A note on the effect of heat on the rinderpest immune bodies / by J.D.E. Holmes.

Bulletin No. 43. 1914.

Agricultural Research Institute, Pusa

A Note on the Effect of Heat on the Rinderpest Immune Bodies

BY

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A note on the effect of heat on the Rinderpest Immune Bodies.

RINDERPEST anti-serum, when protected from light and kept at a temperature under 90°F., maintains its original potency for long periods (two years).

Certain tests carried out by Lingard showed that serum kept in the plains of India, when exposed for a length of time to a shade temperature of over 90°F., deteriorated in potency.

Serum kept in the plains for six and a half months, during which the maximum shade temperature varied from 78°F. to 126°F., is said to have lost 40 to 66 per cent. of its protective value.

A sample of serum deposited in Madras for two months, during which the maximum shade temperature rose to 105·5°, apparently lost 55 per cent. of its potency.

Another sample of serum, kept for twenty-six months, in the plains and exposed to two hot seasons, when the maximum temperature rose to 108°F., was found to have deteriorated in value by 55 per cent.

The conclusion has, consequently, been arrived at that prolonged exposure to a shade temperature of over 90°F. is detrimental to Rinderpest anti-serum.

In field operations in the plains of India the serum is frequently exposed for short periods to a high temperature.

In order to ascertain if exposure to a moderately high temperature for several days or to high temperature for a short period had any detrimental effect on the serum the following tests were carried out. Another object of these experiments was to determine the effect of sterilisation on the potency of the serum.

The effect of keeping serum at 45°C. (113°F.) for seven days.

A quantity of serum (Brew 84) was kept at 45°C. for seven days after which its protective power was tested on hill cattle.

The original potency was fixed at a dose of 90 c. c. per 600 lbs. body weight for hill cattle.

Two bulls were injected with a quantity of serum at the rate of 90 c. c. per 600 lbs. body weight and at the same time received an inoculation of 0·5 c. c. fresh Rinderpest blood.

Bull No. 2078.—Inoculated with 22·20 c. c. serum heated for seven days at 45°C.+0·5 c. c. defibrinated V. B. from Bull No. 2073.

5th day	40·9°C.	
6th „	40·6°C.	
7th „	40·4°C.	
8th „	40·2°C.	
10th „	40·5°C.	Vesicles.
11th „	39·7°C.	Ulcers, diarrhœa.
12th „	38·6°C.	„ „
13th „	37·9°C.	Ulcers healing, diarrhœa.
14th „	38·2°C.	„ „ fæces soft.
15th „	39·2°C.	„ healed up.
24th „	Discontinued.	

Bull No. 2075.—Inoculated with 22·80 c. c. serum heated for seven days at 45°C.+0·5 c. c. defibrinated V. B. from Bull No. 2073.

9th day	40·5°C.	Vesicles.
10th „	40·0°C.	„ ulcers, diarrhœa.
11th „	38·5°C.	Ulcers, diarrhœa.
12th „	36·1°C.	„ „
13th „	Died.	

The following was the original test of this Brew of serum No. 84.

Serum Testing.

Bull No. 1032. Inoculated with 10·20 c. c. serum+0·5 c. c. defibrinated V. B. from Bull No. 1005. (36 c. c. per 600 lbs. body weight.)

4th day	40·1°C.	
5th „	40·8°C.	
6th „	41·1°C.	
7th „	41·1°C.	
8th „	41·0°C.	Vesicles.
9th „	40·7°C.	Vesicles and ulcers.
10th „	39·8°C.	Ulcers healing.
11th „	39·0°C.	„ „
12th „	Died	

Bull No. 1031.—Inoculated with 10·32 c. c. serum+0·5 c. c. defibrinated V. B. from Bull No. 1005. (36 c. c. per 600 lbs. body weight.)

