THESIS FOR THE DEGREE OF M. D.

A CLINICAL STUDY OF KOPFTETANUS.

by

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The chief symptoms of Tetanus have been known since the days of Hippocrates, and apparently that ancient physician recognized an acute or unfavourable, and a chronic or favourable form. Modern writers on this disease distinguish an acute form, with short incubation period, with rapid course and generalized spasms, ending usually in death. And in the next place a chronic form, of very variable type, well described by Rose as "lentus incompletus", of much more favourable prognosis.

Kussmaul has also described a special kind of mild tetanus - "Abortive tetanus" - where transient spasm of the muscles of neck and jaw may be the only symptoms.

These varieties appear to be only a variation in degree, and are far surpassed in interest by another form now generally known as Kopftetanus.

Kopftetanus was first described as a distinct variety of tetanus by Rose in 1870. He had first observed a case in 1863, but he cites a case observed by Pollock in 1847, and one by Langenbeck in 1869. Cases had, however, been observed by Von Richter (cited by Brunner) in 1791, by Mitchell in 1813, and by Humphry in 1837 and 1858.

Thus/
Thus, though Rose was the first to draw special attention to this variety of tetanus, and to give it a name he was by no means the first to give clinical description of a case.

Since 1870 a variety of names have been suggested, Kopftetanus or Cephalic Tetanus.
Tetanus Hydrophobicus (Rose)
Cephalic Tetanus with Facial Paralysis (Villar)
Bulbar Tetanus (Janin)
Tetanus Paralyticus (Klemm)

The last mentioned name, for reasons yet to be mentioned, is the best. The term in general use, however, is Kopftetanus: and consultation of the literature under this heading soon shows, that it is made to cover two classes of cases. In both classes, there is history of a wound in the region of head or face, there are tetanic spasms either localised to muscles of face, jaw, and neck, or generalised and there is the frequent presence of dysphagia, or of dyspnoeic attacks. We find, in short, the symptoms of ordinary tetanus, with perhaps a tendency to localisation of the spasms in the neighbourhood of the wound, and to dysphagic and suffocative phenomena.

In/
In one of these classes, by much the larger, we find mention made of an altogether distinguishing symptom, viz. paralysis, affecting as a rule, the facial nerve.

I do not think that the non-paralytic group can be so satisfactorily distinguished from ordinary tetanus as to deserve a special name, and it should not be included under the term Kopftetanus, if that name is to be applied to cases exhibiting paralytic phenomena.

In the following discussion of the cases collected from the literature, these two groups of cases will be treated separately.

In February 1903, while acting as resident-physician with Dr. Andrew Smart. I had the opportunity of observing a case of Kopftetanus, which came under his care. By Dr. Smart's kind consent, I am enabled here to give an account of this unpublished case.

Alexander McPherson, 31, single, railway porter.
Admitted to Ward 32, R.I.E. February 24, 1903
Complaint Stiffness of jaw, back, and legs, with cramping/
/cramping pains in the muscles. Twisting of face
to the right and inability to close the left eye.

**History of Onset** On February 6 patient was moving
grain-sacks from a railway wagon into a cart. A
sack falling against him, caused him to fall and to
strike his left temple on the edge of the wagon.
He sustained two small cuts, described later, but
paid no attention to them, and went on with his
work.

On February 8 he noticed a twitching of the left
side of his face, and that also he could not close
his left eye. He began to be much annoyed by dust
e tc. getting into his left eye. The facial
twitching continued until February 11, when it ceased;
the mouth became drawn to the right side, and food
tended to accumulate in the left cheek. At this date
he had no difficulty with his speech, and could move
his jaw freely. He was able to work, and was only
troubled by dust getting into his left eye.

On February 18 the muscles of the lower jaw began
to feel stiff and sore, when patient attempted to
eat solid food. This stiffness increased until on
February 20 he became unable to open his mouth, and
could only take fluid food. Two or three times he
felt/
felt his jaw becoming tightly clenched and experienced severe cramp-like pain in the muscles of the jaw, at the same time the right side of his face twitched. Also the muscles of his back and of his thighs began to feel stiff and sore. On this day he first sent for his doctor, who prescribed medicine, which eased him.

February 21. Patient felt cramping pains between his shoulder-blades, at intervals of a few seconds.

Similar pains began also to affect the muscles of his neck. When they came on, his head was drawn backwards and his back was arched, this caused him great agony. The spasms lasted some seconds.

Since this date his head has been turned to the right, and even with the greatest effort he could not turn it to the left past the mid-line of his body.

February 22. The muscles of neck, back, and legs were so stiff that he was unable to move in bed. He began to suffer from cramping pains in his thighs and calves as well as in the back and neck. These pains varied in site, and severity, in duration and frequency. Average frequency about one/
/one attack per minute, average duration about five seconds.

February 23. No improvement.


Previous Illness.

Has had a leg fractured twice.

Influenza 4 weeks before accident. No venereal history.

Personal History. Nothing of note.

Family History. Nothing of note.

General Appearance and Condition on Admission.

Patient, who looks extremely anxious and distressed, lies in bed with his legs and back rigid and with his head semi-rotated to the right. He is unable to turn his head to the left past the mid-line, nor can he bend his neck and back, nor flex his hip and knee joints.

A small scar with dried scab is seen at outer angle of left orbit, also there is a small scab on the left side of forehead above left eyebrow. Both of these are so small as to be scarcely noticeable, but patient says that these are the places where he cut himself on February 6, i.e. 19 days ago.
ago.

The left eye is permanently open, if patient is asked to shut it, the eyeball tends to roll upwards under the lid. The right eyelid droops concealing the right eye, which, on raising the eyelid, is found to show a degree of internal strabismus. He has difficulty in fixing his eyes on an object and has marked nystagmus in all directions. The left half of forehead is smooth, and the left eyebrow cannot be elevated. The right half of the forehead shows marked furrows and the right eyebrow can be raised. The right angle of the mouth is markedly drawn to the right side, the left cheek and the left half of lips are immobile and flaccid. The facial folds are all obliterated on the left, whilst, on the contrary, they are very pronounced on the right side of the face. The nose is slightly drawn over to right side.

The jaw is tightly clenched; the tongue cannot be protruded, but appears to be freely movable within the buccal cavity. Patient can speak fairly well through his clenched teeth.

The normal lordosis of lower dorsal spine is markedly increased, the closed fist can be passed freely under the region of patient's back, as he lies/
lies in the dorsal decubitus. The muscles of the neck and back and thigh feel hard, particularly those of lumbar and gluteal regions.

Apart from this tonic contraction of various groups of muscles, the patient at intervals of one to two minutes suffers from tetanic spasm ("Crisés" or "redoubléments Convulsives" of the French) of the muscles of the trunk and neck, also sometimes of the muscles of the lower extremities. (Gluteal muscles and Quadriceps Extensor of thighs chiefly affected). These spasms caused the patient intense cramping pain; they vary in extent and severity, every fourth or fifth attack of spasm being more severe than the others. When spasm affects the muscles of the back the already marked lordosis is greatly increased, and the head is forcibly bent backwards. The thoracic and abdominal muscles also become rigid and prominent.

When patient feels the spasm coming on, he makes a deep inspiration, and the breath is held till the spasm is over. The spasm lasts about five seconds.

When the lower extremities are affected, the groups of muscles are seen to stand out in rigid spasm, e.g. the attachments of the gluteal muscles can be very clearly seen during a spasm. Spasms affecting/
affecting the trunk muscles, and those affecting the muscles of the lower extremities, usually occur separately, but occasionally they occur together thus constituting a more severe tetanic seizure. The right arm is sometimes drawn up into a flexed position during spasm of the trunk muscles, but it does not exhibit the same stiffness and rigidity as are seen in the legs. The left arm is absolutely unaffected by contracture or spasm.

The right half of the face shows occasional muscular twitching, sometimes accompanying spasm of other muscles, sometimes alone. The muscles of left half of face were absolutely unaffected by spasms. The muscles of jaw in addition to their tonic contracture, are sometimes affected with a spasm, which gives patient intense pain and makes him feel as though his teeth would break with the strain. A spasm of jaw muscles is usually accompanied by spasm of right facial muscles.

If either knee be forcibly flexed, the right one is the more rigid, the adductor and flexor muscles of the thigh can be seen to stand out very prominently; they feel almost as hard as iron, and in places feel knotty. This knotting of the muscles can be best felt in the left thigh. The calf muscles also show this peculiar knotting.

The knee jerks cannot be elicited, the plantar reflex/
reflex is exaggerated on both sides. If either sole be tickled a spasm of varying severity and extent is produced.

Owing to the grave condition of the patient no prolonged physical examination was justifiable.

**Circulatory System.**

No evidence of cardiac disease.

Pulse beats regular in time, equal in force.

Tension moderate. Rate 90 per min.

During a spasm rate and tension increased.

**Respiratory System.**

Apparently healthy.

**Nervous System.**

Particulars as to facial paralysis, ptosis, and muscular spasm already given.

**Urine.** contained a small quantity of albumin.

No blood. Heavy deposit of urates.

**Blood.**

Red Blood Corpuscles. 4500,000.

White Blood Corpuscles. 10,000.

Haemoglobin. 70 p.c.

Leucocytosis. due to increase in polymorphonuclear leucocytes. No increase of eosinophiles.

**Course and Treatment.** (very briefly).

February 24, 1 p.m. Patient is having spasms every one or two minutes; these are mostly limited to one or two groups of muscles, but about one in five is /
is generalised. Bromide of Potassium 30 grs. and Chloral Hydrate 20 grs. ordered four hourly.

Patient takes fluid nourishment well. About 10 p.m. the spasms began to get less frequent, and from 2-15 a.m. to 6-40 a.m. patient had only seven spasms, in the interval between which he slept quietly.

February 25. Patient feels greatly refreshed by his snatches of sleep. Temperature, which last night was 100°, is now normal. In forenoon spasms became slightly more frequent, about every quarter of an hour, but were not severe. In the afternoon patient was given a subcutaneous injection of Tetanus Antitoxine 30 c.c. (Tizzoni). In afternoon, condition much the same but had one severe spasm affecting muscles of neck, trunk and lower extremities, urine passed involuntarily during the attack.

During each spasm the pulse rate goes up from 90 to 120 per minute, and the respirations from 30 to 42 per minute. Profuse sweating follows the more severe spasms.

8-30 p.m. Patient given a ¼ gr. Morphia Hypodermically.

11-30 p.m. Very severe attack - Features distorted by spasm of right facial muscles.

Left eye which remains open and staring, is rotated upwards and outwards. The jaws are so tightly clenched that the teeth can be heard/
Patient drank a cup of beef tea, and immediately after said he felt sick. Suddenly his breathing stopped, the muscles of the right side of the face became spasmodically contracted, and the arms were rigidly flexed over chest. The back muscles were not affected and the jaw was not clenched. Face rapidly became cyanosed. Chloroform administration was at once attempted, but as respiration had ceased, and as artificial respiration failed to induce air to enter the lungs, this treatment was of no avail. The thorax was absolutely rigid. The pulse was feeble but was felt to go on beating for 40 seconds after respiration had stopped.

Post mortem Examination (performed by Dr. Beattie)

Rigidity general and marked. Lividity marked.
Pin point punctured wounds - one above left orbit, one at its outer angle.

Heart shews commencing hypertrophy of left ventricle.


Spleen Soft. Shews perisplenitis.

Kidneys Tubules swollen, glomeruli congested, Pyramids congested. /
Section of branches of paralysed facial nerve from Dr. Smart's case.

x 75. Haematoxylin. (Photo by Richard Noss.)
congested.


No haemorrhages, no gross lesion, no meningitis. Cultures, made from wound and from blood, negative.

I made sections of the facial nerve, but no definite microscopic changes were determined. The micro photograph on the opposite page was taken from a section through some of the smaller divisions of the facial nerve, stained with Haematoxyline and Eosine.

The electric reaction of facial muscles could not be determined, as the condition of patient did not admit of the application of the electric current.

Contrasted with the average case, the chief points to be noted about Dr. Smart's case are -

1. The early facial paralysis, preceeded by twitching of facial muscles on same side. In only 11. p. c. of the collected cases does facial paralysis preceed trismus, and in no other cases does facial twitching preceed paralysis of facial muscles.

2. The presence of ptosis is unusual, this phenomenon is only noted in four other cases, in two of which the ptosis was on the same side as the
facial paralysis. Strabismus is only noted in two other cases.

3. No mention is made of the presence of nystagmus in the records of any other case.

4. During a severe spasm on Feb. 25, there was apparently temporary spasm of the superior of the right rectus, causing temporary rotation of left eye upwards.

Taking the first appearance of the facial paralysis as evidence of the presence of tetanus toxine, the incubation period was much shorter than the average, being only two days. It is remarkable that the first tetanic symptoms should only appear 12 days later. The absence of difficulty in swallowing was favourable as regards prognosis, as also the very infrequent attacks of dyspnoea. (Only two, including the fatal one, during the 9 days on which the tetanic phenomena were present).

The patient's age falls in a decade (30-40) in which the mortality is high, 69.2 p.c. The early appearance of facial paralysis is associated with unfavourable cases.

There is nothing unusual in the insignificance of the wound, nor in the negative result of bacteriological examination.

There appeared to be marked amelioration of symptoms under the influence of Bromide and Chloral; but there was a tendency, noted in many other cases.
for the attacks, diminished in frequency, to become more severe in type. Antitoxine was given too late in the case, for much good to be expected from its use. The patient died from asphyxia, probably due to spasm of the diaphragm and the thoracic muscles. Death in these cases is more often due to heart failure.

