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UNIVERSITY
MEDICAL MAGAZINE.

JUNE, 1895



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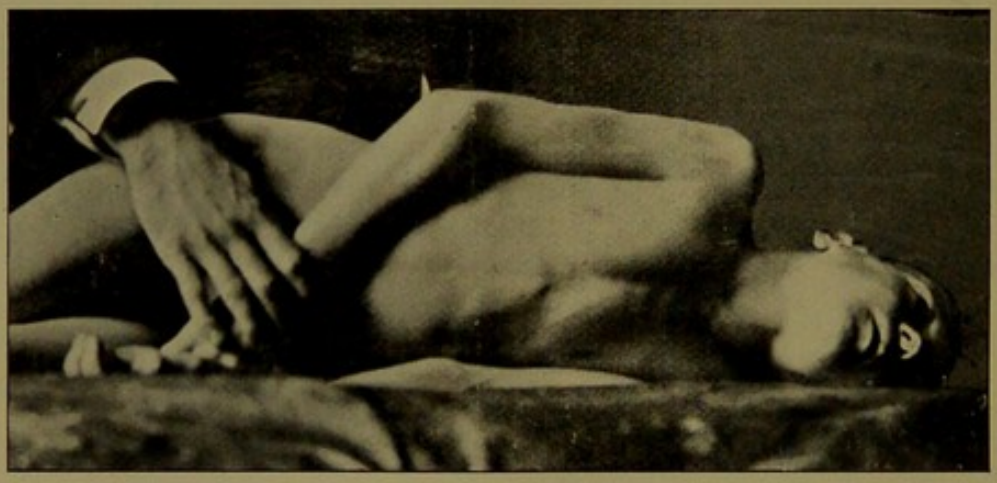

Cephalic Tetanus.

# CEPHALIC TETANUS; GENERAL TETANUS ASSOCIATED WITH HEMIFACIAL PARALYSIS; RECOVERY.[1]

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The coexistence of unilateral facial paralysis with tetanus is a sufficiently peculiar combination to warrant the classification of the cases presenting these two prominent symptoms into a distinctive class, to which has been applied the title of cephalic tetanus, or head tetanus, Kopftetanus, tetanus hydrophobicus, etc. The latter term was employed by Rose from the fact that the spasm of the muscles of deglutition often occasioned such dysphagia as to resemble hydrophobia; but its use is misleading. In the majority of the cases of recovery the spasm did not extend beyond the muscles of the jaw and pharynx; but in the case about to be reported the disease became general, and all the muscles of the body were involved, thus making a case of general tetanus.

The rarity of cephalic tetanus is illustrated by the fact that this patient is the only reported living case in America, while but two other American cases have been placed upon record, that of Phelps,[2] which died on the sixteenth day, and Hunt's case,[3] which died on the sixth day.

In the variety known as cephalic, the tetanus is produced by injury affecting one of the cranial nerves, and is accompanied by hemifacial paralysis. The history of the majority of cases of head tetanus is that of slow onset, and, as in ordinary tetanus, the severity of the disease is determined by the rapidity of onset and of development, the acute cases being speedily fatal, while the chronic ones frequently recover.

*History.*—Joseph T., 12 years of age, was admitted to the hospital wards November 23, 1894, with the following history: Ten days before admission, while at play, he was struck by a small, dirty stick taken from the ground. A slight wound was inflicted near the inner canthus of the right eye, just breaking the skin. The wound was dressed in the receiving ward of the hospital, and the patient referred to the surgical dispensary, where two or three days later the wound was again

[1] Read before the College of Physicians of Philadelphia, March 6, 1895.
[2] Transactions New York Academy of Medicine, November 12, 1888.
[3] Transactions College of Physicians, Philadelphia, 1862, 466.

dressed and found in good condition. The patient did not return again for about a week, when it was noticed that he had a peculiar spastic gait, and that he also had paralysis of the facial nerve on the right side, with trismus. While in the dispensary he had a slight attack of spasm of the muscles of the jaw and neck. He stated that he first began to walk unsteadily about three days before, and found he could not whistle nor open his jaw.

The symptoms probably commenced about one week after the receipt of the injury. The child was taken into the hospital ward at once, on probably the fourth or fifth day of the disease.