4th day	40·5°C.	
5th „	40·7°C.	
6th „	40·6°C.	Fæces soft.
7th „	40·5°C.	Vesicles, fæces soft.
8th „	40·5°C.	Ulcers, diarrhœa.
9th „	40·8°C.	„ „
10th „	37·5°C.	Ulcers healing.
11th „	37·3°C.	„ „ diarrhœa.
12th „	36·0°C.	„ „ „
13th „	Died.	

Bull No. 1033.—Inoculated with 20·52 c. c. serum+0·5 c. c. defibrinated V. B. from Bull No. 1005. (54 c. c. per 600 lbs. body weight.)

6th and 7th day . .	41·0°C.
23rd day	Discontinued.

Bull No. 1034.—Inoculated with 22·56 c. c. serum+0·5 c. c. defibrinated V. B. from Bull No. 1005. (54 c. c. per 600 lbs. body weight.)

4th day	40·1°C.	
5th „	40·4°C.	
6th and 7th day . .	40·6°C.	
8th day	40·5°C.	
9th „	40·6°C.	
10th „	39·7°C.	Vesicles.
11th „	39·8°C.	„ and ulcers.
12th „	39·0°C.	Ulcers.
13th „	39·8°C.	„ healing.
14th „	39·0°C.	Ulcers healed up.
23rd „	Discontinued.	

Bull No. 1037.—Inoculated with 31·50 c. c.+0·5 c. c. defibrinated V. B. from Bull No. 1005. (108 c. c. per 600 lbs. body weight.)

4th day	40·6°C.	
5th „	41·5°C.	
6th „	41·3°C.	
7th „	41·2°C.	Vesicles.
8th „	41·2°C.	Ulcers.
9th „	40·8°C.	„
10th „	40·6°C.	„ healing.
11th „	40·0°C.	„ „ diarrhœa.
12th „	39·6°C.	„ „ „
13th „	39·8°C.	„ „ „
14th „	39·8°C.	„ healed up.
23rd „	Discontinued.	

Bull No. 1036.—Inoculated with 31·86 c. c. serum + 0·5 c. c. defibrinated V. B. from Bull No. 1005. (108 c. c. per 600 lbs. body weight.)

4th-6th day . . .	40·5°C.
7th day	40·0°C.
23rd „	Discontinued.

The result shows that serum heated to 45°C. (113° F.) for seven days does not lose any of its protective value.

The effect of heating serum to 55°C.(131°F.) for one hour.

After heating, the serum was tested on two hill bulls in the usual methods. The dose of serum used was at the rate of 90 c. c. per 600 lbs. body weight.

Bull No. 1098.—Inoculated with 25·60 c. c. serum heated for 1 hour at 55°C.+0·5 c. c. defibrinated V. B. from Bull No. 1021. (90 c. c. per 600 lbs. body weight.)

6th day	40·2°C.	
7th „	40·2°C.	
8th „	40·5°C.	
9th „	41·4°C.	
10th „	40·3°C.	Vesicles.
11th „	39·5°C.	Ulcers.
12th „	39·6°C.	„ healing.
13th „	39·4°C.	„ healed up.
17th „	Discontinued.	

Bull No. 1105.—Inoculated with 25·60 c. c. serum heated for 1 hour at 55°C.+0·5 c. c. defibrinated V. B. from Bull No. 1021. (90 c. c. per 600 lbs. body weight.)

6th day	40·4°C.	
8th „	40·0°C.	
10th „	39·5°C.	Vesicles.
11th „	39·4°C.	Ulcers.
12th „	39·7°C.	„ healing.
13th „	39·4°C.	„ healed up.
17th „	Discontinued.	

The following was the original test of this Brew of serum No. 82 :—

Serum Testing.

Bull No. 577.—Inoculated with 10·62 c. c. serum+0·5 c. c. defibrinated V. B. from Bull No. 531. (36 c. c. per 600 lbs. body weight.)

5th day	40·8°C.	
6th „	41·0°C.	
7th „	41·3°C.	
8th „	40·6°C.	
9th „	40·8°C.	Vesicles.
10th „	40·1°C.	Ulcers.
11th „	40·5°C.	Ulcers healing, diarrhœa.
12th „	36·5°C.	„ „ „
13th „		

Bull No. 578.—Inoculated with 12 c. c. serum+0·5 c. c. defibrinated V. B. from Bull No. 531. (36 c. c. per 600 lbs. body weight.)