By the kind consent of Dr. Gillet of Andover, Hants, I am able to make mention of another unpublished case, which came under his care about four years ago.

On Wednesday October 10, 1900, at 2p.m. a lady aged 65 was driving along a road, when the horse shied, and she was thrown head first out of her dog-cart. Shortly after, Dr. Gillet saw her, and found that she had sustained a scalp wound, about four inches long, down to the bone, and situated in the right fronto-parietal region. An anaesthetic was given, and the wound thoroughly cleansed from the road mud, with which it was contaminated.

Thursday. Progress was satisfactory: Temp. normal. Pulse 90.

Friday. Patient complained of pains in the region of muscles of lower jaw, which she attributed to bruises. In evening the muscles of lower jaw were stiff and the mouth could only be opened/
opened with difficulty. The wound was opened up
and strong antiseptics were again applied.
Saturday. In the morning patient, who had slept the
previous night, noticed that she could not shut
her right eye. She was unable to open her mouth.
On examination the right facial muscles were found
to be flaccid, the right half of the forehead smooth,
the right facial furrows lost. On the other hand
the left half of forehead shewed marked wrinkling,
the furrows of the left half of the face were
exaggerated, and the mouth drawn markedly to the left
side. This condition was evidently due to a
complete right facial paralysis and to contracture of
the left facial muscles. The right pupil was
markedly dilated and did not respond to light nor
accommodation. Trismus was marked, and the neck
muscles were stiff. Patient was given Bromide and
Chloral, large doses, and antitoxine was sent for.
Sunday. Patient had a fair sleep. Condition
unchanged. In the evening tetanic spasms commenced,
affecting the muscles of jaw, neck and trunk,
frequency about every half-hour.
Monday. Sleepless night; spasms more severe,
muscles of thighs and calves also affected by them,
frequency/
In the evening patient began to find difficulty in swallowing. Any attempt to swallow some liquid brought on generalised spasms, affecting especially the respiratory muscles. There was marked opisthotonos during the spasms which lasted a few seconds. The reflex irritability became very marked, very slight external stimuli tending to excite attacks of spasm. Antitoxine treatment commenced.

Tuesday. No improvement, arm muscles also affected by spasms. Temperature 104°. Pulse 120.

Wednesday. A week after the commencement of the symptoms, patient died from heart failure during a spasm of great severity.

Consent for post-mortem examination could not be obtained.

This case is typical of the most severe class of Kopftetanus. Two points may be noted as unusual. 1st. Dilatation of the pupil on the same side as the facial paralysis, 2nd. Affection of the arms by tetanic spasm.

In addition to these two unpublished cases, I have found under the heading of Kopftetanus, in the literature/
literature accessible to me, 102 cases described. The descriptions are in many instances very imperfect and very brief, thus being useless for purposes of comparison. I do not intend to give all these cases in full, but append a summary of them in tabular form of cases showing paralyses, which are included in Brunner's summary.

Since the publication of three cases by Rose in 1872, collections of the recorded cases have been made by several writers, of which I shall mention three.

In 1888: Villar gave a critical account of 29 cases, with personal observations on one case. His paper is constantly referred to by French writers on this subject. He limited his cases to those with facial paralysis, and employed the lengthy term "Tetanus Céphalique avec paralysie faciale".

In 1892: Brunner of Zurich published his very important monograph on Kopftetanus. He includes cases with and without facial paralysis. He also gives an account of the elaborate inoculation experiments on rabbits and guinea pigs, which he made with a view to the production of an artificial Kopftetanus. In this however he was not successful, though he proved that under certain conditions a large injection of crude toxine may cause a localised paralysis of the muscles in immediate neighbourhood of/
of the injection. He gives summaries of 62 cases.

In 1895: Willard, in America, published an account of a case observed by himself, and gave a table of 75 cases, which is simply Brunner's collection with a few additional cases. Beyond the few headings of the table no summary is given of the cases.

Of the 102 cases of Kopftetanus, of which I have found descriptions, 21 have quite clearly suffered from no symptoms of paralysis, and must be considered separately. If the latter are entitled to a special name, and that name is to be Kopftetanus, the former class would be better designated by the term Tetanus Paralytius. Taking the larger and more important group first, we find 83 cases in which there is evidence, more or less satisfactory, of paralysis affecting one or more of the cranial motor nerves.

1. The sex. Of 80 cases, where sex is mentioned, there were 66 males and only 14 females. This disparity is accounted for by the greater exposure of males in their occupations to possible infection with the tetanus bacillus. Of the 14 females 5 were German and 4 were French, all of these being outdoor workers. No fewer than 12 boys under 16 were/
21.

were affected, while no girl of corresponding age suffered from this disease. The boys were nearly all infected by falls on the ground, by wounds from stones, or scratches with thorns etc.

2. **Nationality.** While not of much importance, may be noted:— of 80 cases: German 20, French 19, English 13, Austrian 8, Scottish 5, Italian 4, Swiss 4, American 3, Russian 2.

3. **Age.** In several cases this particular is curiously omitted. The following is derived from 73 cases.

<table>
<thead>
<tr>
<th>1st decade</th>
<th>7 cases or 9.5%</th>
<th>Mortality 1 case or 14.2%</th>
</tr>
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<tbody>
<tr>
<td>2nd &quot;</td>
<td>11 &quot; or 15%</td>
<td>5 cases or 45.5%</td>
</tr>
<tr>
<td>3rd &quot;</td>
<td>13 &quot; or 17.8%</td>
<td>11 &quot; or 84.6%</td>
</tr>
<tr>
<td>4th &quot;</td>
<td>13 &quot; or 17.8%</td>
<td>9 &quot; or 69.3%</td>
</tr>
<tr>
<td>5th &quot;</td>
<td>13 &quot; or 17.8%</td>
<td>9 &quot; or 69.2%</td>
</tr>
<tr>
<td>6th &quot;</td>
<td>12 &quot; or 16.4%</td>
<td>8 &quot; or 66.6%</td>
</tr>
<tr>
<td>7th &quot;</td>
<td>4 &quot; or 5.4%</td>
<td>4 &quot; or 100%</td>
</tr>
<tr>
<td>Age 72</td>
<td>1 case or 1.3%</td>
<td>1 case or 100%</td>
</tr>
</tbody>
</table>

This table shews that Kopftetanus occurs most frequently in patients whose ages range from 21 to 50 inclusive, though the extremes are 2 years (Hadlich) and 72 years (Bourgeois). The table also bears out to some extent, what is usually affirmed with regard to ordinary tetanus, viz. that youth is favourable to/
to recovery. The mortality ranges from 14.2% of cases occurring in the first decade of life to 84.4% of cases occurring in the third. It is worthy of note, however, that in this third decade the mortality is markedly higher, not only than in the preceding decades, but also than in the three succeeding decades, which shew a mortality stationary at 69.2%. Thus it would appear that after 30, advancing years do not affect the prognosis unfavourably until the age of 60 has been reached.

4. Occupation. The occupations, which figure prominently in the histories, are, as one might expect, road-labourers, farm-labourers and grooms. Only three of the patients were of higher social status than working people.

5. Time of Year. Billroth in his "Lectures on Surg. Path. and Therapeutics" 1878. says "I have hitherto only seen an accumulation of cases of Tetanus with high close thunder temperature", and also "some observers came to the conclusion that a hot close temperature favoured the development of tetanus". In most text-books it is stated that tetanus is most common in the hot months of the year/
year. I find that 55.8% of the cases under consideration occurred from October to March inclusive, and that 44.2% occurred from April to September inclusive. During the four months October, November, December and January 41.5% of the cases occurred, compared with 28.5% during May, June, July and August, which may be taken as the warmest months of the year.

6. Method of Injury. In practically all the cases there is history of a definite wound, though in many cases only a trifling abrasion is mentioned. Of 78 cases -

35 cases, wound caused by fall, mostly on roads, 21 deaths
(6 of these were contaminated directly by dung, 3 "

18 cases wound caused by blow with stick or stone, 10 "

4 " " " splinter of wood, 2 deaths"

6 " " " kick of horse, 3 "

2 " " " firearms, 2 "

One case followed the bite of a horse, - recovered.

One case followed a peck from a peacock, - "

Nine miscellaneous cases, followed, respectively -

Ulcer on lip, blister on face, operation on nose, excision of a dermoid cyst, a burn, irritation of external meatus of ear, decayed tooth, a sore throat. The last three mentioned (cases of Behr), Zsigismondy/
Zigismondy and Euisson) are examples of the so-called "idiopathic tetanus", which, at the present day, is simply another way of stating, that the mode of entrance of the bacillus was not determinable with certainty.

It is interesting to note that the cases directly contaminated with pure horse dung do not shew a higher mortality nor a greater severity, than those whose wounds were not so obviously contaminated with material likely to harbour the tetanus bacillus in quantity. A remarkable case is that described by Schütze - A woman was attacked by a peacock, which pecked her on the forehead with such force, that the point of its beak broke off and was left embedded in the wound. This peacock had been feeding in a well manured garden, and on bacteriological examination of the portion of beak removed from the wound, there was found a mixed culture of tetanus bacilli and strepto-cocci.

7. Site of the Wound. The usual site for the wound is in the neighbourhood of the orbital margin. Table of 81 cases, giving site of wound.

<table>
<thead>
<tr>
<th>Site</th>
<th>Cases</th>
<th>Deaths</th>
<th>Mortality</th>
</tr>
</thead>
<tbody>
<tr>
<td>Forehead</td>
<td>22</td>
<td>15</td>
<td>53%</td>
</tr>
<tr>
<td>Nose</td>
<td>14</td>
<td>8</td>
<td>57%</td>
</tr>
<tr>
<td>Cheek</td>
<td></td>
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</tbody>
</table>
The wounds of forehead, though some were extensive, were mostly situated immediately above the orbital margin. Wounds of the cheek were generally in the malar region. This table seems to show that as a rule wounds in immediate vicinity of the orbit are associated with a higher mortality than those more remote. In one case, that of Larger, the wound was far removed from the head being situated on a finger, thus appearing to contradict one of the fundamental points in the definition of Kopftetanus. This case will be referred to more fully under another heading.

3. Incubation Period and Duration of Symptoms.

Taking 77 cases, where the necessary data are satisfactorily stated, I have made the following table -
In 32 non-fatal cases the duration of illness lasted for periods varying from 8 to 60 days, the average duration being 29 days. Of these cases, 19 had incubation periods of 8 days and under (5 cases having incubation periods of only 2 days), and 12 had incubation periods varying from 9 to 21 days.

The average incubation period in non-fatal cases is 8.8 days.

Of 45 fatal cases, 25 lasted less than one week, the shortest lasting only one day, 20 lasted for periods of over 9 days, the average duration including both groups was 7.4 days. 35 of these cases showed incubation periods of 8 days and under, 10 showed such periods varying from 9-20 days. The average incubation period for all the fatal cases is about 6.9 days.

From these facts it is evident, that the most striking difference between fatal and non-fatal cases is in the average duration of the symptoms, which in the former is 6.9 days and in the latter 29 days.

The difference between the average incubation periods is insignificant, fatal 6.9 days, non-fatal 8.8 days. Moreover, in no fewer than 5 non-fatal cases, there /
there were incubation periods of two days (Janin).
Holub, Reclus, Dumolard, Wartmann.)
Whereas only two fatal cases (Smart, Gillett) had such short periods. Hence as far as these cases of Kopftetanus shew, it must be concluded that a short incubation period does not necessarily mean a bad, nor a long period a good prognosis. It may also be stated that, with three exceptions (the cases of Thenee, Caird, Nerlich) any case in which the symptoms have lasted beyond 12 days, has recovered.
It is generally held with regard to tetanus, that a short incubation period denoteth an acute and unfavourable case, (Gibson's Textbook of Medicine etc.) this belief is not borne out by the facts just stated.

1. Trismus appeared in 32 cases
   16 deaths - mortality 50%
2. Trismus and Paralysis simultaneous in
   25 cases 16 deaths - mortality 61.5%
3. Paralysis of one or more cranial nerves in
   9 cases 7 deaths - mortality 77.7%
4. Contracture of Cheek in 7 cases
   4 deaths - mortality 50%
5. Stiffness of Neck muscles 4 cases
   1 death - mortality 25%
This table appears to show that early facial paralysis is a bad prognostic sign, as evidenced by the high mortality of paralysis - first cases. The cases are those of Hunt, Kirchner, Charvot, Wagner, Braennecke, Shütze, Caird, Smart and Hadlich.

If the last mentioned, which is a doubtful case, be omitted, then the mortality rises from 77.7% to 88.8%. Cases where paralysis appears simultaneously with the trismus have a higher mortality than those where trismus appears first. In one case, included in class 4, the initial symptom took the form of an uncontrollable impulse to laugh (Hale).

10. The Previous Histories. Speaking generally, there is an absence of facts in the histories of these cases, which would help to account for any form of paralysis. In nearly every case the patient has been quite healthy previous to the attack of Kopf tetanus.

A history of alcoholism is given in the cases of Dumolard, Hulka, Maryland, Caird and Larger. Epilepsy is noted in the cases of Lannois and Bouchard. Preceding erysipelas occurred in the cases of Nankiwell, Croly, Mothnagel. Janin Janin's first case suffered from phthisis, and Lannois' case from heart disease.

In one or two cases, such as Villars, the patients/
patients were exposed to cold and chill at the time of injury, but in the vast majority no such exposure occurred.