On admission, the patient was suffering from peripheral paralysis of the facial nerve on the right side, which was the seat of injury. He had a peculiar jerking gait, and suffered from stiffness of the muscles of the legs, abdomen, back, and neck, with marked trismus. The paralysis involved the fibres supplying the frontal and orbicular muscles, as well as those of the cheek. The risus sardonicus was naturally present only on the unaffected side.

From the history the patient had had no spasm before that seen in the dispensary. Near the inner canthus of the right eye was a red, edematous area, the seat of the wound, now healed. There were no signs of suppuration of the wound, rendering it impossible to obtain material for making cultures. He complained of no pain in the wound or elsewhere. The pupils were normal and reacted readily both to light and distance, while the right eye could not be closed and was the seat of conjunctivitis. Trismus was by this time so marked that the patient could open his mouth only about one-fourth the normal amount, and with difficulty could protrude his tongue, which was coated and at the edges bore evidences of having been bitten. The muscles about the neck were prominent and rigid, especially the posterior group. The chest was well formed, and the head and lungs were normal. The abdominal muscles and those of the back were hard and prominent; otherwise the abdomen was negative on examination. The scrotum and testicles were normal, the prepuce was adherent, but was easily pushed back, and considerable smegma was removed. The temperature was normal and his general condition was good.

The first night in the hospital the patient slept well, but the following night he was quite restless and cried out several times in his sleep. Three days after admission, the eighth of the disease, the trismus was much more marked, the muscles of the neck and back were stiffer, and a tendency to opisthotonos was first noticed. He then for the first time complained of continuous pain in the back and legs, and he was quite rigid all over except his arms, which could be readily used, although his grip was weak and his thumbs presented a tendency

to be drawn into the palms of the hands. He resisted the most vigorous efforts to flex the legs on the thighs, but could slowly flex them voluntarily when told to do so. The right leg was the more rigid.

On searching for the cause of the facial paralysis, an indistinct history of a "running ear" on the right side was obtained, and a careful examination was made by Dr. C. A. Burnett, aurist. The tympanic membrane was found to be normal, as well as the meatus and the auditory canal, and there were no evidences of mastoid disease. On this date the patient bit his tongue twice, but was seen in no marked spasm.

The day following the boy was etherized and the cicatrix loosened by a tenotome, as the scar could not be excised without causing subsequent contraction of the lower lid and eversion. The opisthotonos and trismus increased, and only liquids through a tube could be taken. During deglutition slight spasms of the muscles of the pharynx occurred (hydrophobicus). Pain was complained of principally in the right heel and calf of leg. When the patient was turned on his side, the arching of the back and legs and the retraction of the head were greatly marked and accompanied with flushing of the face.

On November 29, the twelfth day of the disease, the temperature was 101.4° F., pulse 98, and respiration 20. This was the highest temperature during the course of the disease. By December 1 some improvement of the lad's condition was noticed. He slept better, pulse and temperature were normal, he could swallow much better, and the trismus was not so great. At this time he could flex his leg on the thigh (the right one), but not the left. The right side was the side of the injury. The opisthotonos remained about the same, as far as the head, neck, and body were concerned, but he still complained of pain in the back and legs. There were still occasional spasms of pharyngeal muscles, accompanied by flushing of the face during deglutition, and the patient bit his tongue once or twice.

On December 5 (nineteenth day) the trismus and opisthotonos again increased, pains in the right leg and heel were more marked, and the temperature rose to 99.8° F., pulse 100, but weaker, and respiration 24. The entire body was now so rigid that when rolled upon his side the position of the limbs presented precisely that of a frozen cadaver. The feet were extended in a straight line with the legs, and none of the joints of the lower extremities could be flexed except by full force of a man's strength. The bowels were constipated, requiring enemata.

On being visited on the morning of this date he was found by the resident uncovered and with the bedclothes in disorder and soiled by feces and urine, possibly the result of an opisthotonos spasm, although heretofore no general convulsion had been seen. The next day he

complained of pains in the head and back, was quite rigid, and had an involuntary discharge of urine. During the night previous he had bitten his tongue and cried out with pain in his right leg, which was relieved by rubbing. He then improved, and by December 11 his condition was the best since admission to the hospital. There was no change in the facial paralysis. At this time a careful examination of his eye-ground was made by Dr. George Strawbridge, and the following report made: "There is anemia of both optic nerves, especially the left. There is no choked disk and no signs of tortuosity of the veins. Both show physiological excavations." Otherwise the eye-fields were normal, and there was no evidence of intracranial pressure.