4th day	40·0°C.	
5th „	40·6°C.	
6th „	40·9°C.	
7th „	40·6°C.	
8th „	40·7°C.	Vesicles.
9th „	40·4°C.	Ulcers.
10th „	38·6°C.	„ diarrhœa.
11th „	Died.	

Bull No. 580.—Inoculated with 24·84 c. c. serum+0·5 c. c. defibrinated V. B. from Bull No. 531. (54 c. c. per 600 lbs. body weight.)

8th and 10th day	40·0°C.
17th day	Discontinued.

Bull No. 582.—Inoculated with 24·84 c. c. serum+0·5 c. c. defibrinated V. B. from Bull No. 531. (54 c. c. per 600 lbs. body weight.)

5th day	40·5°C.
6th and 7th day	41·2°C.
8th day	41·0°C.
9th „	40·5°C.
17th „	Discontinued.

Bull No. 579.—Inoculated with 38·52 c. c. serum+0·5 c. c. defibrinated V. B. from Bull No. 531. (108 c. c. per 600 lbs. body weight.)

5th and 7th day	40·5°C.
8th day	40·7°C.
9th „	40·1°C.
17th „	Discontinued.

Bull No. 581.—Inoculated with 39·60 c. c. serum+0·5 c. c. defibrinated V. B. from Bull No. 531. (108 c. c. per 600 lbs. body weight.)

No lesions or temperature reaction.

17th day	Discontinued.

Serum heated to 55°C. (131°F.) for one hour does not lose any of its potency.

The effect of heating serum to 60°C. (140°F.) for 1 hour.

The heated serum was tested on four hill bulls. The dose of serum used was at the rate of 45 c. c. and 90 c. c. per 600 lbs. body weight.

Bull No. 78.—Inoculated with 13·05 c. c. serum heated for 1 hour at 60°C.+0·5 c. c. defibrinated V. B. from Bull No. 79. (45 c. c. per 600 lbs. body weight.)

8th day	40·4°C.	
9th „	40·0°C.	Vesicles.
10th „	39·5°C.	„
11th „	39·5°C.	Ulcers, diarrhœa.
12th „	39·0°C.	„ healing, slight diarrhœa.
13th „	38·1°C.	„ healed up.
18th „	Discontinued.	

Bull No. 77.—Inoculated with 13·80 c. c. serum heated for 1 hour at 60°C.+0·5 c. c. defibrinated V. B. from Bull No. 79. (45 c. c. per 600 lbs. body weight.)

5th day	40·0°C.	
6th „	40·0°C.	
7th „	40·4°C.	
8th „	40·4°C.	
9th „	40·1°C.	Vesicles.
10th „	39·2°C.	„ ulcers, slight diarrhœa.
11th „	39·3°C.	Ulcers healing.
12th „	28·4°C.	„ healed up.
18th „	Discontinued.	

Bull No. 61.—Inoculated with 27·15 c. c. serum heated for 1 hour at 60°C.+0·5 c. c. defibrinated V. B. from Bull No. 34. (90 c. c. per 600 lbs. body weight.)

6th day	40·5°C.
7th „	40·0°C.
17th „	Discontinued.

Bull No. 72.—Inoculated with 28·50 c. c. serum heated for 1 hour at 60°C.+0·5 c. c. defibrinated V. B. from Bull No. 34. (90 c. c. per 600 lbs. body weight.)

6th day	40·0°C.
7th „	40·5°C.
17th „	Discontinued.

The following was the original test of this Brew of serum No. 86 :—

Serum Testing.

Bull No. 1.—Inoculated with 12·66 c. c. serum+0·5 c. c. defibrinated V. B. from Bull No. 4. (36 c. c. per 600 lbs. body weight.)