**The Symptoms:**

A general consideration of the symptoms shows what very important factors dyspnoea and dysphagia are in many of these 83 cases of Kopftetanus. I am not able to state in what percentage of ordinary cases of tetanus, one or other, or both, of these symptoms occur. But, as far as one can judge from the facts given about tetanus by the highest authorities, difficulty in respiration and especially difficulty with deglutition, must be considerably more frequent in cases of Kopftetanus.

The cases under consideration may be divided into four classes,

I. Those cases in which dyspnoea and dysphagia are both present.

II. Those in which suffocative phenomena are the chief feature.

III. Those in which dysphagic phenomena are prominent.

IV. Cases free from both of these symptoms.

<table>
<thead>
<tr>
<th>Class</th>
<th>Cases</th>
<th>Deaths</th>
<th>Mortality</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>26</td>
<td>24</td>
<td>92.3%</td>
</tr>
<tr>
<td>II</td>
<td>16</td>
<td>10</td>
<td>55.3%</td>
</tr>
<tr>
<td>III</td>
<td>19</td>
<td>6</td>
<td>31.5%</td>
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<td>IV</td>
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<td>2</td>
<td>13.1%</td>
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It is apparent that the prognosis is influenced very unfavourably by the presence of both, and likewise, though to a less extent, by the presence of one of these symptoms. As far as ordinary tetanus is concerned, a case where the tetanic phenomena are localised to the muscles of the jaw and the neck rarely shows these symptoms, whereas, in a number of the Kopftetanus cases, dyspnoea and dysphagia have been associated with comparatively mild and localised tetanic phenomena.

Respiratory difficulties may arise from glottic spasm. There is no evidence that paralysis of the abductors of the vocal cords has ever occurred. The spasm may occur spontaneously, or may be set up by any sensory stimulation, or may be specially associated with spasm of the pharyngeal muscles provoked by an attempt to swallow. The glottic spasm is of short duration ending in death, or in rapid relief, as the case may be. Tracheotomy overcomes the respiratory obstruction satisfactorily. Examples of cases suffering from glottic spasm are

(1) Specially associated with pharyngeal spasm, cases of Rose, Widenmann, Brunner (I, II, and III), Behr, Nerlich, Maylard and Plucker. To quote from the last mentioned writer: "There is difficulty in swallowing. A trial with fluid brings/
brings on severe attacks of spasm. During the night there were severe fits of dyspnoea, due to glottic spasms, so tracheotomy was performed with relief to the patient." This case died a few days later from asthenia.

(3) Unassociated with pharyngeal spasm. Cases of Nankiwell, Hulke, Crouzon and Charvet. The following is taken from the last mentioned case,—
"Death occurred with phenomena of laryngeal spasms and of asphyxia. The muscles of inspiration, including the diaphragm, make great efforts." No difficulty in swallowing is mentioned.

"Dyspnoea may be due to spasm of the thoracic muscles and of the diaphragm, occurring separately or simultaneously. The stoppage of respiration is less complete, and more prolonged than that due to glottic spasm. Spasm of the thoracic muscles gives rise to a feeling of constriction round the chest, and the chest is held rigid. Diaphragmatic spasm causes severe epigastric pain and leads to immobility of the abdominal wall as far as respiratory movements are concerned.

The chest tends to be held in the position of deep inspiration; there may be shallow rapid respirations while the spasm lasts.

(1) Associated/
(1) Associated with pharyngeal spasm. - cases of Behr, Güterbock, Lehrnbecher, Reclus and Bourgeois.

The following description is from the last mentioned case. "On attempt to swallow a little milk the greater part was rejected, as any attempt at deglutition provoked pharyngeal spasm, and augmentation of the trismus. A little later there arose attacks of dyspnoea during such spasms. The patient sat up in bed and the hands were carried to the mouth and throat. Respirations were frequent and shallow, with noisy expiration. There was foam at the lips. It was a spasm of the muscles of inspiration rather than of the glottis. Death occurred from Asthenia."

(2) Unassociated with pharyngeal spasm, as in the cases of Braennecke and Schrotter. Neuman in his description of Schrotter's case says - "During the spasm, respiration is difficult and rapid, there are attacks of coughing. Patient asks for water and takes sips. In two or three minutes the muscles (thoracic) relax gradually and the patient though exhausted is quiet."

The great majority of cases bear so trifling a resemblance to true hydrophobia, that an alternative name "Tetanus Hydrophobicus" suggested by Rose, is not/
not applicable. The following cases bear the closest resemblance to hydrophobia, as in them the reflex excitability of the pharyngeal muscles is very marked.

1. Case of Reclus. Patient shewed great apprehension of the attacks of spasm, which were caused by the attempt to swallow liquid. "The sight of a glass of water provoked a veritable attack of hydrophobia."

2. Case of Kirchoff. Difficulty in swallowing and breathing appeared early. There was marked salivation. At the sight of water patient resisted with all her might. General spasms did not always accompany the respiratory and deglutition spasms.

3. Case of Von Wahl. Several days after the onset of the symptoms patient had a suffocative attack, for which tracheotomy was performed. Violent spasm of the throat was produced at the sight of water.

4. Bouchard. "Patient is not even able to swallow liquids, if liquid be put into the buccal cavity, it is rejected by the suffocating patient."

Dysphagia. This term is applied by writers on Kopftetanus to all grades of difficulty in swallowing, from slight trouble in taking solid food to absolute inability to swallow anything. Dysphagia may be unaccompanied/
unaccompanied by any respiratory difficulty, and the difficulty may be limited to the swallowing of solid food as in the case of Janin, which however shewed this peculiarity, that, whereas most fluids could be swallowed, wine and chloral tended to provoke pharyngeal spasm.

In a number of cases even fluids cannot be swallowed e.g. Cases of Thanée and Huntingdon. The difficulty is generally due to a spasm on the pharyngeal muscles; but in a few cases, spasm of the muscles attached to the Hyoid and Thyroid cartilages is described as leading to fixation of the larynx, and to interference with the mechanism of swallowing. Schütze describes a case in which every attempt to swallow, brought on a spasm of all the muscles of the throat, the head being retracted; there was no dyspnoea. A similar case is described by Middledorp.

The cases associated with dyspnoea are the more numerous group. The two symptoms, dysphagia and dyspnoea, as regards their respective severity, do not bear any direct ratio to each other. A probable cause of their frequent association is, that difficulty in swallowing combined with profuse salivation lead to saliva getting into the larynx, and setting up a cough, which stimulus may excite spasm.
spasm in the muscles of the respiratory mechanism. An attack of coughing is frequently noted as a prelude to respiratory difficulty.

**Trismus.** This is the most constant, and usually the earliest symptom. In some cases of Kopftetanus it tends to affect the muscles of mastication more strongly on one side than on the other, rarely it is strictly unilateral. I have not been able to find a reference to unilateral trismus in accounts of ordinary tetanus cases.

Out of 77 cases of Kopftetanus: -
63 cases shewed bilateral trismus, Mortality 57.1%
14 cases shewed unilateral trismus, Mortality 42.8%

In only three of these unilateral cases is the contracture confined to the masseters of one side, in the majority the contracture is simply much more pronounced on one side than the other. The jaw muscles chiefly affected are invariably those on the side corresponding to the entrance of infection. Unilateral affection occurs in the less severe cases as a rule.

Localisation of the tetanic phenomena to the head and neck is generally held to affect the prognosis in tetanus favourably. Regarding Kopftetanus all one/
all one can say is, that the prognosis is good in the few cases, where there is only localised tonic contracture of the muscles, and where true spasms ("redoublements convulsives") are absent altogether.

The following table is founded on 76 cases -
1. Localised Contracture, 13 cases, 11 recoveries,
   mortality 15.3%
2. Localised Cont. and localised spasm, 24 cases,
   9 recoveries, mortality 62.5%
3. Generalised tetanus, 39 cases, 14 recoveries,
   mortality 64.1%

It is evident that localised cases are almost equal in number to the generalised ones, and that the mortality of the ordinary localised cases (class 2) is practically equal to that of cases with generalised spasms. This latter fact is in contrast to what is usually stated about the relative mortality of localised and generalised tetanus.

The statement is commonly made that the early appearance of tetanic spasms indicates a severe case of tetanus and that accordingly the prognosis is grave. As regards the cases of Kopftetanus, no definite conclusion can be drawn. Spasms are described as occurring very early in some of the mildest
37.

mildest cases e.g. in those of Janin, Carettil and Humphry, which all ended in recovery. Spasms have appeared late in some of the worst cases e.g. in those of Maylard, Theene, and Villar, which all died.

Paralysis. This is the most interesting, and the distinctive feature of Kopftetanus. It is generally referred to in definitions and so forth as facial paralysis; but it must be understood that the seventh nerve is not the only one affected; as in some of the more recent cases, paralysis of the third and of the twelfth nerves has been noted.

There is no record of paralysis occurring in a case of tetanus following a wound outside the region of the cranial nerves, except perhaps in the case of Larger, described later. There is no evidence of paralysis having occurred outside the area of the cranial nerves in any case of tetanus. In stating this I exclude cases described by Larrey, Helstig (quoted by Brunner), and Hunt, where hemiplegia, probably due to a gross cerebral lesion, was associated with tetanus.

1. Paralytic phenomena connected with the seventh nerve. Taking 81 cases into consideration-

1. In 68 cases the facial paralysis was unilateral, on the same side as the wound.

II. In 5 cases (those of Terrillon, Wagner, Pollock)
9. Pollock, Schmidt and Kirchner) the paralysis was unilateral, but on the opposite side to the wound.

III. In 8 cases (Huntingdon, Théneé, Roberta, Caretti, Crouzon, Jamin, Bourgeois and Bouchard) the paralysis was bilateral.

In 68 cases, where paralysis occurred on the same side as the wound, 44 showed complete paralysis; of these 24 were fatal. There were 13 partial cases, only one set of branches of the facial nerve being affected, with 5 deaths. In 10 cases there was merely a paresis, in some cases affecting all the distribution of seventh nerve, in others limited to a few branches of the nerve. Out of the latter ten cases, 5 were fatal. It cannot be said that an incomplete paralysis is distinctively associated with a mild case of Kopftetanus.

In 5 cases, shewing paralysis on the opposite side to the wound, there is nothing specially distinctive as regards site of wound, or type of case. Paralysis was partial in 3 cases (Terillon, Wagner, Schmidt, and Schmidtmann), complete in the case of Pollock, and only amounted to a rather doubtful paresis in that of Kirchner.
In 6 bilateral cases, the wound has been as a rule small and situated about the mid-line of the nose, about the level of the nasal bones. In the cases of Caretti and Janin, however, the wounds were fairly extensive, reaching from about the root of the nose across the forehead in each case. Robert's case had a small wound on the left lower eyelid. The tendency for the wound to be about the mid-line of the face in these cases is certainly a fact of interest.

Of the 8 cases five were fatal, which is a high mortality. It is remarkable that these fatal cases all suffered from distinctly localised and comparatively mild tetanic phenomena, but all had difficulties of deglutition or respiration. (They are the cases of Roberts, Théée, Bourgeois, Bouchard, and Crouzon).

The paralysis was complete and the onset on each side simultaneous in four cases (those of Huntingdon, Caretti, Crouzon and Bourgeois). In Bouchard's case the onset was simultaneous but the paralysis affected the upper facial muscles chiefly, the orbicularis oris being practically unaffected. Théée's case showed complete paralysis of both facial/
facial nerves, but the left nerve was affected two days before the right one. Robert's case on the sixth day had complete paralysis of the facial nerve on the same side as the wound, followed a day later by a partial paralysis of the opposite facial nerve. It is doubtful if Janin's case should be included amongst these bilateral-paralysis cases. Apparently, an almost complete right facial paralysis lasted about a month, by the end of which time it had disappeared. At the same time a partial paralysis of the left facial nerve appeared.

The cases of Bouchard and Roberts were complicated by a degree of paralysis of the third cranial nerve, and reference will be made to them again.

There are two cases (Hale, Smart,) where paralysis of the facial nerve on one side is accompanied by partial paralysis of the oculo motor nerve of the opposite side, thus giving a variety of bilateral paralysis, which will be considered later.

Localised paralysis or Paralysis of the seventh nerve is found in 20 cases, and though the records of many of the cases are meagre in detail, the following/
following general facts may be stated. In twelve cases (Zigigmondy, Terillon, Middledorf, Janin, Dumolard, Widenmann, Sériens, Klemm, Nerlich, Crossouard, Schütze, and Bouchard) the paralysis affected a portion of the facial nerve area situated nearest the wound. The upper branches of the facial nerve were paralysed in 8 of these cases, in some the frontalis or orbicularis palpebrarum being alone affected, in others the orbicularis oris alone escaping. In 4 cases the lower branches of the facial nerve were affected; two of these (Crossouard and Widenmann) only shewed a paralysis of the muscles about the angle of the mouth. The cases of Janin, and Bouchard were bilateral. Seven cases (Wagner, Solmsen, Hadlich, Caird II. Larger, Holub, Nothnagel) shew paralysis affecting portions of the facial nerve not lying in closest proximity to the wound. For instance in Wagner's case - Wound on right forehead, paralysis affected middle and lower branches of left facial nerve - ; in Caird's second case - Wound on left frontal eminence paralysis affected lower portion of left facial nerve. Solmsen's case is peculiar, as the patient had a wound of the right occipital region and a complete right facial paralysis with the exception of the orbicularis palpebrarum which/
which was unaffected.

In one case, that of Roberts, there was a wound on the left lower eyelid and complete left facial paralysis, also paralysis of the upper portion of right facial nerve, and the third and fourth nerves on this side were affected.