An examination of the ears was again made at this time, and the tympanic membrane was found to be normal on both sides, and no pus was found in the external auditory canal. The handles of the mallei were plainly seen. The patient now was on the highway to recovery.

On December 14 (twenty-eighth day), while the paralysis of the face was still complete, reactions to the different electric currents were tested by Dr. James Hendrie Lloyd, as follows: "To faradism on the right side the response is free to a moderate current, perhaps slightly less than on the left. To galvanism the response is free to a medium current (eight cells). Because of the excitation of the patient reactions of the different poles were not taken." Dr. Lloyd kindly saw the case daily, and made many helpful suggestions.

On the following day the patient attempted to get out of bed in the absence of the nurse, and, falling to the floor, received a wound of considerable size on the scalp, which required suturing. For two nights before the accident he had been very noisy, rebellious, and evidently delirious. At one time he was so boisterous that morphia hypodermically was given, and for some time afterwards he was quite stupid, although only a small dose had been employed. For a short time after the accident the patient was much more rigid, but this soon passed away.

On December 19, about one month after admission to the hospital, he was bright, quiet, and rational. There was but slight rigidity, while he could move all his limbs readily and open his mouth easily. He expressed himself as being very hungry,—for a month he could take nothing but fluid food, and would have nothing but milk. At this time he still occasionally voided urine involuntarily. For the next ten days he improved very slowly and was very irritable, seeming much better in the evening than in the morning hours. He was now moved back into the general ward from which he had been isolated. His facial paralysis, which had begun to improve a few days before, was now rapidly disappearing, and there were scarcely any signs of

trismus, but there was still some hardness of the muscles of the back and abdomen. His pain had almost disappeared, and by January 7, 1895 (fifty-two days), the patient was up out of bed. On January 9 he was able to walk alone, but with a halting, spastic gait. The facial paralysis had now almost disappeared, and both eyes could be readily closed. His gait improved slowly, and at the present time the boy is perfectly well.

*Remarks.*—The features worthy of note in the above case seem to be, first, that the patient suffered evidently from general traumatic tetanus of a chronic nature, and recovered after an illness of about two months; secondly, there was associated with it a unilateral facial paralysis, which disappeared with recovery from the tetanus; third, the wound was an insignificant one, but involved the filaments of a cranial nerve; fourth, throughout the course of the disease the patient was not at any time in a markedly asthenic condition nor threatened with asphyxia; fifth, there was a decided improvement of the symptoms for a week after the subcutaneous section of the nerves in the injured area; sixth, the infecting bacillus probably came from the earth and reached the system through the markedly susceptible infection-atrium of the eyelid. The stick may have been soiled by manure also, which is a favorite soil for the growth of the bacillus.

Hulke[1] reports a case of tetanus with facial paralysis occurring in a man, 27 years of age, in which there was both a wound of the finger and of the nose. He had a right-sided facial paralysis with initiative symptoms of trismus. The patient died about two weeks after the receipt of the wound, of asphyxia, due to spasm of the muscles of respiration. There was here complete absence of tetanic spasms of the trunk and limbs, they being of a clonic character.

Nankivell[2] also reports a case of acute traumatic tetanus complicated with facial paralysis, which proved fatal four days after trismus first appeared, and ten days after a slight attack of facial erysipelas from a wound on the bridge of the nose. In this case, also, the paralysis was on the side of the face. There was no opisthotonos, but the patient had several general spasms, and died of asphyxia caused by spasm of the muscles of respiration. The man had attended the out-patient department for his erysipelas, and the day he paid his last visit and was discharged cured being cold and wet, Nankivell offers for an explanation for the facial paralysis the probability of his catching cold and suffering from an inflammation in the aqueductus fallopii, and thus producing the palsy.

[1] London Lancet, July 12, 1882.
[2] London Lancet, July 14, 1883.

Huntingdon[1] also reports a case of tetanus with double facial paralysis which recovered. In this case a lad of 14 years fell and received a small wound on the forehead. Trismus appeared in ten days, and six days later the jaws were firmly locked and there was complete paralysis of both sides of the face. This patient also suffered from frequent cramps in the legs and abdomen, while the arms were but slightly involved. One week after the paralysis became complete it began to subside on the left side, and in another week the right side began to improve. He was discharged in six weeks almost well, and when seen six months later was in perfect health. In this case dysphagia was the prominent symptom.