6th day	40·0°C.	
7th ,,	40·1°C.	
9th and 10th day	40·0°C.	
21st day	Discontinued.	

Bull No. 5.—Inoculated with 12·66 c. c. serum+0·5 c. c. defibrinated V. B. from Bull No. 4. (36 c. c. per 600 lbs. body weight).

4th day	40·3°C.	
5th ,,	40·7°C.	
6th ,,	41·0°C.	
7th ,,	40·9°C.	
8th ,,	40·6°C.	
9th ,,	40·4°C.	Vesicles.
10th ,,	40·5°C.	,, diarrhœa.
11th ,,	40·0°C.	,, ulcers.
12th ,,	39·0°C.	Ulcers healing.
13th ,,	38·9°C.	,, ,, diarrhœa.
14th ,,	38·5°C.	,, ,, ,,
15th ,,	38·2°C.	,, ,, ,,
16th ,,	37·6°C.	,, ,, ,,
17th ,,	38·8°C.	,, ,, ,,
21st ,,	Discontinued.	

Bull No. 6.—Inoculated with 22·08 c. c. serum+0·5 c. c. defibrinated V. B. from Bull No. 4. (54 c. c. per 600 lbs. body weight.)

7th day	40·1°C.	
11th ,,	39·0°C.	Vesicles.
12th ,,	39·6°C.	Ulcers.
13th ,,	39·1°C.	,, healing.
14th ,,	40·0°C.	,, healed up.
21st ,,	Discontinued.	

Bull No. 15.—Inoculated with 25·56 c. c. serum+0·5 c. c. defibrinated V. B. from Bull No. 4. (54 c. c. per 600 lbs. body weight.)

7th-10th day . . .	40·0°C.
21st day	Discontinued.

Bull No. 19.—Inoculated with 37·98 c. c. serum+0·5 c. c. defibrinated V. B. from Bull No. 4. (108 c. c. per 600 lbs. body weight.)

9th-11th day . . .	40·0°C.
21st day	Discontinued.

Bull No. 24.—Inoculated with 38·70 c. c. serum+0·5 c. c. defibrinated V. B. from Bull No. 4. (108 c. c. per 600 lbs. body weight.)

6th-7th day . . .	40·0°C.
8th day	40·4°C.
9th ,, ...	40·3°C.
21st ,, ...	Discontinued.

A serum heated to 60°C. (140°F.) for one hour retains its original potency.

Conclusions.

The results of these tests are in support of the conclusion that Rinderpest anti-serum does not become altered in potency by a short exposure to a high temperature ; also that the sterilisation of this serum can be accomplished without any detriment to the value of the serum.

The results are also of a further interest inasmuch as they show that the action of the Rinderpest anti-serum is not dependent on a complement contained in itself.

The experiments of Pfeiffer demonstrated that a serum was inactivated by heating to 55°C. and that inactivated serum did not kill or dissolve the bacilli against which it was prepared. It was necessary to add fresh complement obtained from the serum of a healthy animal in order to restore the destructive action.

Immune bodies are not altered by exposure to a temperature of 60-65°C. for a period of an hour whereas the complement of serum is destroyed by heating to 55°C. for half an hour.

The Rinderpest serum evidently obtains a suitable complement in the body of injected cattle.

The experiments of Todd and White[1] on the hæmolysin of the ox showed that though a suitable complement for the hæmolytic immune serum could not be demonstrated in vitro, from the serum it was actually present in the animal body.

They found that the isolysin present in the serum of the immune cattle did not act with the complement free in the blood of these cattle but appeared to require a foreign complement. This is not in accordance with what was found by Ehrlich and Morgenroth[2] in their investigation with isolytic goat sera. In the case of the goat the injection of the animal with the corpuscles of another individual gives rise to an isolysin which acting in conjunction with the complement normally present in the serum of the goat causes the solution of the corpuscles.

Todd and White, however, were able to demonstrate that although the isolysin did not act with the complement present in ox serum, a suitable complement does exist elsewhere in the body.

Fresh immune serum which showed no reaction on ox corpuscles in vitro unless foreign complement were added was injected into a bull.

