spinal cord lower down, is this: Nerve fibres are seen curving back from the motor cells of the anterior horns, which, joined by fibres emerging from the central gray matter, pass from the gray matter to a point midway between the anterior and posterior horns. (See Fig. 7).

Here also are seen longitudinal bundles of fibres coming from cells lower down, and from time to time curving over (see Fig. 6), and passing obliquely through the lateral columns to emerge a little nearer the posterior than the anterior roots. (See Fig. 7).

This is the first appearance of the spinal accessory root fibres.

In the central gray matter, from which so many fibres come, existed all along engorged blood-vessels, enlarged perivascular spaces, with exudation and vacuoles. (See Fig. 8).

A peculiarity worthy of note existed in the central gray matter here. In sections all along here for several millimetres there existed, very near the central canal of the cord, an enlarged perivascular space containing a large artery and vein, and several small vessels.

The artery was empty, the vein distended with blood-corpuscles, and around and between all there was in the perivascular space, a coarsely granular material holding in its substance some small cells, (probably white blood-corpuscles), and quite a number of the large black cells above described, as existing in such quantities in the pia mater. Their

relations to the blood-vessels are thus pretty clearly set forth for they are not seen in the fissures, where the pia mater extends without large blood-vessels.

The central gray matter around the enlarged perivascular space and also around the central canal, (the lumen of which was occluded), was thickened and infiltrated with granular matter. (See Fig. 8).

Among the ascending bundles of the accessory root-fibres existed also the same lesions here and there. The fibres traversing the lateral columns were also accompanied for long distances, and crossed here and there by stuffed blood-vessels, enlarged perivascular spaces and cell exudations.

These lesions, of themselves, would be of little moment did they appear elsewhere in the section, but the fact of their being almost exclusively confined to the spinal accessory tract, renders them objects of suspicion. (See dotted line Fig. 7).

The inflamed pia mater affects, as was before stated, the spinal accessory as well as the other nerve-roots.

In sections higher up in the decussation of the pyramids there is quite general distention of blood-vessels, but the gross vascular lesions and vacuoles are almost exclusively confined to the spinal accessory tract. Here and lower down the central canal is filled with desquammated epithelium and the gray matter around it has a dense, granular look.

The next group of sections show the central canal just opening out into the fourth ventricle.

There was seen in the origin and course of the hypoglossal nerve very marked vascular lesions, (see Fig. 9), and, in its course through the olivary bodies, large vacuoles and areas of disintegration.

In the origin and course of the vagus numerous small vascular lesions exist, but no changes of magnitude.

In sections higher up when the glosso-pharyngeal and

higher still, the auditory nerves occupy the posterior and lateral regions of the medulla, occupied lower down by the vagus, the lesions were chiefly confined to the hypoglossal tract, whose long nucleus traverses the whole of this space.

In the course of its fibres through the olivary bodies, the lesions were of more magnitude and consisted of immense perivascular spaces and large vacuoles.

In the glosso-pharyngeal tract the minor vascular lesions were quite abundant, as they were also in the lower facial nucleus. Higher than this, at the lower margin of the pons, in sections bringing the common nucleus of the abducens and facial into view, very few lesions are detected and these are of a vascular nature.

In these sections, the commencement of the motor nucleus of the fifth nerve comes into view.

No marked lesion exists here except a rather granular condition of the large motor cells forming the nucleus.

Higher than this, sections begin to disclose more serious lesions.

In the ascending branch of the fifth pair, there are few lesions except occasional over-distension of the blood-vessels and a few small areas of disintegration. In the course of the descending branch, and among the bladder-shaped cells from which it arises, many vacuoles appear.

In the locus cæruleus many excavations appear among the large, pigmented cells, and also where these cells merge, by losing their pigment and acquiring processes, into the motor nucleus of the fifth, occupying an anterior position.

In the course of these fibres emerging from and crossing the raphe from the opposite locus cæruleus, large vascular lesions exist. (See Fig 5).

Having passed the nucleus of the sixth and seventh sections begin to disclose more serious lesions. In the lower region of the trigeminus there begin to appear here and

there small and large cavities filled with a clear, colloid material. (Figs. 4 and 1).