As will be noted, in none of these cases was there marked opisthotonos; they all suffered from dangerous symptoms of asphyxia, while the present case had very marked opisthotonos, but practically had no embarrassment of respiration, suffering only from pharyngeal spasm occasionally during deglutition.

Gowers, writing of cephalic tetanus (Kopftetanus), called also hydrophobicus by Rose, says that it results from wounds of the head, chiefly of the fifth nerve. Its chief peculiarity is that the initial trismus is associated with paralysis of the face on the same side of the injury. He also states that all parts of the facial nerve are involved, but the cause is not known, as no degeneration of the nerve has been found; hence it has been supposed to be of a reflex character.

Cultures were not made in the present case, as the wound was healed at the time of admission to the hospital. Later, and in the absence of pus, it was deemed unwise to open the wound or to attempt any inoculation experiments on the white mouse, as the bacilli are but infrequently found in the blood.

Examination of the urine on two occasions gave negative results.

The treatment pursued in the case was as follows:

The patient was put to bed in a darkened and isolated room, which was kept at a temperature of about 72° F., and guarded from draughts of air, from noises, and from disturbance. The diet throughout consisted almost entirely of milk, the patient preferring this to other forms of liquid food, and never tiring of it. As there was a marked tendency to constipation, the bowels were kept open by calomel with an occasional enema or brisk saline. The surgical treatment consisted of subcutaneous section of the nerve filaments leading to the cicatrix with a narrow tenotome, which procedure was followed for several days by a subsidence of the symptoms. The medical treatment consisted of the administration of bromides, chloral, and opium, increased from small doses (he was 12 years of age) until he took each twenty-four hours

[1] London Lancet, September 17, 1892.

seventy-five grains of bromide, thirty grains of chloral, and thirty-six drops of tincture opium deodorata. These doses were maintained steadily for four or five weeks. He was never narcotized, and during the day slept but little. On December 15, when he became so noisy and unmanageable, he was given one-eighth of a grain of morphia hypodermically, which was the only hypodermic given. At no time were we called upon to give an anesthetic for convulsions. When he began to convalesce and was on a fuller diet milk-punches containing two drachms of whiskey were given three times a day.

On February 15 the patient was discharged from the wards cured. All signs of facial paralysis were absent and the boy was the picture of health. During the early part of attack an attempt to procure and use the antitoxin for tetanus, as recommended by Tizzoni and Cattani,[1] was made, which proved futile, as none could be obtained. This was unfortunate for the antitetanin, but possibly not for the boy, as he is now entirely recovered, and the plan of treatment by antitoxins is still in its experimental stage. When dealing with a poison of such intense virulence, however, as that of tetanus any and all means of cure should be employed. At the time I was deeply disappointed at the failure to secure the antitoxin, but the favorable result under the plan of treatment adopted is most satisfactory.

Klemm,[2] as well as Brunner, believes that the injury need not be limited to the distribution of the facial nerves, but that the paralysis, which is toxic, may be due to injury of any of the cranial nerves. Klemm very properly draws a distinction between the cases where the spasm remains limited to the muscles affected by the initial symptoms, as in true cephalic tetanus and in those (as in the case under consideration) where the spasm becomes general and in whom the conditions practically become those of ordinary tetanus. It is advisable to distinguish such cases from those not associated with paralysis. It is quite possible that all cases of tetanus commence with these local spasms in the region of the injury; but the local condition, being in the hand or foot, is unobserved, especially in acute cases where more prominent symptoms speedily develop. The facial region being so abundantly supplied with nerve filaments is probably more susceptible to irritation of the tetanus bacillus and is a most favorable infection-atrium. The facial muscle-mechanism is also exceedingly delicate. The presence of this bacillus in the earth must be limited, as a very large proportion of injuries become soiled with earth.

The poison of tetanus has two modes of action; *first*, peripheral,

[1] Medical Press and Circular, London, 1894, LVIII, 153; also Berliner klinische Wochenschrift, 1893, Vol. XXX, pp. 1185, 1215, 1245, 1265; 1894, Vol. XXX, pp. 64, 772.

[2] Berliner klinische Wochenschrift, 1893, p. 63.

by direct alteration of the peripheral nerves; *second*, by reaching the central nervous system through the circulation.

It is difficult to decide why the symptoms in grave cases remain distinctly local. But the slow onset would indicate that an inherent germicidal action was at work destroying the invading bacilli in some instances; while in other cases this phagocytosis is overpowered by the virulence of the poison, and the toxin is admitted to the general system.