One litre of the serum was injected intravenously and a few hours after the injection the urine was very darkly hæmoglobin stained, showing that a suitable complement had been forthcoming.

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[1] Todd and White. On the Hæmolytic Immune Isolysins of the Ox and their relation to the question of Individuality and Blood Relationship. *Journal of Hygiene*, Vol. X, 1910, pp. 185—195.

[2] Ehrlich and Morgenroth. Studies on Hæmolysis.

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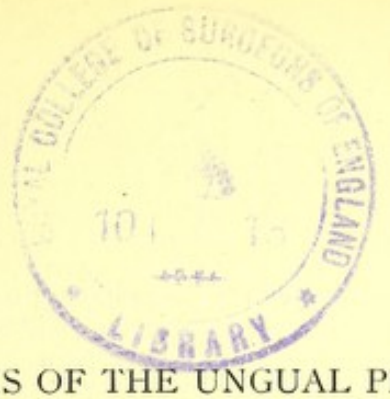

9

PARALYSIS OF THE UNGUAL PHALANX OF THE THUMB FROM SPONTANEOUS RUPTURE OF THE EXTENSOR POLLICIS LONGUS

THE SO-CALLED DRUMMER'S PALSY

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The structure of tendons represents so perfect a combination of strength and elasticity that rupture under normal conditions is practically impossible. When such accidents occur as the result of violent strains, either the muscle tears or the periosteal insertion of the tendon gives way, often carrying with it a fragment of the subjacent bone.

Before a rupture of the tendon itself takes place, it must first have been weakened by disease, the resulting degenerative changes rendering it incapable of bearing the normal strain of muscular action. For example, the long head of the biceps may rupture after the tendon structure has first been weakened by inflammatory changes secondary to an arthritis of the shoulder joint.

The particular accident which is the subject of this paper is an extensor paralysis of the distal phalanx of the thumb from spontaneous rupture of the tendon of the extensor pollicis longus. The affection is evidently rare, and the case which is herein described is the only one which has ever come under my observation, nor am I aware of any contributions to this subject in American literature. Most of the observations are of German origin, and more especially the medical reports of the German army for reasons which will be referred to later.

REPORT OF CASE

History.—The patient, a tailor, aged 31, devotes from ten to twelve hours each day to his occupation, and much of this

time is spent in sewing with a coarse, heavy needle. In addition he also uses an iron in pressing clothes. He is very insistent that there was no warning pain or discomfort in the affected thumb previous to the onset of the paralysis, which occurred in the following manner:

Oct. 25, 1914, after a day's work, he was preparing to leave the shop, and inserted his right hand in the trousers pocket searching for some object, twisting and turning the hand about as one does when trying to explore its various angles