If a reason for the partial paralysis be looked for, and one contrasts these 20 cases with those suffering from complete paralysis, no special peculiarity of the mode of injury, nor the size, nor site of the wound is noted.

Regarding other particulars of the 20 cases, 9 were fatal, mortality 45%; 16 had localised and 4 had generalised spasms; 7 suffered from both dysphagia and dyspnoea, 5 from the former, 3 from the latter, 5 from neither. It may be noted that of the 9 fatal cases 6 suffered from dysphagia and dyspnoea. Although the proportion of cases with localised tetanic phenomena is very high, yet the general mortality 45% is not markedly lower than that of all the 83 cases of Kopftetanus, which is 55%.

From these facts one is hardly warranted in saying that partial facial paralysis is definitely associated with a mild case of Kopftetanus.

Facial Spasm. Before discussing the symptoms of paralysis of other cranial nerves, /
nerves, I intend at this point to refer to another symptom chiefly concerning the seventh nerves — viz facial spasm. This phenomenon is, of course, quite frequently present in ordinary tetanus cases, giving rise to the classic "risus Sardonicus." It has however, a special interest in Kopftetanus since it complicates the symptoms of facial paralysis, and may lead to difficulties and uncertainties in the diagnosis.

In six of the cases with complete bilateral paralysis, and in about 16 of the unilateral cases (e.g. Rose, Willard, Janin, Trevelyam, Plücker, Massurianz, Wagner, Brunner etc.) no facial contracture nor spasm is recorded.

In 28 cases of complete unilateral paralysis, on same side as wound, there is noted, facial contracture or facial spasm on the unparalysed side: with one or two astonishing exceptions, where contracture is said to occur on the same side as the paralysis. It is worthy of remark, that it is possible to have a strictly unilateral tonic contracture of the facial muscles, with the appearance of occasional spasms. Such a condition is apt to suggest the presence of paralysis or paresis of the opposing facial muscles e.g. case of Terrier (included in non-paralytic group of cases). Terrier writes/
writes - "a month after the accident (wound on right side of scalp) there was tonic contraction of the right facial muscles; the right half of the forehead was wrinkled and more furrowed than the left half. The right eye was closed by contraction of the orbicularis and the right angle of the mouth was drawn to the right etc." The comparatively smooth left face, together with deviation of the mouth to the right, might easily suggest at least a partial left facial paralysis. A similar case is that of Gosselin, who notes the simulation of a facial hemiplegia by unilateral contracture. In all cases of Kopftetanus deviation of the mouth us an unsatisfactory sign of facial paralysis, unless supplemented by undoubtedly abnormal flaccidity of the cheek and lips on the side of the supposed paralysis. In cases where there is inability to close the eye and to wrinkle the forehead no doubt as to the paralysis can exist.

Well marked facial paralysis may be present on one side several days before contracture and spasm affect the muscles of the other side of the face, as in Villar's case. More usually the appearance is simultaneous or both are present when the case is first observed.

Where/
Where the facial paralysis is complete, the facial contracture gives rise to no difficulty; but an incomplete form, especially if limited to the lower facial muscles, may be difficult to establish satisfactorily. Take, for example, the case of Larger, from which I take the following:

"et ce qui frappe de suite, avec asymétrie faciale. En effet, le rire sardonique, est très accentué à gauche, avec relevement de la commissure labiale, exagération des rides de la patte d'oie, œil demi clos, par la contracture de l'orbicularis. A droite, au contraire, flaccidité complète, effacement du sillon naso-jugal, commissure labiale abaissée, fente palpébrale normale. Les mouvements des yeux restent normaux de part et d'autre."

It is evident that the author assumes a paralysis affecting the lower branches of the right facial nerve. I refer to this case particularly, because if there was any degree of real paralysis or even paresis present, it becomes one of singular interest, as the mode of infection was a splinter under the nail on one of the fingers. It is unfortunate from our present point of view that, in such a very exceptional case, the paralysis did not affect the orbicularis palpebrarum or the frontalis on the left/
left side, which are in no way influenced by spasm of the opposite facial muscles.

**Simultaneous Contracture and Paralysis.**

In the consideration of a few cases one finds the description of contracture affecting the paralysed muscles, or at least occurring on the paralysed side of the face.

The French Surgeon Reclus (Cliniques Chirurgicales, Paris 1894 page 150) after discussing the five instances of this phenomenon which had till then been described, finally says that he cannot understand the paradox of the contracture of paralysed muscles, and most writers take up the same position. I now give short extracts from some of the cases.

Güterbock describes a complete right facial paralysis and adds "on right side of face, in spite of the paralysis there is stiffness of the muscles of the cheek as well as of the jaw."

Terillon says "The face is asymmetric, the left side having drawn over the right side, the left eye is narrowed the angle of mouth is depressed and drawn to the left. In a word one found the ordinary signs of right facial paralysis. Nevertheless, in looking at it more closely, I assured myself/
myself that the right side of the face was not as flaccid as in ordinary paralysis; the cheeks could be blown out, without much loss of air at the right angle of the mouth."

Bernhardt describes a complete left facial paralysis, and then adds that the left side of the face in spite of this had a "contracted appearance."

Duvergey. La visage est grinçant, il semble figé dans un sourire bizarre qui ne varie pas et qui nous rappelle de suite le "rire sardonique" des tétaniques. Tous les muscles de la face sont dans un état de contracture permanente. Il existe cependant de la parésie très accentuée du côté droit de la face." He then describes how the right half of the face remains inert, while the left half behaves normally, when various tests, such as asking patient to whistle etc, are applied. The right eye cannot be completely closed. He adds "Tous les muscles de la face sont contractures, on sent surtout à gauche, mais chose curieuse, aussi à droite, le plan resistant du muscle contracture."

These cases seem to suggest the co-existence of paralysis and contracture of the facial muscles, though the descriptions are not sufficiently detailed to be convincing.
Before proceeding to contrast with the above cases some others not unlike them, I would emphasise the fact, that it is possible to have a localised facial spasm just as there may be localised facial paralysis. The following are examples of this localised facial contracture.

Maylard, Left facial paralysis; Contracture of muscles at right angle of the mouth.

Nerlich Left facial paralysis; Contracture of muscles supplied by lower branches of the right facial.

Lannois Left facial paralysis; Contracture of right orbicularis palpebrarum.

Kirchner Left facial paralysis; Spasms affecting right eyelid.

The following group of cases may be held to exhibit a localised paralysis or paresis of certain facial muscles, together with a certain amount of contracture or spasm of others. Some of them have been described as examples of contracture present in paralysed muscles; compare the four cases already mentioned. I shall give a few extracts.

Sériens. Right half or face shews normal physiognomy, normal furrows of left half of face exaggerated except on the left half of forehead, which/
which is smooth and contrasts with the right half by reason of the effacement of its transverse furrows. The left eye is closed, the result of a paralytic ptosis.

Solmsen. The right half of face looks distinctly slacker than the left and is not affected by reflex movements. The right angle of the mouth is depressed, the right palpebral fissure is smaller than the left, which must be ascribed to spasm of the orbicularis palpebrarum. All the muscles supplied by right facial nerve are paretic except the orbicularis palpebrarum, which is in a condition of tonic excitement.

Kadlich found a right facial paralysis, but when the child cried, the right palpebral fissure became smaller than the left, due to spasm of the orbicularis. He mentions that there was also a tendency for the right half of orbicularis oris to be affected by spasm.

The cases of Dumolard, Von Wahl and Brunner appear to shew a paralysis or paresis of the facial muscles on the same side as the wound, with the exception of the corresponding half of the orbicularis oris, which is contractured or affected by spasms.

Neuman/
Neuman in describing Professor Schrotter's case says, 'Patient lies quietly in bed, when, with a sudden scream, he desires to sit up. The right muscles of expression are contracted, the forehead being furrowed, the eye closed and the nose drawn to the right. The muscles at the right angle of the mouth stand out prominently. The muscles of expression on the left side are not attacked by spasms, the forehead is smooth, the eye wide open, the angle of mouth smooth: but there seems to be an increase of muscular tone, the lips being firmly pressed together, the mouth is drawn a little to the right side.' Further he says, 'the paralysed muscles were free from spasm (i.e. the orbicularis palpebrarum and the frontalis), but in the paretic muscles (the left lower facial muscles), during the general attacks of spasm, if there was not a spasm, then surely there was an increase of muscular tone.'

From the fact that a localised paralysis of certain branches of the facial nerve can occur, and that similarly a localised contracture may be present, it does not seem to me impossible that these two phenomena can occur together, affecting muscles supplied by different branches of the facial nerve. This is certainly much more likely, than the occurrence of contracture.
contracture of paralysed muscles, and certainly seems to have been the case in these last mentioned cases.

In one case, that of Wartmann, the immobility of the facial muscles is ascribed to tonic contracture, but the possibility of paralysis is also assumed. "The face had a stiff expression, muscles of expression moved less than normally. Next day patient could scarcely move the lips; the muscles of the cheek felt hard, both halves of the face had the same appearance. The eyelids on both sides could be closed."

There are a few general facts about the affection of the facial nerve, yet to be mentioned. In at least four cases (Böhr, Roberts, Dumolard, Pollock) contracture or spasm of the facial muscles has been succeeded by paralysis. In two cases the paralysis was complete, but in Robert's case it only affected the upper branches of the facial nerve, and in Dumolard's case all the branches save those supplying the orbicularis oris. This latter case has already been cited as an example of the co-existence of facial paralysis and facial contracture/
contracture on the same side of the face.

There is no evidence that facial paralysis has ever been succeeded by facial spasm of the same side. In Smart's case facial paralysis was preceded by twitching of the muscles affected.

In the cases of Zigismondy, Massurianz, Brunner and Duvergey there was preceding pain in the distribution of the facial nerve. In the last mentioned case, the course of the temporo-facial and cervico-facial branches of the facial nerve could be traced out as special lines of tenderness on pressure.

Involvement of the sense of taste, such as is associated with involvement of the trunk of nerve VII. between the genu and the origin of the Chorda Tympani (involv. of nervus Intermedius of Wrisburg), is never mentioned. Increased sensitiveness to musical notes, indicating paralysis of the Stapedius muscle, has not been noted.

With regard to the time relative to other symptoms, at which paralysis (including paralysis of any cranial nerve) appears, see table of initial symptoms. The earliest time of appearance after the wound is 2 days (Smart), the latest 28 days (Oliva); the average time is 10.4 days.

In most cases the facial paralysis passes off contemporaneously with the trismus, but in a/
but in a few cases, as in \textit{Middledorpf}'s, it disappears first, whilst in some it persists after the trismus has disappeared.

The average duration of the paralysis was 23.3 days in those cases which were not fatal. The paralysis lasted only 11 days in \textit{Crossouard}'s case and 15 days in that of \textit{Middledorpf}, whereas in the cases of \textit{Dumolard} and \textit{Janin}, the duration of the paralysis was about 65 days.

Persistence of the paralysis, and that only in a slight degree, is recorded in four cases, those of \textit{Reclus III.}, \textit{Buisson}, \textit{Schultze} and \textit{Rockcliffe}(in the last mentioned only a slight ptosis persisted).

Branches of the \textit{Oculo-motor Nerve} are affected with paralysis next in frequency to those of the \textit{Facial}. The most frequent manifestation of this is ptosis in varying degree.

A bilateral ptosis, most marked on the side opposite the wound, unaccompanied by facial paralysis, is described by \textit{Rockcliffe}. There was a wound caused by a thorn in the left orbit. A diplopia on convergence for near objects is also noted in this case.

\textit{Zigismondy}/
Zigismondy and Seriens describe unilateral ptosis, with partial facial paralysis on the same side, in their respective cases; the former infected from the mouth, the latter from a large scalp wound.

In the cases of Smart and Hale ptosis occurred on the opposite side to the facial paralysis, a form of bilateral paralysis.

Robert's case, previously referred to, is a complicated one; the following is a brief extract from the case:

July 2, "Left eye is fixed, the pupil dilated, no reaction to light. Pupil of right eye small. Right facial muscles in tonic contraction, left facial muscles completely paralysed etc.

July 3, Ptosis of right eyelid, the right eye can only be moved outwards. Paralysis of 3rd and 4th nerves is thus indicated. The upper portion of right facial nerve is paralysed." (The wound was on the left lower eyelid). There are thus six instances of ptosis, one bilateral, five unilateral; of the latter, two shewed facial paralysis on the same side, two shewed it on the opposite side, and one had bilateral facial paralysis with ptosis on the side remote from the wound.

One/
One or other of the ocular muscles were affected in several cases. Caird mentions bilateral internal strabismus accompanying left facial paresis. Von Wahl describes external strabismus of right eye appearing the day before death. Bouchard mentions in his case that the movements of the eyes upwards and downwards were limited.

In Roberts' case the right eye could only be moved outwards; Smart's case showed internal strabismus of the right eye, accompanying right ptosis and left facial paralysis.

Roberts ascribes the condition in his case to paralysis of nerves III. and IV., while in Smart's case the condition was probably due to spasm, as the movements of the eyes were practically concomitant.

In the latter case during a severe spasm a temporary rotation of the left eye upwards and outwards was noted, probably due to spasm of the superior, and of the external rectus.

Mydriasis. In two cases the pupil is noted as permanently dilated on the same side as the facial paralysis (Gillett and Humphry), probably due to paralysis of ciliary branches of the third nerve.
Paralysis of the fourth cranial nerve has never been observed, nor can paralysis of the sixth nerve be definitely shewn to have existed.