Billroth[1] states that the character of the symptoms points evidently to a local contagion.

Not every case of injury of the cranial nerves, even when followed by tetanus, is accompanied with facial paralysis. In a case seen some years ago in consultation with Dr. J. A. Ogden, the wound was in the forehead and had nearly healed when symptoms of tetanus developed, but there was no paralysis. I promptly excised the scar, but the patient died in two days. Dr. Ogden, in a recent letter to me, reports two cured cases of tetanus, but in neither case was there associated paralysis.

Merlich[2] gives a most valuable paper on this subject, and a large number of references to the literature. These, with the references found in the *Index Catalogue* and the *Index Medicus*, show that much valuable work has been done upon this subject. Merlich also gives several excellent microscopical drawings of the appearances found in the motor region of the trigeminus.

Taf (XIII, 1, 2, 3) demonstrates the degeneration and vacuolation incident to this condition. He also agrees that this variety of Hopftetanus may follow an injury to any of the cranial nerves, but is more frequently found after injury to the orbicular and nasal regions.

The influence of the tetanus bacillus in the production of this condition seems thoroughly demonstrated, thanks to the work of Nicolaier, in 1884, the first one to show the causative action of the soil in the production of this disease. His experiments, followed by those of Rosenbach, and especially those by Kitasato,[3] who isolated the pin-shaped bacillus and obtained pure cultures, are most valuable. Guinea-pigs and white mice are the animals especially susceptible to this poison. The pinhead and drumstick appearance of the short rod is evidently due to sporulation at the end or ends. The bacillus is markedly anaërobic.

A number of alkaloids, tetanin, tetanotoxin, muriate of spasmo-

[1] Lehrbuch der allgemeine Chirurgie, S. 507.

[2] Archiv. Psychiat., 1892, XXIII, p. 672.

[3] Deutsche medicinische Wochenschrift, 1889, XV, No. 31, p. 635; also Zeitschrift für Hygiene, 1890. Bd. VII.

## ERRATUM.

THE twenty-fifth line on page 8, "Taf (XIII, 1, 2, 3) demonstrates," etc., should read "Taf XIII, 1, 2, 3 demonstrate," etc., and should be a continuation of the previous paragraph.

toxin have already been separated, and it is probable that in time the product of the bacillus will be employed for the cure of this disease, as is indicated in the present use of the various antitoxins. The use of tetanus antitoxin has been chiefly advanced by Tizzoni and Cattani.[1]

Brunner,[2] by a series of experiments, determined that injections of cultures of the tetanus bacillus into animals, or of the cultures free from the bacilli, will produce tetanus, the spasm starting after a few hours in the muscles in the region injected. If the injection is made in one side of the face,—*e.g.*, the left, this is first involved, then the right side of the face, then the left forelimb, then the right, and then the rest of the body.

Post-mortem examinations of the animals revealed no characteristic microscopic changes in the nervous system or any of the tissues. He gives a number of excellent illustrations of the effects on rabbits and guinea-pigs.

Injections of a considerable amount of the poison in the face often determine a paralysis of the muscles in the immediate region ; injection into the blood-current or peritoneal cavity produces general tetanic manifestations, not preceded by local spasm. The bodily temperature of the injected animal sank steadily. Attempts to obtain from the tetanus poison separate alkaloids, as tetanin, tetanotoxin, spasmotoxin, as causes of the different manifestations, were not successful. The symptom-complex is produced by the undivided filtrate of cultures.

The tetanus poison circulates in the blood of animals suffering with tetanus ; it is also found in the urine and saliva.

Injections of the poison into a motor sensory nerve—*e.g.*, sciatic—produce the same effect as a subcutaneous injection, first local, then general spasm. Injection under the dura mater produces, first cephalic, later general tetanus.

The tetanus poison cannot cause muscular spasm by direct action, but must act through the nervous system ; and if a motor nerve to a region is cut, injections of the toxin in this region cannot produce spasm. The centres in the medulla, pons, or cord must also be intact for the production of spasm, but the higher centres are not essential. The poison does not act reflexly, but by a direct action on the central motor apparatus, probably reaching this along the nerve-trunks rather than by the blood or lymph-channels. It can ascend the motor as well as the sensory tract.

The cause of facial paralysis has given rise to much discussion.