These are visible to the naked eye, occupy the central motor region of the pons, and increase in number and size till the region of the fifth nerve is reached. They were over twenty in number, one of them being 5 mm. in its longer diameter.

This large vacuole lay in close proximity to the root of the fifth, as it passed from its nucleus forwards through the pons and still nearer the motor nucleus of the fifth.

Many of these large vacuoles lay just in front of, and some in the region of the pons occupied by the motor nucleus, and the scattered large cells giving origin to the motor root of the fifth.

These cavities were formed between the transverse bands (arciform fibres) which pass from the raphe outward, and did not destroy them, but put them on the stretch.

It is in these bands also, that the nuclear proliferation of the connective-tissue is most distinctly shown.

The raphe, too, here presents gross lesions chiefly of a vascular nature.

Higher in the pons the lesions were, vacuoles in the motor tract not going far enough back to implicate the nuclei of the third and fourth nerves, which were perfectly normal.

To summarize then, lesions of various magnitude and nature have been found in the tracts occupied by the trigeminus, part of the facial, spinal accessory and hypoglossal, while slight departures from the normal structure appear in the glosso-pharyngeal and pneumogastric tracts.

These lesions were, let it be understood, of a peculiar nature.

Take for example the lesions found in the whole hypoglossal tract. At no one point do we find the whole or a

large part of the nucleus destroyed, or the course of a great number of fibres interrupted, but throughout the entire tract we find lesions varying in severity from simple vascular engorgement, to areas of disintegration and vacuoles. These, moreover, are scattered here and there among the cells of the nucleus, (see Fig. 9), and along and across the course of the fibres, not causing the destruction of much nerve-tissue, but yet, by their topographical distribution and their proximity to important parts, rendered objects of suspicion.

With one exception the gross lesions were confined to the motor nerves. In the origin and course of the sensory root of the trigeminus many severe lesions were found.

The lesions are, moreover, of such a nature and distribution, that an excitation not an abolition of the functions of a nerve would most likely ensue.

Let it now be seen how far a correlation of lesions and symptoms will carry out this supposition.

In the first place the inflamed pia mater, surrounding the cord and including the anterior, posterior and spinal accessory nerve-roots, claims the attention.

Looking over the symptoms in the present case, those, which will strike the reader as possibly caused by this lesion, are pain and stiffness in the back of the neck, and as these are put down in all text-books as symptoms of cervical spinal meningitis, they can plainly be attributed in this case to the meningeal lesions above mentioned.

In the second place there have been demonstrated vascular and cavernous lesions in the whole spinal accessory tract and also inflamed pia mater about the spinal accessory roots.

The spinal accessory is a motor nerve. It innervates in whole or in part the constrictors of the pharynx, (through the glosso-pharyngeal and pneumogastric), the muscles of the larynx (through the pneumogastric), and, in conjunction

with the cervical nerves, the sterno-mastoid and trapezius.

The irritation of its centre and roots might be supposed to give rise to signs of irritation at its peripheral distribution.

What is there among the symptoms to point to this? On looking them over there are seen dysphagia, spasm of the sterno-mastoid and trapezius, and spasm of the glottis.

Dysphagia is due largely to spasm of the constrictors of the pharynx, while the other two are due to spasm of muscles almost wholly under spinal accessory control.

The unimportant lesions described as occurring in the pneumogastric and glosso-pharyngeal tracts may also have played a reflex part in the causation of spasm of the glottis and dysphagia.

In the third place the excited state of the hypoglossal nerve must find some outward manifestation.

It too, is a motor nerve, and besides the lingual, supplies other muscles brought into play in the first act of delutition.

The spasmodic action of these, too, finds its manifestation in the dysphagia.

The lesions in the lower facial nucleus now demand attention.

There is a well-known disease called labio-glosso-pharyngeal paralysis. In this disease there is developed, sometimes suddenly, more often slowly, paralysis of the muscles of the mouth, tongue and pharynx causing impaired articulation, and imperfect or impossible deglutition.

In this disease the lesion is found in that part of the medulla containing the hypoglossal, glosso-pharyngeal and lower facial nuclei.

The position of this lesion has led many observers to consider this nucleus the centre for the muscles of the mouth, which are the only facial muscles implicated in this disease.