Paralysis of the Hypoglossal Nerve. The twelfth nerve has been described as paralysed by Neuman and by Holub. It is of course only in a case with mild trismus, or where trismus is beginning to disappear that even a hint can be had of this symptom. In Dr. Smart's case the tongue could never be protruded, but there was no doubt that it was freely mobile behind the clenched teeth. But even with one Hypoglossal paralysed or paresed, the tongue would still seem to be fairly mobile within the closed buccal cavity.

I now give short extracts from the two cases - Professor Schrotter's Case described by Neuman.

October 28th "Wound under the left orbital ridge; some days later trismus appeared, also a left facial paralysis.

September 22nd Jaws could be separated a little, and point of tongue could be protruded a little, apparently deviating to the left.

December 2nd. Trismus less marked, left facial paralysis much better but still present. The deviation of tongue, when protruded, is now more distinct.
distinct. The tongue curves from left to right and over to left side again, the tip being well to the left of the mid-line.

December 7. Hypoglossal paralysis diminished.

December 21. Facial paralysis has disappeared, but tongue still deviates a little to the left."

Neuman mentions two cases, where deviation of tongue was ascribed to the presence of facial paralysis, viz. that of Schultze, where, associated with a left facial paralysis, deviation of the tongue to the right was noted, when the trismus began to get better. Schultze thought the deviation was only apparent and was accounted for by the facial paralysis. Also a case mentioned by Rose, where there was no apparent facial paralysis, but where the tongue deviated to the right. Rose thought the deviation to be due to paralysis of the stylo-mastoid branch of the facial nerve; the tongue deviating to the healthy side in consequence of an altered position of the hyoid bone brought about by inaction of the digastric and Stylo-hyoid muscles of one side. This is an ingenious, though far-fetched explanation. The possibility of such a localised paralysis of the seventh nerve may be admitted, yet it is open to doubt, if it would cause a deviation of the tongue.

Holub/
Holub describes the case of a boy wounded with a stone on the left temple. Trismus and partial paralysis of the left face followed, no deviation of the tongue could be seen at first. On the fifteenth day after the wound facial paralysis had almost disappeared, and the tongue could be protruded, shewing deviation to the left. On the twentieth day the jaws could be opened, and the protruded tongue deviated distinctly to the left. On the thirty-eighth day, all other symptoms having disappeared, the tip of tongue still deviated slightly to the left side.

Both Neuman and Holub appear certain that their cases presented paralysis or paresis of the hypoglossal nerve, on the same side as the lesion and the facial paralysis.

If Schultze's case was an example of hypoglossal paralysis, then the nerve on the opposite side to the wound and the facial paralysis was affected.

In Wartmann's case the following is found, "September 20. The boy cannot put out his tongue; September 21. Spasm of masseter's and facial muscles occurred". Bouchard - " June 19. Patient appears to move tongue with/
with difficulty".

This inability or difficulty in moving the tongue was ascribed to a degree of spasm of the muscles of the tongue. That such spasm may occur is most clearly shown in one of the cases included in the non-paralytic group, that of Mitchell. Here the tongue is described as being "as hard as wood". Thus it is evident that the possible presence of a partial contracture or spasm of the tongue muscles must be taken into account when deviation of the tongue is met with. In such cases it is probable that the patient would complain of a feeling of stiffness of the tongue, and the contracted muscles would give a feeling of hardness, on palpation, to one or other side of the tongue.

As regards the soft palate and uvula, as might be expected, few observations are recorded.

Behr notes in his patient, who had a right facial paralysis, that the soft palate moved more distinctly on the right side than on the left. Speech was indistinct with a nasal twang.

Schultze found deviation of the uvula to the right (left facial paralysis).

According to Horsley and Beevor the soft palate and/
and uvula are innervated by the accessory nerve to the vagus. Whether the phenomena in the above cases were due to a paralysis of this nerve or due to localised contracture of certain muscles, cannot well be determined.

Deviation of the uvula is also noted in one of the unparalysed group of cases. (Nicolaysen).

In Brunner's third case it is noted that the uvula was perpendicular.

The Cervical Sympathetic. There is evidence that this has been affected by paralysis, at least as regards the pupil dilating fibres. 42

In Brunner's fourth case very marked perspiration of the right cheek and right half of the forehead was associated with right facial paralysis. This was associated with narrow pupils.

In Brunner's third case the left side of the face was paralysed; There was marked perspiration from the forehead, but none from the rest of that half of the face. The pupils were narrow.

In Charvot's case the pupils were contracted and equal and did not dilate. (Right facial paralysis).

Lannois mentions double insensitive pin-point pupils. (Left facial paralysis).

It may be said that the narrow pupils were due to a/
to a spasmodic condition of the sphincter pupillae; this may have been so, but in Brunner's two cases there was concomitant vaso-motor and secretory disturbance pointing to an affection of the cervical sympathetic. In none of these cases was there evidence of the third nerve being affected.

I have not been able to find any reference to a satisfactory instance of paralysis affecting motor nerves, other than the cranial nerves, in any class of tetanus case. In the case of Hunt quoted by Brunner, Willard, and others as a case of Kopftetanus, there was a complete right hemiplegia, which supervened two or three days after severe injuries to the left frontal and parietal bones. Tetanic symptoms appeared the day after the hemiplegia. Laceration of the left side of brain and an abscess in the right anterior lobe were noted post mortem. The case of Holsti (Virchow) is somewhat similar. It is probable that gross cerebral injury was the cause of the hemiplegia in both cases.

**Sensory Phenomena.**

The fifth nerve is the seat of sensory disturbance in ten cases. The cases of Villar, Nerlich, Klemm, Braennecke, Charvot, and Triglia shew/
shew a varying degree of hyperaesthesia.

In these cases the wounds are all well within the area of distribution of the fifth nerve; the hyperaesthesia is always on the same side as the paralysis, and may be local or general over the one side of the face. The degree of severity varies. I give particulars of two cases as examples.

Villar - Wound left orbit and left fronto-parietal region, left facial paralysis, intense hyperaesthesia over root of nose, orbital margin and forehead on the left side.

Nerlich - Wound bridge of nose, left facial paralysis, whole of left face hyperaesthesia.

The cases of Von Wahl, Lehrnbecher, Buisson, and Bernhardt shew some degree of anaesthesia. For example -

Bernhardt - Wound left orbital region, left facial paralysis, numbness of chin and lower lip.

Buisson - No apparent wound, left facial paralysis, degree of anaesthesia of left half of face.

The time of appearance of these phenomena varies from very early to quite late; no information is given as to their precise duration.
On the whole, there seems little tendency for the sensory branches of the fifth nerve to be affected.

The eighth nerve appears never to have been affected in cases of Kopftetanus. Deafness occurred in the cases of Buisson and Behr but in both appears to have been due to middle ear disease.

Other Symptoms.

The remaining symptoms and physical signs may be rapidly reviewed, as they are in no wise peculiar to Kopftetanus.

**Alimentary System.** There was no digestive disturbance, where nourishment could be swallowed. In many cases excessive salivation was marked, which could not be accounted for solely by facial paralysis or by pharyngeal spasm. (Braennecke, Bouchard, Hadlich, Neuman etc.)

In some cases there was no such increase e.g. cases of Holub and Janin.

Constipation is noted in the majority of cases; incontinence of faeces has occurred in a few e.g. Bourgeois.

**Circulatory System.**

Tetanic spasms have a marked effect on the pulse rate, for example in Lehrmecher's case it varied from 50 beats per minute between the attacks of
of spasm, to 120 beats during spasms. In a few cases it is noted that during spasms the pulse becomes fuller and its tension increased.

The strain on the heart muscles is shewn in Smart's case and in Villar's, in both of which recent hypertrophy of the left ventricle was noted post mortem.

Clinically the effect of the strain is shown in such cases as that of Maylard, where the pulse became small and finally uncountable after tetanic spasms; that of Braennecke, where 5 minutes after crisis pulse could not be felt; that of Widenmann, where before death pulse-rate went up to 180 beats per minute. These three examples all died from gradual heart failure, and this is the commonest mode of ending of cases of Kopftetanus (53% of fatal cases).

Contrast the above details with the following cases fatal from asphyxia - Hulke, "day before death pulse 64 regular and hard"; also the case of Dr. Smart where the heart went on beating quite regularly and vigorously for about half a minute after respiration had entirely ceased.

Respiratory System. I have nothing to add to what has already been stated with regard to dyspnoea.

One case died of pulmonary oedema, and two of pneumonia.
pneumonia.

Nervous System. Under this general heading I shall only mention one or two facts. In two or three cases there has been a degree of delirium; in one case (Kirchoff) there were hallucinations followed by unconsciousness; in one case (Bohm) coma preceded death.

As a general rule the mind has been clear from beginning to end of fatal cases.

The patellar reflex has been noted as increased by Bond, Klemm, Brunner (3rd case), Halub, and Neuman, as almost absent by Brunner (4th case); as unchanged by Crouzon, Bourgeois and others.

In cases where muscular contracture affected the lower extremities no response could be expected. The plantar skin reflex was found increased by Bond, and Solmsen.

Urinary System. Incontinence of urine during tetanic spasms observed in the cases of Willard, Bourgeois and Smart. Retention of urine, due to spasm of the compressor urethrae, occurred in the cases of Widenmann, and Brunner (second and third).

Small quantities of albumin were present in 5 cases/
cases. Braennecke notes the presence of sugar, Kirchoff the presence of a reducing substance, in the urine.

Temperature. This has been a favourite subject of controversy amongst writers on tetanus and Kopftetanus. From a survey of the cases under consideration, it cannot be concluded that the prognosis is affected one way or the other, by the presence or absence of a febrile temperature. The great majority of cases show an irregular mild febrile temperature. In such cases as those of Mayer, Dumolard, Sériens, Théneé, Solmsen and Duvergey, representing all varieties of Kopftetanus, the temperature is definitely given as normal. Caird mentions a subnormal temperature in his first case.

In two cases Brunner notes post mortem rise of temperature; in one case from 38° to 39.8° (cent) two hours after death, in the other from 40.2° to 41° (cent) half an hour p.m.

Electrical Reactions. In 13 cases the electrical reactions of the facial nerve and the muscles supplied by it have been more or less incompletely examined.

Reaction of degeneration has never been found. Loss of reaction to the faradic current is only noted in/
in Reclus' third case, where it was only temporary, as a week later the reaction was normal.

Nerlich, Kirchoff, Oliva and Brunner noted increased irritability to the faradic current on the paralysed side of the face, in the latter three cases only relative to a general increased irritability.

Oliva and Solmsen note decrease of faradic irritability on the paralysed side of the face.

Kirchoff says "The muscles of the left face (paralysed side) are excited to contraction by the faradic current, more strongly if it be applied over the terminal branches of the plexus anserinum, than when applied over the trunk of the facial nerve at the stylomastoid foramen, - a proof of the peripheral origin of the paralysis."

**Prognosis.** Reference to this has frequently been made in the foregoing discussion of the various symptoms. I give here a summary of the chief points concerning the prognosis.

1. Below the age of 20 years there seems to be greater chance of recovery.

2. The size of wound, and amount of gross contamination e.g. with horse dung etc., have little influence/
influence on the severity of the toxaemia.

3. Cases with wounds in the vicinity of the orbit have proved more fatal than those, where the wound has been remote from the orbit.

4. A short incubation period does not necessarily mean a bad case.

5. If the patient survives the onset of the symptoms for twelve days, the hopes of recovery are good.

6. The early appearance of facial paralysis is associated usually with a severe case.

7. The presence of difficulties in swallowing, and the occurrence of attacks of dyspnoea, affect the prognosis unfavourably.

8. Localisation of tetanic spasms to the muscles of the jaw and neck is not specially favourable.

9. Early or late appearance of the tetanic spasms has no special significance.

9. While double facial paralysis has been associated chiefly with severe and fatal cases, a partial paralysis, or mere paresis, is not definitely associated with a mild case.

11. The paralysis almost invariably disappears in cases which recover.

Mode of Termination.
Mode of Termination. Considering all the fatal cases of Kopftetanus, I find that 31% suffered from some degree of dyspnoea, whereas only 30% actually died of suffocation.

About 53% died of gradual heart failure; 10% had lung or cerebral complications. As regards the rest detail is lacking.

Mortality. This has already been discussed in relation to various symptoms. The general mortality of 83 cases is 55.4%, compared with a mortality of 80% in ordinary cases of tetanus (Ostler).

Post-Mortem Examinations. These have been performed in 18 cases, with practically uniformly negative results, which do not merit detailed description here, as they differ in no respect from those obtained in fatal cases of ordinary tetanus.

Microscopical examination of the facial nerve has been negative.

With regard to possible nuclear changes in the pons and medulla, the cases of Nerlich and Bourgeois will be specially referred to later.

Bacteriology.
Bacteriology. In eleven cases reference is made to bacteriological examination. The results of this were negative in all respects in the cases of Smart, Plucker, Bernhardt, Larger, Villar, Behr, and Solmsen.

Schütze obtained cultures of tetanus bacilli from portion of pea-cock's beak embedded in the wound, and from the same source inoculated mice, producing tetanic phenomena. Brunner in his third case, did not demonstrate the actual presence of the bacillus, but inoculation of the blood serum, into mice caused tetanic spasms. Schmidt also found that 2 c.m. of blood serum caused fatal symptoms of tetanus in a mouse.

Brunner found that the saliva of his fourth case when injected into a mouse caused fatal tetanus. Injections of blood serum were negative, and no bacilli were found.

Brunner and Solmsen both tested the toxicity of the urine, by injecting guinea-pigs with a few cubic cent. from their respective cases. The result was negative.