[1] Medical Press and Circular, London, 1894, Vol. LVIII, p. 155. Berliner klinische Wochenschrift, 1893, Vol. XXX, pp. 1185, 1215, 1245, 1265 ; 1894, Vol. XXX, pp. 64, 72.

[2] Exper. u. klin. Studies ufer de Kopftet.; Beitrag für klinische Chirurgie, Bd. LX, pp. 83 and 269; Berliner klinische Wochenschrift, 1891, Vol. XXVIII, p. 881; Deutsche Zeitschrift für Chirurgie, Bd. XXX, S. 574.

In my own case a most careful investigation was made to exclude the possibility of its being merely a coincidence due to independent causes, and the absence of middle-ear disease, intracranial growths, brain abscess, etc., were thoroughly demonstrated.

In the present stage of our bacterial knowledge it seems much more rational to refer it to a direct toxic effect of the poison acting upon the filaments of the seventh and fifth nerves rather than to a "cold, rheumatism, etc."

I have succeeded, with the kind assistance of Dr. Alfred Hand, Jr., in collecting 75 cases of cephalic tetanus; 57 males and 13 females are noted. In 45 acute cases (counting those acute ones in which the symptoms have developed within the first week), 39 died and only 4 recovered; a mortality of 90 per cent. Of 32 chronic cases (where the symptoms arose after the first week), 8 died and 24 recovered; a mortality of 25 per cent.

Gowers states that all cases over 25 years of age died; but my table shows that of acute cases (one week) there were 2 recoveries in 32 cases in persons over that age, and 13 recoveries in 15 persons who were classed as of the chronic variety. Under 25 years, in 12 acute cases there were 2 recoveries, and in 16 chronic cases, 13 recoveries.

In the 75 collected cases when the spasm extended to the muscles of the jaw, throat, and neck they have been denominated "local;" while if opisthotonos and spasm of limb muscles occurred they were denominated as "general." There were 29 cases of the former condition and 46 of the latter. Of the "local" cases, 14 recovered and 15 died; while of the "general," 14 recovered and 32 died.

In this form of tetanus, as well as in general tetanus, the actual mortality is far higher than statistics will indicate, as in my personal experience I have certainly seen twenty deaths from tetanus to one of recovery, and yet none of the twenty fatal cases are on record.