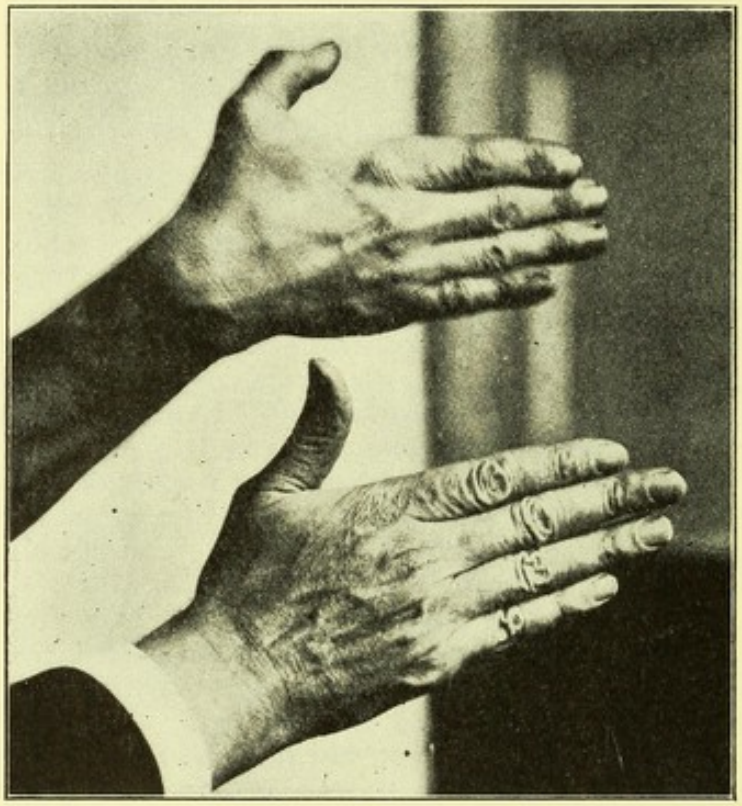

Fig. 1 (Case 1).—Rupture of the tendon of the extensor pollicis longus. Note the isolated extensor paralysis of the ungual phalnax of the thumb. Below is a normal hand showing the thumb in full extension.

and recesses. While doing this he was suddenly seized with a sharp and very severe pain over the posterior surface of the wrist on its radial side, and the back of the first metacarpal bone. On removing his hand from the pocket he was quite unable to extend the end phalanx of the thumb, while flexion could be readily performed. He had great pain over the dorsal surface of the hand and first metacarpal bone which continued for several days, and this region was very sore and sensitive to the touch for a fortnight. Since the accident he

has been unable to use the needle and pursue his accustomed calling. In addition to pain and tenderness, there was also some swelling in the same region.

Examination.—Dec. 1, 1914, there was a complete isolated paralysis of the extensor of the end phalanx of the right thumb (Figs. 1 and 2). The patient could flex quite strongly this segment of the thumb, but there was not the slightest power of extension. In contrast to this, there was very fair ability to extend the basal phalanx of the thumb as well as the first metacarpal bone. All the other movements of the hand and fingers were perfectly normal in every way. The paralysis and disability were strictly limited to the distribution of the extensor pollicis longus muscle or, as it is sometimes termed in our anatomic nomenclature, the extensor secundi internodii pollicis.

When the tendons on the dorsal surface of the thumb were rendered prominent by strong extension, the normal prominence of the extensor pollicis longus was found to be absent. This tendon, as is well known, forms the ulnar border of that small triangular space which the early anatomists termed the snuff-box. The radial border of the so-called snuff-box or *tabatière* is formed by the tendons of the extensors of the basal phalanx and the metacarpal bone of the thumb, which emerge together in this region from a groove in the styloid process of the radius. The prominence of this tendon and the absence of the long extensor are well shown in Figure 2.

If the finger was drawn along the course of the long extensor of the thumb, there became palpable just below the posterior annular ligament a small, movable, rounded swelling which was still slightly sensitive to pressure. This nodule corresponds to the distal end of the ruptured tendon. The proximal end was not palpable.

Electrical examination failed to elicit any kind of response in the extensor pollicis longus muscle. Reactions of all the other muscles of the hand and forearm were normal. There were no reactions of degeneration. The sensations of the hand were normal, and a careful general neurologic examination showed normal pupillary, tendon and skin reflexes and normal musculature without evidences of atrophy or fibrillation. The urine was free from albumin and sugar, and the visceral examination was negative.

The man was told the nature of the lesion, and a suture of the severed tendon was recommended, but operative intervention was declined.

COMMENT

This peculiar type of paralysis has been well known to German military surgeons for a number of years, under the title of drummer's paralysis or drummer's tendon. In the medical reports of the Prussian army,

extending from 1870 to 1904, von Wurthenau[1] collected sixty-two cases, all occurring in drummers. The condition is the result of a chronic tenosynovitis induced by a peculiar method of holding and using the left drumstick, which causes mechanical irritation of the long extensor tendon of the thumb as it emerges from its special compartment in the posterior annular ligament of the wrist. This in time causes pathologic alterations in the tendon and its sheath, and eventually rupture takes place, either during a paroxysm of drumming or more rarely quite spontaneously. It is said that the affection occurs usually in beginners and exclusively on the left side.