Various continental observers have stated that
the urine of tetanus patients contains toxine; for example, Bruschettini (Rif. Med. April 11, 1892.) produced fatal tetanus in a rabbit by the injection of 15 c.c. of his patient’s urine.

Cases not exhibiting Paralysis.

Twenty-one cases without paralysis have been published as examples of Kopftetanus.

The chief facts about this group are as follows.

Sex. Males approx. 70%; females 29%.

Age. 1st decade. one case. no death.
     2nd " four cases. " "
     3rd " four cases. two deaths.
     4th " three cases. two deaths.
     5th " three cases. no deaths.
     6th " two cases. two deaths.

Age unknown four cases.

Method of Injury. As in the preceding group chiefly falls, kicks from horses etc. In three cases point of entrance of the bacilli indeterminate. (Mitchell, Gustalla, Hamilton).

Site of Wound. Forehead 3 cases; Nose 4 cases; Cheek 2 cases; Eyelids 1 case; Parietal region 4 cases; Lips 2 cases; Occipital region 2 cases; Idiopathic or intrabuccal 3 cases.

Thus about 33% of the wounds were on nose, cheek or/
or eyelid, as compared with 41.7% in the group of cases shewing paralysis.

**Incubation Period.** Shortest four days (Gosselin), the longest was twenty days (Brunner); the average period was about 9.9 days.

The average duration of symptoms was twenty days.

**Cases without paralysis.**

<table>
<thead>
<tr>
<th></th>
<th>Average Incubation</th>
<th>Average duration</th>
</tr>
</thead>
<tbody>
<tr>
<td>Non-fatal</td>
<td>11 days.</td>
<td>31.5 days.</td>
</tr>
<tr>
<td>Fatal</td>
<td>7 days.</td>
<td>9.3 days.</td>
</tr>
</tbody>
</table>

**Cases with paralysis.**

<table>
<thead>
<tr>
<th></th>
<th>Average Incubation</th>
<th>Average duration</th>
</tr>
</thead>
<tbody>
<tr>
<td>Non-fatal</td>
<td>8.8 days.</td>
<td>29. days.</td>
</tr>
<tr>
<td>Fatal</td>
<td>6.8 days.</td>
<td>7.4 days.</td>
</tr>
</tbody>
</table>

The unparalysed group shew a tendency to a longer incubation period and a longer duration of symptoms than the other group. The comparatively long duration of the fatal cases (seven in number) is due to the fact that in two, those of Billroth and St. Thomas' Hospital the duration was remarkably long—fifteen and thirteen days respectively.
Initial Symptom. This was trismus in 13 cases, spasm of cheeks or lips in 5 cases, pains in throat in 8 cases, difficulty in swallowing 1 case, stiff neck in 1 case.

Presence of Dysphagia and Dyspnoea.

<table>
<thead>
<tr>
<th>Unparalysed Cases</th>
<th>Paralysed Cases</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dysphagia in seven or 35%, two deaths.</td>
<td>24.8%</td>
</tr>
<tr>
<td>Dyspnoea in two or 10%, two deaths.</td>
<td>23.3%</td>
</tr>
<tr>
<td>Both in four or 20%, three deaths.</td>
<td>33.8%</td>
</tr>
<tr>
<td>Neither in seven or 35%, two deaths.</td>
<td>18.1%</td>
</tr>
</tbody>
</table>

Contrast the absence of either of the above symptoms in 35% of unparalysed cases, with their absence in only 18.1% of the other group.

Tetanic phenomena. - generalised in 47.9% of unparalysed, and in 51% of paralysed cases.
- local in 50% of unparalysed and in 48% of paralysed cases.

Thus the proportion of generalised to localised examples of tetanus is about the same in both groups of cases.

Facial Contracture and Spasm. The facial muscles were affected by contracture or spasm in eighteen cases. In six cases this was bilateral, in eight cases unilateral, on same side as the wound. In two/
two cases the side of the face opposite to the wound was affected (Gosselin, Van Spange), and in two cases contracture is described as affecting the muscles acting on the lips only. (Buss and Larry II.)

Van Spange was in some doubt as to whether there might not be some facial paralysis present in his case. Gosselin's case has been already mentioned, when the simulation of facial paralysis by contracture was discussed.

Unilateral trismus is mentioned by Terrier, Gosselin, and Duplay, the latter of whom gives a fairly full account of his case, where there was a tonic contracture of all the muscles of the right side of the face, as well as of the right masseter.

One or two points of interest have already been noted, viz. - the deviation of the uvula to the left side, with left facial contracture in Nicolaysen's case - the stiffness of the tongue in Mitchell's case. Gustalla states, that the tongue in his case was affected by spasms.

Neuman found that a partial protrusion of the tongue, which was stiff, caused a peculiar fixation of the larynx; all the muscles attached to the hyoid bone being thrown into spasm.

Sensory/
Sensory disturbances are not mentioned in any case.

Concerning the general symptoms there is nothing particular to be noted.

**Mode of Termination.** In three cases death was probably due to asphyxia. Jackse gives oedema of the lungs as the cause of death, and Larrey (second case) describes fatal meningitis supervening on the 25th day, till when the patient was progressing favourably.

**Mortality.** 21 cases; 11 recoveries; 10 deaths; mortality 47.6%.

Contrast this with the mortality of the previous group of cases, viz. 56.4%.

Post Mortem examinations have been conducted in six cases with negative result.

Hough demonstrated the presence of tetanus bacilli in cultures made from the wound, otherwise bacteriological examination was negative.

Some writers, such as Neuman, contend that these non-paralytic cases represent the mildest degree of Kopftetanus, and that when the bacterial infection is more/
is more virulent, then paralytic phenomena become manifest. The longer incubation period, the more prolonged duration of symptoms, the less frequent occurrence of dyspnoea and dysphagia, might justify one in holding that these cases are milder and more chronic in type than those showing paralysis; but the slight difference in mortality (see table above) between the two classes of cases does not bear this contention out. The cases of Gosselin, Van Spanje and Larrey, for instance, are practically as severe as any of those included in the paralysed group. The latter group, as has already been shown, includes many mild cases.

Let the facts, which are held to justify the inclusion of these non-paralysed cases under the heading Kopftetanus, be for a moment considered.

1. A wound in region of cranial nerves.
2. Onset of tetanus usually localised to muscles of face, jaw, and neck.
3. Frequent presence of difficulties in swallowing and respiration.

Many cases of tetanus occurring after wounds on the head or face are to be found in the literature, which are in all respects ordinary cases of tetanus, and are described as such. For instance a case described by Dr. G. B. Morgan in the British Medical Journal 1898, which would satisfy the three conditions/
If tetanus is localised, the localisation of spasms is always in the muscles of the jaw and neck, whether the wound be on the toe or on the head. There is nothing peculiar in the localisation of tetanic phenomena in the cases just considered. It is questionable if the dyspnoeic and dysphagic phenomena are more frequently present in these so-called cases of Kopftetanus than in cases of ordinary tetanus; at any rate they are not present in them so often as in the paralysed cases.

Dyspnoea and dysphagia present in 65% Non-par. cases.
" " " present in 82% Par. cases.

I do not think that these unparalysed cases merit a particular designation, still less should they be included under the term Kopftetanus. A good example of such a case will be found described in the British Medical Journal 1896 (Dr. C. B. Morgan) under the heading of Tetanus, which would be the best heading for the 21 cases here considered.

Treatment. I have delayed reference to this, as I intend to consider very briefly and generally the methods of treatment adopted in all the cases which I have collected. The treatment of course, is simply that of tetanus/
of tetanus. The facial or other paralysis requires no special measures, as in nearly every case it improves "pari passu" with the trismus, where the patient recovers. In one or two cases improvement has been long delayed, and some slight trace of paralysis or paresis existed some weeks after the tetanic phenomena had entirely disappeared, but this is very exceptional. If the patient suffers from marked bilateral paralysis, there may be some difficulty in feeding the patient with fluid nourishment, apart from that which may be due to pharyngeal spasm. Also the flaccid lips may act like valves and impede respiration to some extent, unless means are taken to keep them apart.

The general conclusion, which is forced upon one in the accounts of the treatment and progress of these cases, is that the different methods of treatment seem to have little real influence on the course of the disease.

As far as can be judged from reading the notes of their respective cases, Michell who fought and won with Calomel and Jalap in 1814, has just as much right to claim success for his treatment as Neuman or Duvergey with tetanus antitoxine in 1903.

The obvious/
The obvious indications, which all have recognized, are (1) to keep up the strength of the patient by plenty of nourishment, (2) to secure sleep by controlling the spasms, (3) to prevent asphyxiation, (4) to eliminate or to counteract the tetanus toxine.

The giving of Nourishment. In some cases (e.g. Reclus III, Schultze, Duvergey) there was no difficulty in swallowing and hence nourishment was taken well. In the majority of cases nutrient enemata have had to be relied on for longer or shorter periods, but in a few cases they excited spasm and were rejected.

Forcible opening of the mouth by gags and the passage of a stomach tube, or the passage of a nasal tube, have been tried by Von Wahl, Oliva, Nerlich, and Behr. All the cases died except that of Oliva, where distinct benefit resulted from this forcible procedure.

Maylard gave his patient chloroform and passed a tube several times, with but temporary benefit, however.

Brunner III, Humphry, and Güterbock attempted to pass a tube, but failed.

If the reflex irritability of the muscles is great, the passage of a tube should not be attempted as/
as it only leads to severe and prolonged general spasms, which exhaust the patient. In a case such as Oliva's, where this reflex irritability is not marked, some good may be expected from this form of feeding.

Sir George Humphry has suggested the performance of Gastrostomy to overcome the difficulty, but this has not been attempted.

The Control of Spasms and the securing of sleep have in most cases been attempted by the free use of Bromide and Chloral, per orum or per rectum. These drugs have generally been of at least temporary benefit. After their effect passes off there is a tendency for spasms to return, with less frequency, but with greater severity; the increase of the patient's strength, after even a brief sleep, is, however, sufficient to justify their liberal use. Mayer, Lemberger; and others found great benefit from the use of these drugs.

Morphia has frequently been used, the same remarks apply to it.

Antipyrine has been tried in some of these cases, but without any good effect.

Chloroform has been used frequently to control spasms; Braennecke, Von Wahl, Kirchoff and Güterhock/
Güterbock, found that its use tended to complicate the respiratory difficulties, which existed in their cases. The effect of its use in one instance in Dr. Smart's case was distinctly beneficial, threatened asphyxia being averted.

To prevent Asphyxia tracheotomy has been performed in seven cases, which I give here:

Maylard, for threatened suffocation from spasm of the glottis, relief given, death from Heart failure three days later.

Plücker, for glottic spasms, relief given, death from heart failure some days later.

Von Wahl, for suffocative attacks, relief given, death not attributed to asphyxia.

Brunner (cases one and two) both improved by tracheotomy as far as respiratory difficulties were concerned.

Both were fatal.

Wagner, patient died in a suffocative attack in spite of tracheotomy having been performed.

Humphry (third case) for laryngeal spasms - recovery.

Tracheotomy will afford relief to the patient, if the respiratory difficulty is due to laryngeal spasm.
spasm, but is of no service if the diaphragm and thoracic muscles are chiefly affected.

**Elimination or Counteraction of the Toxines.**

Excision, Canterization and the antiseptic cleansing of wounds have been carried out with varying degrees of thoroughness, but apparently with little benificial effect in these cases.

Willard thought the subcutaneous division of the nerve, which was in-proximity to the wound, benifited his case; though the reason why is not clear.

Different preparations of antitoxine (Debrin, Park, Davies, and Tizzoni) have been employed in 13 cases, with 9 recoveries. The mortality of cases so treated was thus 30%, compared with 55% of paralysed, and 47.6% of non-paralysed, cases.

This mortality contrasts favourably with the figures given in a review on the results of antitoxine treatment of 90 cases of tetanus by Schuckmann, in the Deutsch.Med. Woch. March 5, 1903. He found that the mortality of cases treated with antitoxine was 43.3%, and of those untreated 53.3%.

When, however, one examines the cases of Kopftetanus treated success fully with antitoxine, one/
one finds that without exception they are of essentially mild type, and that vigorous medicinal treatment was being carried out at the same time; while the antitoxine was given too late to be of much service in several.

The following are the cases which recovered -

Croly - only gave one dose and then discontinued the antitoxine as he thought no benefit accrued from the first dose.

The cases of Duvergey, Hale, and Caretti had no respiratory nor deglutition troubles, tetanus very localised, and very chronic.

Schütze - tetanus localised and chronic, antitoxine not given till 23rd day after onset of symptoms.

Neuman - (No paralysis) very mild tetanus, serum not given till 9th day after onset of the symptoms.

Schröter (Neuman) serum given 10 days after onset.

Holub - tetanus of more severe type, but patient was able to swallow and took abundant fluid nourishment. Serum given 5 days after onset.

Janin - (second case) also more severe, but patient progressed favourably under Chloral treatment till/
till the 13th day after onset of symptoms, when antitoxine was begun.

In the fatal cases of Smart, Gillett, Trevelyan, and Plücker, the antitoxine had no effect upon the severity of the symptoms.

The antitoxine was administered subcutaneously in all the cases except that of Holub, where lumbar puncture was employed.

Injections of Carbolic Acid (Bacelli's method) were tried by Duvergé, 10 centigrammes being injected under the skin at first, subsequent injections were increased until, after 5 days, 50 centigrammes were given. As antitoxine and chloral were both being given in addition, one cannot judge of the efficacy of this procedure.