| No. | Reporter. | Reference. | Sex. | Age. | Acute or Chronic. | Local or General. | Result. |
|---|---|---|---|---|---|---|---|
| 1 | In Richter's Chir. Bibliothek, 1791. | Cited by Brunner, Beitr. f. klin. Chir., 1892–93. | M. | 10 | Acute. | General. | Recovery. |
| 2 | Hurst. | Trans. Phila. Coll. Phys., 1892, III, 465. | M. | 16 | Acute. | General. | Death. |
| 3 | Larrey. | Cited by Rose. | M. | 30 | Acute. | General. | Death. |
| 4 | Zuicke. | Charité Ann., IX, p. 366. | F. | 39 | Chronic. | Local. | Recovery. |
| 5 | Jacksch. | Wien. med. Presse, 1888, No. 18. | F. | 38 | Chronic. | Local. | Death. |
| 6 | Duplay, cited by Séreins. | De la contracture réflexa d' origine traumatique, Thèse, Paris, 1880, No. 435. | M. | 19 | Chronic. | Local. | Recovery. |
| 7 | Nicolaysen, cited by Séreins. | De la contracture réflexa d' origine traumatique, Thèse, Paris, 1880, No. 435. | M. | 23 | Chronic. | Local. | Recovery. |
| 8 | Terelon and Schwarz. | Revue de Chirurgie, 1888, No. 1. | M. | 16 | Acute. | Local. | Death. |
| 9 | Travers, cited by Pflüger. | Die sensorieschen Functioners des Ruckenmarke, p. 81. | M. | 11 | Chronic. | General. | Recovery. |
| 10 | Larrey, | Memoires de Chir. melit., III, p. 307; also Pflüger, loc. cit. p. 82. | M. | . . | Chronic. | General. | Death. |
| 11 | Crossonard. | Étude à l'Appre de l'Origine infect. du tetanos, Thèse, Paris, 1887. | F. | 33 | Acute. | Local. | Recovery. |
| 12 | Dumshard. | Cited by Séreins, loc. cit. | M. | 55 | Chronic. | Local. | Recovery. |
| 13 | Kirchhoff. | Berl. klin. Wochen., 1879, No. 25. | F. | 52 | Acute. | General. | Death. |
| 14 | Pollock. | Cited by Rose. | M. | 33 | Chronic. | . . . . . | Death. |
| 15 | Rockliffe. | Brit. Med. Journ., 1890, No. 1591, p. 86. | M. | 7 | Chronic. | General. | Recovery. |
| 16 | Middledorpf. | Bresl. ärztl. Zeitschr., 1883, p. 8. | M. | 9 | Chronic. | General. | Recovery. |
| 17 | Gütterback. | Arch. f. klin. Chirurg., B. 30, p. 836. | M. | 21 | Chronic. | General. | Death. |
| 18 | Von Wahl. | St. Petersburg med. Wochen., 1882, No. 39. | M. | 41 | Acute. | Local. | Death. |
| 19 | Klemm. | Deutsche Zeitsch. f. Chir., 1889, XXIX, 3, p. 168. | F. | 40 | Chronic. | Local. | Recovery. |
| 20 | Hadlick. | Berl. klin. Wochen, 1885, XXII, p. 266. | M. | 2⅓ | Chronic. | General. | Recovery. |
| 21 | Rose. | Ibid., p. 86. | M. | 28 | Acute. | General. | Death. |
| 22 | Rose. | Ibid., p. 88. | M. | . . | Acute. | General. | Recovery. |
| 23 | Kirchner. | Aerztl. Bericht. Preuss Feld-dazarth, etc., Erlangen, 1872. | . . | . . | Acute. | General. | Death. |
| 24 | . . . . . | St. Bartholomew's Hosp. Rep., 1874, Appendix, p. 40. | . . | . . | Acute. | Local. | Death. |
| 25 | Von Langenbeck. | Berl. klin. Wochen, 1869, No. 35. | M. | 7 | Acute. | General. | |
| 26 | Lehrnbecher. | Bar. ärztl. Intelligenzblatt, 1882, No. 46. | M. | 49 | Acute. | General. | Death. |
| 27 | Mayer. | Prag. med. Wochen, 1883, No. 34. | M. | 18 | Chronic. | General. | Recovery. |
| 28 | Bond. | Brit. Med. Journ., November 10, 1883, p. 918. | M. | 23 | Chronic. | General. | Recovery. |
| 29 | Bernhardt. | Zeitschr. f. klin. Med. 1884, p. 410. | M. | 32 | Acute. | General. | Death. |
| 30 | Maissurianz. | St. Petersburg med. Wochen, 1887. | M. | 35 | Chronic. | Local. | Recovery. |
| 31 | Triglis. | Gironale di Neuropatologia, 1884, I, p. 80. | M. | 50 | Chronic. | Local. | Recovery. |
| 32 | Genffré. | Lo Sperimentale, 1887, p. 380. | M. | 51 | Chronic. | . . . . . | Recovery. |
| 33 | Lannois. | Rev. de Méd., 1890, II, p. 168; also Lyon Médicale, 1889, LXII, p. 380. | M. | 69 | Acute. | Local. | Death. |
| 34 | Charcot. | Soc. de Chir., October 10, 1888. | M. | . . | Acute. | General. | Death. |
| 35 | Perret. | Cited by Albert, Étude sur tetanos cephalique, p. 26. | M. | 59 | Acute. | General. | Death. |
| 36 | Phelps. | New York Acad. of Med., November 12, 1888. | M. | . . | Acute. | General. | Death. |
| 37 | Remy and Villar. | Gaz. des Hôp., 1888, No. 142. | M. | 36 | Acute. | General. | Death. |
| 38 | Brennecke. | Inaug. Dissert., Göttingen, 1890. | M. | 12¼ | Chronic. | General. | Death. |
| 93 | Widenmann. | Würt med. Correspondenz-blatt, 1889, No. 8. | M. | 8 | Acute. | General. | Death. |
| 40 | Roberts. | Lancet, July 11, 1891. | M. | 64 | Acute. | General. | Death. |
| 41 | Brunner. | Berl. klin. Wochen., 1886, No. 7. | M. | 27 | Acute. | Local. | Death. |