It may be assumed that the same mechanism and pathologic consequences were involved in the case of the tailor which is reported above. The fact that there was no warning pain has been observed in other cases and does not exclude previous weakening of the tendon at its site of rupture below the annular ligament.

In a few of the cases, microscopic studies of the diseased ends of the severed tendons have shown hyaline swelling, necrosis and separation of the tendon fibers.

The symptomatology is essentially the same in all of the cases, namely, a sudden paralysis of the ungual phalanx of the thumb which may or may not be preceded by pain or soreness along the course of the tendon. The actual rupture may occur quite painlessly; but more frequently a sharp, piercing pain is felt over the back of the wrist, often shooting up into the forearm. The tendon may be swollen and tender, and if ruptured, a tender, movable nodule is often palpable just below the annular ligament which corresponds to the distal stump of the severed tendon.

Electrical stimulation for obvious reasons is without visible effect on the special extensor movement which is lost. Although the muscle may respond, its contraction is not registered by an extension of the ungual phalanx, owing to the tendon separation. This fact is an important one, for if the paralysis were of neural origin, as is held by some writers, reactions of degeneration would be demonstrable.

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1. Von Wurthenau: Beitrag zur Trommlerlähmung, Deutsch. mil.-ärztl. Ztschr., 1899, xxvii, 554.

Another important diagnostic sign is the absence of the prominence of the extensor pollicis longus tendon which normally forms the ulnar border of the snuff-box (Fig. 2). All of the evidence tends to show that the usual seat of tendon rupture is just after its emergence from beneath the posterior annular ligament. This has been confirmed in a number of instances by operation, and the divided tendon found to be the seat of chronic inflammatory and degenerative changes. In addition to drummers, isolated instances of its occurrence in other occupations have also been recorded, namely, in a typesetter, a wood-carver, a farmer and in waiters.

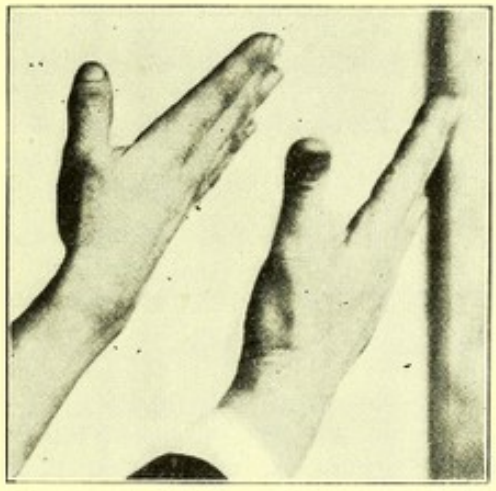

Fig. 2.—Extensor paralysis of the end phalanx of the thumb. Observe the absence at the wrist of its long extensor tendon, which forms the ulnar margin of the snuff box. The tendon of the extensor brevis pollicis stands out prominently. Below is a normal hand for comparison.

In Duplay's[2] case, reported in 1876, which appears to be the earliest description of the condition, it had occurred in a cane-maker.

Of special interest are those traumatic cases in which rupture of the tendon has followed some weeks after fracture of the radius at the wrist, the tendon having suffered injury at the time of the accident, but rupture not taking place until after the fracture had healed and the function of the hand was restored. In other cases, rupture of the tendon has followed straining injuries, such as a backward fall on the hand.

2. Duplay: Rupture sous-cutanée du tendon du long extenseur du pouce, au niveau de la tabatière anatomique, Bull. et Mém. de la Soc. de Chir. de Paris, 1876, ii, 788.

The indications for treatment are very clear. If the condition is seen immediately after rupture has occurred, an effort may be made to effect union by fixation of the thumb. In older cases and when union does not take place promptly, the only chance of restoring function is by uniting the divided tendon by some method of suture. In some of the cases it has been impossible to bring the divided ends together because the distance separating them was so great. Under these circumstances the proximal end has been sutured to the tendon of the extensor carpi radialis (Duplay,[2] Schlatter[3] and Zur Verth[4]) or to the extensor indicis longus (Hager[5]).