An obvious method of eliminating a quantity of toxine is that employed according to "La Semaine Médicale" Sept 28, 1904, by Dr. Hodson of Cairo, who has successfully treated several cases of tetanus by drawing half a litre of blood from the arm, and injecting intravenously two litres of physiological salt solution, with a small quantity of brandy in addition. This has not been tried in any of the cases/
cases under consideration, but the method seems worthy of a trial.

I do not find that the administration of diuretics is mentioned by writers, when discussing the treatment of tetanus. As it seems probable, from the researches of Bruschettini and others, that toxine may be present in the urine, it might possibly be of service to give a powerful diuretic early in the case, with a view to eliminating a portion of the toxine.

The chief point of interest in the study of Tetanus Paralyticus, abandoning the term Kopftetanus, is of course the paralysis.

The older writers on this subject, only took paralysis of the facial nerve into account, thus Villar in 1888 stated that "paralysis has never been observed affecting other nerves than the seventh cranial nerve." From the facts already given, it is evident that the Oculomotor, the Hypoglossal, and possibly the cervical sympathetic, may all be affected by paralysis.

There have been those who said, that this paralysis was a mere coincidence, due to some cause apart from the tetanus, and there have been those, who/
who, like M. Vaillard in his Traité de Médecine et de Thérapeutique, have held that paralysis of one side of the face was only simulated by tonic contracture of the muscles of the opposite side.

It is true that in one or two cases the facial paralysis might justly be attributed to exposure to cold, and that in one or two cases, like that of Gosselin, spasm has been noted to simulate a paralysis of the opposite side; but a consideration of the 82 cases shews that in the majority the paralysis is real, is no mere coincidence, and is due directly to the presence of the tetanus bacillus.

Many attempts have been made to explain the cause of this most perplexing symptom, a few of which may be mentioned.

Rose regarded the facial paralysis as due to an inflammatory condition of the nerve trunk, which led to compression of the nerve in its passage through the petrous portion of the temporal bone. No such inflammatory condition of the trunk of the facial nerve has ever been shown microscopically, and this theory does not consider paralysis of other cranial motor nerves.

Triglia thought that the condition might be accounted/
accounted for by capillary haemorrhages in the bulb affecting the motor nuclei. Such haemorrhages have never been shown to exist. The nearest approach to them is recorded in Caird's case, where there was marked congestion about the aqueduct of Sylvius. Nankiwell attributed the paralysis to the effect of cold, hence regarding it as a coincidence. In such cases as those of Villar and Buisson the cold theory might hold good, but I would point out that in the statistics given in "The Medical and Surgical History of the War of the Rebellion" (Surgical Vol. III. p.9 918) there is no mention of facial paralysis occurring; and what cases would more certainly be exposed to cold and chill than those there dealt with?

The possible influence of arheumatic diathesis is hinted at by Nankiwell, as by many of the older writers.

Giuffre thought paralysis might be due to a functional disturbance of the trunk of the nerve, determined by the same trauma which provokes the tetanus.

Charcot thought heredity and hysteria had an effect in producing some forms of facial paralysis.

He asked - Could these have effect in any cases of tetanus?
tetanus? As the majority of cases occurred in robust males, and no family history of facial paralysis or hysteria is given by any of the patients, this hypothesis need not be considered. Gowers (Diseases of the Nervous System p.9. 631) says - "No reaction of degeneration has been found, and no disease of the facial nerve has been shown after death; hence the paralysis must be of reflex origin. Irritation of the fifth nerve may cause paralytic ptosis, and such reflex ptosis may follow division of the fifth nerve, or the extraction of a tooth."

This reflex explanation would certainly seem to fall short, in those cases where the paralysis is on the opposite side of the face to the wound, in those where the wound is remote from the fifth nerve (Larger, Solmsen, Nothnagel), and in those where the appearance of paralysis is long delayed.

The discovery of the tetanus bacillus by Nicolaier, and the experimental work of Brunner, Klemm, Mayer, Marie, Brieger, Binot and others with the tetanus bacillus and its toxines has given us a more likely explanation of the agent which causes the paralysis/
the paralysis, though its precise working still remains a complete mystery.

In the first place, I take it for granted that all the cases of tetanus paralyticus were caused by the bacillus of tetanus, though only in one case, (Solmsen) was it actually demonstrated. Inability to obtain cultures of the tetanus bacillus is the rule, rather than the exception, in cases of ordinary tetanus.

Tetanus toxine has been found in the saliva in one case; and in the blood in several cases, as already mentioned.

The modern view is that the cause of the paralysis is to be sought in the toxine produced by the tetanus bacilli located at the wound.

The authorities are divided into two camps, those who uphold a peripheral action of toxine in the production of paralysis, and those who hold that this action is central i.e. on the motor nuclei.

The former are represented by Brunner and Klemm, who both hold that there are a variety of active agents in the toxine produced by the tetanus bacillus, one or more of which have a paralysing effect.

The/
The action of the toxine is both central on the nuclei, and also peripheral on motor nerve endings, the paralysis being due to the latter, and being essentially a peripheral paralysis.

Brunner has succeeded, experimentally, in producing a paralysis of the facial muscles in rabbits, and guinea-pigs, by large injections of crude tetanus toxine into the face. Only the muscles in the immediate neighbourhood of the injections were affected. Similar inoculation with cultures of the tetanus bacillus caused no paralysis, though tetanic phenomena arose.

Attempts to cause the symptom - Complex of tetanus paralyticus, by inoculation with an isolated constituent of the toxine, failed.

Brunner found that the facial nerve in proximity to the point of inoculation showed early and marked decrease of irritability to the faradic current, and that the muscle nearest the same point gave the reaction of degeneration. He thought that only one nerve area was specially affected, and that it was in this area that the toxine had spread from the point of inoculation. Binot (These, Paris 1899) tried, without success to produce an experimental tetanus/
tetanus paralyticus by innoculating guineapigs between the eyes with tetanus toxine. He only succeeded in producing tetanic phenomena.

According to Bouchard, six kinds of "ptomaines tétaniques" have been isolated from cultures by Breiger. Of these there is one "tétano - toxine" which can produce a generalised muscular paralysis.

I mention the work of Binot and Breiger at this place simply for comparison with that of Brunner, and do not intend to imply that they hold the view that the action of the paralysing toxine is peripheral.

The other view, that paralysis is due to central action of the toxine, is upheld by Nerlich, Mayer, Neuman and others, and is founded upon clinical and post mortem evidence.

Nerlich found, on microscopic examination of the brain of his case, bubble-shaped formations in the motor cells of the nucleus of the twelfth nerve on the same side as the wound, and also little vacuoles in the cells of both seventh nuclei. The motor nuclei of the fifth nerves, shewed degeneration of motor cells, vacuoles being in process of formation. From these facts he asserted that the paralysis was of a nuclear nature. Paralysis of the tongue could not/
not easily have been demonstrated in this case, on account of the complete trismus present, and the fatal termination.

Bourgeois reports, that in his case the staining of sections by Nissel's method revealed pigmentation in the nerve cells of the hypoglossal nucleus, but the author attributes this to bad staining. This also was a case in which hypoglossal paralysis on the side corresponding to the pigmentation could hardly have been determined, even if present.

On more purely clinical evidence it is argued by Neumann, Mayer, and others, that the paralysis must result from an effect of the tetanus toxine on the central motor nuclei of the affected cranial nerves. They believe, that the paralysing components of the toxine, led to the centrum through a cranial nerve, there act in the first place upon the nucleus of the conducting nerve, and in the second place upon a neighbouring motor nucleus, in such a way, that paralysis of the nerve, whose nuclei are concerned, is produced.

Thus paralysis of the twelfth nerve arises when the toxine reaches the centrum by way of the seventh nerve, affects the seventh nucleus and then passes on/
on, and affects the twelfth nucleus, which lies in close proximity. It is evident that the twelfth nerve has no immediate connection with the seventh nerve, still less with the great majority of the wounds recorded, except in so far as its nucleus is in proximity to the seventh nucleus. Hence a local peripheral action of the toxine on the twelfth nerve appears unlikely, as the wounds recorded in the two cases of hypoglossal paralysis were remote from the tongue, whilst a central action of the toxine appears probable.

In considering which view is the more probably correct, it may be well to state a few fundamental facts.

1. Tetanus bacilli must have been present in every case of Kopftetanus, whether the point of inoculation was manifest or not.

2. Tetanus toxine produced by the bacilli is absorbed by the motor nerves in the vicinity of the point of inoculation. Ritchie in a paper read before the Pathological Section of the British Medical Association (B.M.J. Sept. 10, 1904) states that "tetanus toxine reaches nerve cells by means of the motor axones only".

Marie/
Marie and Morax (Ann. de l'Inst Pasteur Nov. 26, 1902) state that in the zone of inoculation the toxine containing lymph comes in contact with the terminal expansions of motor nerves, which absorb it, just as the roots of a plant absorb fluids.

3. Tetanic spasm is due to an action of the tetanus toxine on the motor nerve cells of the cord, medulla and pons, in virtue of which their reflex irritability is vastly increased. There are no demonstrable organic changes in the cells beyond very indefinite degeneration in some of the ganglion cells, and in the white matter. There is no effect on the sensory nor motor endings of the nerves. (Manual of Bacteriology Muir and Ritchie 1902.)

All cases of Kopftetanus have at least one thing in common and that is the production of tetanus toxine going on at the point of inoculation. A different paralysing agent cannot be conceived for each case. As the presence of tetanus toxine is the chief common feature, it is in its action that a cause of the paralysis may be looked for. This view is strengthened by the experimental work, which has shown that there are paralysing elements separable from the crude toxine.
In Kopftetanus the motor nerve through which the toxine is chiefly or wholly absorbed is the seventh, for most of the wounds occur in one or other of the regions supplied by its branches. It is possible that in a few cases, such as those where the wound is on the upper eyelid, or involves the orbit, that absorption takes place through the third nerve, (Schröter, Maylard, Trevelyan, Reclus (two cases)). Curiously in not one of these cases is mention made of any paralytic affection of the third nerve.

From the important fact, that practically no organic changes central or peripheral have been found in relation to the pons, medulla, and cranial nerves, we must regard the paralysis as due to a functional derangement of nerve cells, or nerve fibres, or nerve endings.

The absence of reaction of degeneration in the paralysed muscles, and the transitory nature of the paralysis in non-fatal cases, both point to the functional nature of the affection.

The nuclear change noted by Nerlich would require to be supported by observation of similar changes in other cases, where the materials could be subjected/
subjected to the most careful methods of staining, before the idea of a gross lesion of the nuclear cells could be accepted. Bougeois frankly attributes similar nuclear changes found in his case to bad staining. Brunner noted reaction of degeneration in the muscles nearest the point of inoculation in his experimental work, but no similar clinical observation has been made.

The inoculation experiments of Brunner do not throw much light on the question of the peripheral or central origin of the paralysis. The results were held to support the peripheral view, but no account is given of an opposite sided paralysis corresponding to such cases as Terillon's, Wagner's, or Pollock's. Nor could a hypoglossal paralysis following a wound on the left temple (Holub), nor a bilateral ptosis following a wound in the left orbit (Roberts), nor a ptosis and facial paralysis following infection from the buccal cavity, be easily explained on Brunner's hypothesis, that the paralysis is due to the intense local action of the toxine on the motor nerve endings in the vicinity of the seat of inoculation. Still greater would be/
be the difficulty in explaining Larger's case, where a partial right facial paralysis followed a wound on the left ring finger.

On examination of the cases of partial facial paralysis, I find, that the branches of the seventh nerve affected are not always those lying nearest the wound. Thus out of thirteen partial cases, five cases shew paralysis or paresis, in branches of the facial nerve remote from the point of inoculation.

Facial diplegia has been associated, except in Caretti's case, with a median wound of the nose, an association which might seem to suggest some peripheral action of the paralysing toxine. Such an action does not, however, seem possible in the above exception, nor in two cases (Roberts, Smart) showing another form of bilateral paralysis (Nerves III., VII.) in all of which the wounds were remote from the mid-line; also I find that in five cases (Hulke, Oliva, Nankiwell, Humphry (I). Nerlich and 68 Braennecke) there was a wound in the mid-line of the nose, associated with unilateral paralysis.

I think that the clinical facts can most easily be explained by supposing a central action of/
of the paralysing elements of the toxine, and conclude that the poison passes up the motor nerve, most usually the seventh, without a peripheral paralysing effect, but that on reaching the motor nuclei in the pons and medulla, it there causes a functional paralysis of certain motor nerve-cells. The cells affected are most usually those situated in the motor nucleus of the conducting nerve, hence a facial paralysis on the same side as the point of inoculation is the rule. The poison may however pass over to the nuclei of the third, or twelfth, nerve of the same side, or to the third, seventh, or twelfth, of the opposite side, and cause a corresponding paralysis. Nuclei on both sides may be affected, one or other form of bilateral paralysis resulting.*

This result may be supposed to occur, provided that

1. The particular tetanus bacilli injecting the wound are in such condition, or are placed in such surroundings, that the paralysing elements of the toxine are present in unusual quantity.

II./

*If paralyses of the pupil dilating fibres of the facial sympathetic be admitted in some of the cases mentioned on page 60, then it is noteworthy that these fibres originate from a centre in close proximity to the motor nuclei of the cranial nerves, affected by paralysies.
II. Certain motor nerve-cells in the central nuclei (III., VII., XII.) are in such condition as to be specially influenced by the tetanus toxine, and as to have a special affinity for the paralysing ingredients of the toxine.