| No. | Reporter. | Reference. | Sex. | Age. | Acute or Chronic. | Local or General. | Result. |
|---|---|---|---|---|---|---|---|
| 42 | Brunner. | Berl. klin. Wochen., 1886, No. 7. | M. | 38 | Acute. | General. | Death. |
| 43 | Brunner. | Beitr. f. klin. Chir., 1892, p. 291. | M. | 19 | Chronic. | General. | Death. |
| 44 | Van Spanje. | Weekbl. v. nederl. Tijdsch voor Geneestr., 1891, II, No. 10. | M. | 60 | Acute. | General. | Death. |
| 45 | Billroth. | Chirurg. klin. Wien, 1868, No. 77. | M. | 37 | Acute. | General. | Death. |
| 46 | Nankivell. | Lancet, July 14, 1883. | M. | 33 | Acute. | General. | Death. |
| 47 | Oliva. | Gazette de Chir., 1886. | M. | 52 | . . . . . | Local. | Death. |
| 48 | Wagner. | Schmidt's Jahrbuch, 1884, p. 139. | M. | 42 | Acute. | Local. | Death. |
| 49 | Nerlich. | Inaug. Dissert., Halle, 1892. | F. | 46 | Acute. | General. | Death. |
| 50 | Thenée. | Berl. klin. Wochen, 1880, p. 531. | F. | 55 | Acute. | General. | Death. |
| 51 | Terrier. | Cited by Sériens, loc. cit., p. 31. | M. | 49 | Chronic. | Local. | Recovery. |
| 52 | Gosselin. | Cited by Terilon and Schwarz, loc. cit. | M. | . . | Acute. | General. | Death. |
| 53 | . . . . . | St. Thomas's Hosp. Rep., X, p. 461. | . . | . . | Chronic. | General. | |
| 54 | Sériens. | L'Union Méd., December, 1886. | M. | 59 | Acute. | Local. | Recovery. |
| 55 | Buss. | Centralbl. f. Chir., 1890, p. 831. | . . | . . | Acute. | General. | Death. |
| 56 | Guastalla. | Cited by Rose, p. 89. | F. | 40 | . . . . . | General. | Recovery. |
| 57 | Pflüger, loc. cit. | Cited from Med.-Chir. Trans., Vol. IV, p. 25. | F. | 50 | . . . . . | General. | Recovery. |
| 58 | Zsigmondy. | Aerztl. Bericht. v. allg. Krankenhaus, Wien, 1879, p. 76. | F. | 41 | . . . . . | Local. | Death. |
| 59 | Buisson. | Gazette hebdom., 1888, No. 3. | M. | 60 | Chronic. | General. | Recovery. |
| 60 | Wartmann. | Cited by Brunner, loc. cit., p. 310. | M. | 11 | Acute. | General. | Recovery. |
| 61 | Behr. | Inaug. Dissert., Tübingen, 1891. | M. | 25 | Acute. | General. | Death. |
| 62 | Brunner. | Loc. cit., p. 314. | M. | . . | Acute. | Local. | Death. |
| 63 | Brunner. | Loc. cit., p. 314. | M. | 13 | Chronic. | General. | Recovery. |
| 64 | Poisson and Rappin. | Gaz. Med. de Nantes, 1893-94, XII, p. 93. | F. | 30 | . . . . . | Local. | Recovery. |
| 65 | Caird. | Edinburgh Hosp. Rep., 1893, I, p. 491. | M. | 12 | Acute. | General. | Death. |
| 66 | Navarre. | Lyons Médicale, 1894, LXXV, p. 152. | F. | 56 | . . . . . | Local. | Death. |
| 67 | Reclus. | Cliniques Chirurg., Paris, 1894, 8vo., p. 150. | F. | 56 | . . . . . | Local. | Recovery. |
| 68 | Reclus. | Loc. cit. | M. | 43 | . . . . . | Local. | Death. |
| 69 | Reclus. | Loc. cit. | M. | 19 | . . . . . | Local. | Recovery. |
| 70 | Reclus and Chaput. | Loc. cit. | . . | . . | . . . . . | . . . . . | |
| 71 | Caird. | Loc. cit. | M. | 26 | Acute. | General. | Death. |
| 72 | Caird. | Loc. cit. | M. | 57 | Acute. | Local. | Death. |
| 73 | Campos. | Cronicl. med.-quir. de Hebaus, 1890, XVI, p. 255. | M. | 39 | Acute. | Local. | Death. |
| 74 | Hulke. | Med. Press and Circ., London, 1882, XXIV, p. 25. | M. | 27 | Acute. | General. | Death. |
| 75 | Willard. | Trans. Coll. Phys. Phila., 1895; also UNIV. MED. MAG., 1895. | M. | 12 | Chronic. | General. | Recovery. |