For anatomic reasons, a better functional result would probably be obtained by suture to one of the tendons of the thumb, either the extensor ossis metacarpi pollicis or the extensor primi internodii pollicis.

It is of interest to add that in drummers, isolated paralysis of the flexor of the ungual phalanx may also occur, caused by paralysis of the function of the flexor longus pollicis. This occurs, however, much less frequently than the extensor type, and is attributed to a faulty method of hooking the end of the thumb over the end of the drumstick to prevent its slipping from the hand. It is like the extensor palsy, a tendon disease, a chronic tenosynovitis with consecutive fragilitas tendinum and rupture. In von Zander's[6] series, there were twenty-three cases of the extensor and only three of flexor palsy of the end phalanx of the thumb.

The reasons for excluding a neural origin of these extensor palsies of the thumb are: the acute onset of the paralysis and the associated symptoms of tenosynovitis below the posterior annular ligament; evidences of rupture of the tendon either by palpation of the nodular enlargement of the distal end or by actual demonstration at operation, and the total absence of electrical responses in the extensor pollicis longus, either normal or degenerative. An additional argument against the neural theory is to be found in the joint innervation of the extensor pollicis longus and exten-

3. Schlatter: Subkutane Sehnenzerreissung an den Fingern, Deutsch. Ztschr. f. Chir., 1907, xci, 317.

4. Zur Verth: Ueber spontane Zerreissung der Sehne des langen Dammen streckers, Deutsch. Ztschr. f. Chir., 1909 cii, 569.

5. Hager: Pathogenese und Behandlung der spontanen Sehnen ruptur, Berl. klin. Wchnschr., 1886, xxiii, 360.

6. Von Zander: Trommlerlähmung, Inaug. Dissert., Berlin, 1891.

sor indicis from a single small branch of the posterior interosseous nerve, which would render isolated paralysis of the one without the other, on anatomic grounds, very improbable.

CONCLUSION

It may be said that the extensor paralysis of the end phalanx of the thumb is a very rare affection. It results from rupture of the tendon of the extensor pollicis longus as it emerges from beneath the posterior annular ligament of the wrist. Trauma and a variety of occupations have been found to be the cause of this accident, all of which have tended to induce low-grade inflammatory and degenerative changes in the tendon, which predisposes to spontaneous rupture. Drummers are especially liable to this affection; hence the term "drummer's palsy."

If evidences of union do not occur after a brief period of fixation of the part, function can be restored only by a tendon operation.

The term "drummer's palsy" is a poor one and is misleading, for the reason that the lesion is not neural and therefore not a true paralysis, nor is the condition one which is confined solely to drummers.

Therefore, if this term is used, it should be with the understanding that the affection is one of the tendon and not of the nerve.[7]

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7. In addition to the references already given, the following will be found of interest:

Bruns: Zur Pathologie der Trommlerlähmung, Neurol. Centralbl., 1891, p. 98; ibid., 1895, xiv, 897.
Cluzet and Nové-Josserand: Paralysie isolée du long extenseur du pouce, Nouv. iconog. de la Saltpétrière, 1913, xxvi, 234.
Düms: Trommlerlähmung, Handb. d. mil. Krankh., i, 210.
Henck: Beitrag zur Sehnenplastik, Zentralbl. f. Chir., 1882, No. 18.
Heineke: Ueber spontanen rupturen der Lehne des Extensor pollicis longus nach Radiusbrüchen, Deutsch. Ztschr. f. Nervenheilk., 1913, xlvii, 229.
Lindner: Ueber Subcutane Zerreissung der Sehne des Extensor pollicis longus, München. med. Wchnschr., 1890, p. 753.
Schaefer: Ueber Arbeits Paresen, Inaug. Dissert., Berlin, 1890.
Steudel: Die Trommlersehne und ihre Behandlung, Deutsch. mil.-ärztl. Ztschr., 1899, xxviii, 554.

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