I do not see that any other suppositions will explain the very contradictory and perplexing clinical evidence.

The tetanic phenomena are apparently due to an increased excitability of the motor nerve cells, the change being functional and not organic. It is not unlikely that the paralysing elements, when conditions favour their presence and action, may cause a functional change in an opposite direction.

We cannot explain why the tetanising toxine of tetanus sometimes causes localised and sometimes generalised spasms, at times dysphagia, at times dyspnoea, at times both. Mere variation of the virulence of the toxine will not give a satisfactory explanation, as a reference to the cases will show (e.g. localisation is not necessarily associated with a mild infection). Just as we cannot explain this great variability of the ordinary tetanic phenomena, neither/
neither can we explain that of the paralytic phenomena.

Under different conditions, in different individuals there must be a particular vital reaction of the various motor nerve-cells to the elements of the tetanus toxine, leading them to react to that poison, each in its own particular way.

Almost no notice has been taken by various writers on Kopftetanus of the phenomena of hyperaesthesia and anaesthesia, affecting branches of the fifth nerve, in the ten cases already specified.

While the facts given are too meagre to be of much service in the study of these cases yet one or two points may be emphasized.

1. No sensory phenomena have been recorded in any of the non-paralysed cases.

2. No mention is made of the persistence of these sensory disturbances in the non-fatal cases (Klemm, Charvot, Buisson).

3. While in certain cases, as in those of Villar and Lehrnbecher, the disturbance is strictly limited to the vicinity of the wound, and might seem to arise/
arise from irritation of the sensory nerve twigs in relation to the wound; yet in other cases as in those of Nerlich and Bernhardt, the disturbance is more widespread and seems to have no direct connection with the wound.

4. As regards size and degree of septicity, the wounds in these ten cases do not differ essentially from other cases.

Perhaps these sensory disturbances of Hyperaesthesia and anaesthesia may be accounted for by a central action of some of the components of the tetanus toxine on the sensory nucleus of the fifth nerve; but the available data are too scanty to afford ground for a satisfactory conclusion.

From what has been stated, I would derive the following definition -

Tetanus paralyticus (Kopftetanus) is a peculiar form of tetanus, which, usually resulting from specific bacterial infection on the face, forehead, or scalp, is distinguished by the presence of paralysis or paresis of one or more of the motor cranial nerves and also in a few instances by sensory disturbance of the fifth nerve; in which the tetanic phenomena tend to be, but are not necessarily, limited.
limited to the muscles of the jaw and neck; and in which difficulties of déglutition and respiration are frequently, but not always, present.
LITERATURE ON KOPFTETANUS.

The Cases. (104)

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7. Rose II. Loc. cit.
103. Sériens, "De la Contracture réflex d'origine traumatique"
    These Paris 1890 No. 435.
15. Travers cited by Pflüger, Die Sensorischen Funct. des
    Rückenmark. p. 81
16. Billroth Chirurgie Klin. Wien. 1868 No. 77
18/

* In the cases underlined no paralysis is described.
24. Kirchner, Aertzlicher Berichte über des Königl. Preuss.
    Erlangen 1872.
    von. J. 1879. s. 76.
31. Wagner, Schmidt's Jahrbücher 1884 p. 139.
39./
42. Triglia, Giornale d'elle Cliniche 1886.
44. Brunner II., Loc. cit.
46. Reclus I., Cliniques Chirurgicales (Paris) 1894 p.150.
47. Reclus II., Loc. cit.
55. Gosselin, cited by Terrillon, loc. cit.
64. Caird II. Loc. cit.
67. Rockcliffe B.M.J. July 12, 1890.
68. Braennecke, Inang. dissert Goettingen 1890.
69. Behr, Inang. dissert. Tiibingen 1891.
70. Roberts, Lancet July 11, 1891.
73. Brunner V., Loc. cit.
74. Wartmann, cited by Brunner, loc. cit.
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84. Hale, B.M.J. July 1898.
85./


Créouzon, Revue Neurologique 1900.

Bourgeois, Gaz. des Hopitaux (Paris) 1900.

Janin, These Paris 1892.

Janin, These Paris 1892.


Gillett, - hitherto unpublished.


Schultze, cited by Neuman loc. cit.


Smart, - unpublished.


Seriens, already given. (See page 103)

OTHER LITERATURE CONSULTED.

111. Ritchie, B.M.J. Sept. 10, 1904.
On the following pages a short summary is given of those cases of Tetanus Paralyticus not included in Brunner's summary (Beiträge z. Klin. Chir.) 1892

(cases without paralysis excluded)
<table>
<thead>
<tr>
<th>Case</th>
<th>Age</th>
<th>Occupation</th>
<th>Date of Injury</th>
<th>Side &amp; Zone of Wound</th>
<th>Chief Symptoms &amp; Signs</th>
<th>Treatment</th>
<th>Result</th>
<th>Remarks</th>
</tr>
</thead>
<tbody>
<tr>
<td>15</td>
<td>18</td>
<td>Mr. Huntley</td>
<td>Jan. 30, 1877</td>
<td></td>
<td>Left paralytic</td>
<td>7th day after injury.</td>
<td></td>
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<tr>
<td>16</td>
<td>18</td>
<td>Mr. Huntley</td>
<td>Dec. 9, 1877</td>
<td>Left leg</td>
<td>Right leg</td>
<td>10th day after wound.</td>
<td></td>
<td></td>
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<tr>
<td>17</td>
<td>18</td>
<td>Mr. Huntley</td>
<td>Dec. 11, 1877</td>
<td>Left leg</td>
<td>Left leg</td>
<td>10th day after wound.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>18</td>
<td>18</td>
<td>Mr. Huntley</td>
<td>Feb. 4, 1878</td>
<td>Right leg</td>
<td>Right leg</td>
<td>15th day after wound.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>19</td>
<td>18</td>
<td>Mr. Huntley</td>
<td>Feb. 23, 1878</td>
<td>Left leg</td>
<td>Left leg</td>
<td>15th day after wound.</td>
<td></td>
<td></td>
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<tr>
<td>20</td>
<td>18</td>
<td>Mr. Huntley</td>
<td>Mar. 2, 1878</td>
<td>Right leg</td>
<td>Right leg</td>
<td>15th day after wound.</td>
<td></td>
<td></td>
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<td>21</td>
<td>18</td>
<td>Mr. Huntley</td>
<td>Mar. 10, 1878</td>
<td>Right leg</td>
<td>Right leg</td>
<td>15th day after wound.</td>
<td></td>
<td></td>
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<td>22</td>
<td>18</td>
<td>Mr. Huntley</td>
<td>Apr. 1, 1878</td>
<td>Right leg</td>
<td>Right leg</td>
<td>15th day after wound.</td>
<td></td>
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<td>23</td>
<td>18</td>
<td>Mr. Huntley</td>
<td>Apr. 21, 1878</td>
<td>Right leg</td>
<td>Right leg</td>
<td>15th day after wound.</td>
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<tr>
<td>24</td>
<td>18</td>
<td>Mr. Huntley</td>
<td>May 11, 1878</td>
<td>Right leg</td>
<td>Right leg</td>
<td>15th day after wound.</td>
<td></td>
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<td>25</td>
<td>18</td>
<td>Mr. Huntley</td>
<td>June 1, 1878</td>
<td>Right leg</td>
<td>Right leg</td>
<td>15th day after wound.</td>
<td></td>
<td></td>
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<tr>
<td>26</td>
<td>18</td>
<td>Mr. Huntley</td>
<td>July 1, 1878</td>
<td>Right leg</td>
<td>Right leg</td>
<td>15th day after wound.</td>
<td></td>
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<td>27</td>
<td>18</td>
<td>Mr. Huntley</td>
<td>Aug. 1, 1878</td>
<td>Right leg</td>
<td>Right leg</td>
<td>15th day after wound.</td>
<td></td>
<td></td>
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<tr>
<td>28</td>
<td>18</td>
<td>Mr. Huntley</td>
<td>Sept. 1, 1878</td>
<td>Right leg</td>
<td>Right leg</td>
<td>15th day after wound.</td>
<td></td>
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<td>29</td>
<td>18</td>
<td>Mr. Huntley</td>
<td>Oct. 1, 1878</td>
<td>Right leg</td>
<td>Right leg</td>
<td>15th day after wound.</td>
<td></td>
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<tr>
<td>30</td>
<td>18</td>
<td>Mr. Huntley</td>
<td>Nov. 1, 1878</td>
<td>Right leg</td>
<td>Right leg</td>
<td>15th day after wound.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>31</td>
<td>18</td>
<td>Mr. Huntley</td>
<td>Dec. 1, 1878</td>
<td>Right leg</td>
<td>Right leg</td>
<td>15th day after wound.</td>
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**Remarks:**
- Mr. Huntley was killed by a falling block.
<table>
<thead>
<tr>
<th>No.</th>
<th>Name</th>
<th>Date of Injury</th>
<th>Type of Injury</th>
<th>Description of Injury</th>
<th>Treatment</th>
<th>Remarks</th>
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<td></td>
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**Notes:**
- All injuries are treated with 'Architectonic (Antlox) Tincture' and 'cerium' as per the remarks column.
<table>
<thead>
<tr>
<th>Case</th>
<th>Age.</th>
<th>Occupation</th>
<th>Date of injury</th>
<th>Site, side, &amp; wound</th>
<th>Acute symptoms and signs</th>
<th>Treatment</th>
<th>Result</th>
<th>Remarks</th>
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</thead>
<tbody>
<tr>
<td>55</td>
<td>Man</td>
<td>Labourer</td>
<td>Jan 22, 1870</td>
<td>Head, right</td>
<td>Pain, swelling, fever</td>
<td>Chlor.</td>
<td>Recovered</td>
<td>No further details</td>
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<tr>
<td>59</td>
<td>Man</td>
<td>Plumber</td>
<td>Jan 23, 1870</td>
<td>Head, left</td>
<td>Pain, swelling, fever</td>
<td>Chlor.</td>
<td>Recovered</td>
<td>No further details</td>
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<tr>
<td>68</td>
<td>Man</td>
<td>Plumber</td>
<td>Dec 1, 1870</td>
<td>Head, both</td>
<td>Pain, swelling, fever</td>
<td>Chlor.</td>
<td>Recovered</td>
<td>No further details</td>
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<tr>
<td>79</td>
<td>Woman</td>
<td>Plumber</td>
<td>Dec 1, 1870</td>
<td>Head, both</td>
<td>Pain, swelling, fever</td>
<td>Chlor.</td>
<td>Recovered</td>
<td>No further details</td>
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<td>90</td>
<td>Man</td>
<td>Labourer</td>
<td>April 16, 1871</td>
<td>Head, left</td>
<td>Pain, swelling, fever</td>
<td>Chlor.</td>
<td>Recovered</td>
<td>No further details</td>
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<tr>
<td>91</td>
<td>Man</td>
<td>Labourer</td>
<td>June 27, 1872</td>
<td>Head, right</td>
<td>Pain, swelling, fever</td>
<td>Chlor.</td>
<td>Recovered</td>
<td>No further details</td>
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<tr>
<td>92</td>
<td>Man</td>
<td>Labourer</td>
<td>Nov 4, 1872</td>
<td>Head, both</td>
<td>Pain, swelling, fever</td>
<td>Chlor.</td>
<td>Recovered</td>
<td>No further details</td>
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<tr>
<td>Sequence No.</td>
<td>Case</td>
<td>Age</td>
<td>Occupation</td>
<td>Date of Injury</td>
<td>Site &amp; Stage of Inj.</td>
<td>Chief Symptoms &amp; Signs</td>
<td>Treatment</td>
<td>Result</td>
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<tr>
<td>101</td>
<td>Bouchard</td>
<td>36</td>
<td>Farmer</td>
<td>Jun. 5, 1903</td>
<td>wrist &amp; 1st ray hand</td>
<td>Tension appears, could not close his eyes, restless, anxious</td>
<td>Arthritis</td>
<td>Ural.</td>
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<tr>
<td>102</td>
<td>Diversey</td>
<td>35</td>
<td></td>
<td>Feb. 23, 1903</td>
<td>left hand to elbow</td>
<td>Tension appears, could not close his eyes, restless, anxious</td>
<td>Arthritis</td>
<td>Ural.</td>
</tr>
<tr>
<td>Case</td>
<td>Age</td>
<td>Sex</td>
<td>Date of Injury</td>
<td>Site of Injury</td>
<td>Cause of Wound</td>
<td>Chief Symptoms &amp; Signs</td>
<td>Treatment</td>
<td>Result</td>
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<tr>
<td>64</td>
<td>M</td>
<td>Male</td>
<td>Feb 2, 1979</td>
<td>Thigh</td>
<td>Wound in thigh</td>
<td>Pain, loss of power</td>
<td>Death</td>
<td></td>
</tr>
<tr>
<td>65</td>
<td>M</td>
<td>Male</td>
<td>Feb 3, 1979</td>
<td>Hand</td>
<td>Wound in hand</td>
<td>Pain, loss of function</td>
<td>Death</td>
<td></td>
</tr>
<tr>
<td>63</td>
<td>M</td>
<td>Male</td>
<td>Feb 4, 1979</td>
<td>Shoulder</td>
<td>Wound in shoulder</td>
<td>Pain, loss of function</td>
<td>Death</td>
<td></td>
</tr>
<tr>
<td>81</td>
<td>B</td>
<td>Boy</td>
<td>Mar 5, 1979</td>
<td>Chest</td>
<td>Wound in chest</td>
<td>Pain, loss of function</td>
<td>Death</td>
<td></td>
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</tbody>
</table>