Reasoning thus, and finding this nucleus and not the upper, the seat of lesion in the present case, one is not sur-

prised to find the muscles about the mouth in a state of excitement giving rise to the "*risus sardonicus*."

The gross and minute lesions found in the pons are such as, by their direct action through the motor tract, and indirectly through the sensory origin of the fifth pair, would tend to keep that nerve in a state of perpetual excitement.

This excited state would be outwardly manifested by spasm of the muscles of mastication (temporals, pterygoids and masseters), and in this we see the trismus or lockjaw, the constant, and hitherto unexplained symptom of tetanus.

How much the implication of the respiratory centre in the medulla may have had to do with the Cheyne-Stokes' respiration is doubtful.

This peculiar breathing, although usually attributed to irritation or paresis of the respiratory centre in the medulla, may also be caused by peripheral irritation or impediment which occurred in this case in the form of œdema of the lungs.

The part which the irritated general vaso-motor centre in the medulla played in the causation of the fever is hard to say, but in it the septicæmic condition, present in this case, undoubtedly had its share.

The field, tempting in the extreme, of pathogeny and pathological physiology beckons the investigator on ; but the task of the present paper is ended.

The character and location of the lesions in the present case have been pointed out.

An attempt has been made (how successfully the reader may judge) to find the outward expression of each lesion in a symptom, thus making the case typical.

It is earnestly hoped that this method may be more generally adopted, particular attention being paid by subse-

quent investigators to the medulla oblongata and pons varolii.

In closing, let an answer be given to those who will say, "If such lesions exist in every case, how is it that some recover?" The writer does not think that in non-fatal cases lesions as gross as occurred in the present case existed, but that in them the pathological change stopped one step short of the formation of large exudations and cavities, and was limited to vascular engorgements, perivascular dilatations and exudations, which, by their topographical distribution, were all-sufficient to cause the irritative symptoms occurring in cases of tetanus where recovery supervenes.

EXPLANATION OF PLATE.

Fig. 1. Transverse section of the pons varolii above the point of emergence of the trigeminus, and below the nuclei of the third and fourth nerves.

Showing the size and position of the vacuoles.

Semi-diagrammatic. Enlarged 2 diameters.

Fig. 2. From transverse section of upper cord. P. c.=Posterior column next the posterior root P. r. P. h.=tip of posterior horn of gray matter. L. c.=lateral column. P. m.=Pia mater with new material on its surface and new cells in its meshes, 120 diam.

Fig. 3. Granular, pigmented connective-tissue cells teased out of pia mater, more highly magnified, 750 diam.

Fig. 4. Cavity in the motor tract of the pons between the arciform fibres, a. f., and filled with slightly granular, colloid material, 120 diam.

Fig. 5. Enlarged perivascular space with oblique section of enclosed blood-vessels. This lesion existed in the floor of the fourth ventricle where sensory fibres of fifth pair pass across raphe, (200 diam.).

Fig. 6. Enlarged from Fig. 7. A. h.=Ant. horn. P. h.=Post. horn. C. c.=Central canal. S. a. r.=Spinal accessory root, (120 diam.).

Fig. 7. Transverse section of upper cervical cord. Showing A. c.=Anterior columns; L. c.=Lateral columns, P. c.=Posterior columns. Spinal accessory tract enclosed in dotted lines. S. a. r.=Spinal accessory root, 10 diams.

Fig. 8. Trans. sect. central gray substance of upper cervical cord. C. c.=Central canal. A. f.=Anterior fissure. P. f.=Posterior fissure. A. c.=Anterior gray commissure. P. c.=Posterior gray commissure. A. h.=Anterior horn. P. h.=Posterior horn. C.=Enlarged perivascular space containing A.=Empty artery; V.=Engorged vein. Also other small vessels and large pigmented cells, same as in Figs. 2 and 3.

Fig. 9. Section of hypoglossal nucleus, H. n., low down. C. c.=Central canal. H. r.=Hypoglossal roots. R.=Raphe. B. v.=Stuffed blood-vessel and enlarged perivascular space in hypoglossal nucleus, crossing and accompanying the root-fibres. C.=Capillaries.