Thesis.

on a

New Disease

Acute Ascending Rabic Myelitis.

by

James Arnold Waterman. MB. Glas 1923

Port of Spain

Trinidad

B.W.I.

ProQuest LLC.
789 East Eisenhower Parkway
P.O. Box 1346
Ann Arbor, MI 48106 – 1346

To whom it may Concern:-

I hereby certify on honour that this Thesis has been my own work. I have made use of notes of cases treated by other doctors. Some books have been consulted and passages quoted.

James A Watesman
MB (Glas) 3.9.3

THE GENERAL PRACTITIONER AND RESEARCH.

A FEW OBSERVATIONS BY A GENERAL PRACTITIONER.

This thesis is divided into three parts, A.B.C.

A - deals with a short historical account of the disease "Acute Ascending Rabic Myelitis".

B - deals with the clinical features of this disease

C - deals with "the Inferior Mesenteric ganglion as a reflex controller of the bladder". The evidence on which"C"is based is chiefly clinical.

A

As an introdu ction to this thesis I cannot do better than quote a passage from the writings of Dr.James McKenzie - that ideal general practitioner and research worker :-

" the barrenness of a great deal of investigation can be traced to the neglect of the principle that a knowledge of symtoms is necessary for investigation into all the problems connected with disease".

The Clinical observer still remains the solid rock from which all medical discoveries must originate and medical science advance.

The discovery of this outbreak of Acute Ascending Rabic Myelitis in Trinidad is no exception to the above rule. It was due primarily to the work of General Practitioners who although burdened with routine duties were able to diagnose the disease when ever it occurred, and in this way furnished material for bacteriological investigation.

In these days of Laboratory specialisation in diagnosis and of partial neglect of clinical methods, the Clinician is apt to depend too much on the Laboratory and too little on his own observations. The part he plays in advancing the noble art oft times receives little or no recognition compared with the halo which surrounds the Laboratory worker who, may discover a new germ or a new virus, or a new test.

To quote Sir James :

> " Often the Laboratory worker is not only unable, at times, to recognise the symtoms produced in man by the invasion of a microbe, but even, when he has obtained distinct evidence of phenomena produced by microbic invasion he is not competent to say whether the presence of the microbe is incidental, the primary cause, or secondary to some other condition".

An illustration of this occurred during the bacteriological investigation of a disease in animals.

The Bacillus Botulinus was found in the soil where cases of the disease in cows had occurred and also in two cases it was recovered from the Liver and Spleen. The disease was therefore diagnosed to be Botulism which, as will be seen later was Rabies.

Although it is not usual for details of the methods pursued in the elucidation of a new or rare disease to be published, nevertheless, it may be profitable to depart from custom to emphasise the fact, that the physician must always be prepared to investigate disease not only in human beings but also in the lower animals.

AMONG LOWER ANIMALS.

In 1925 a disease broke out in epidemic form among cattle and horses in Port of Spain in the Northern part of the Island. The disease was diagnosed by the Veterinary Surgeons as Botulism.

In 1926 - 27 - 28, cases occurred. But in June 1929 the disease began to occur in the Southern part of the Island - at Siparia, Fyzabad, Parry Lands. (See Map attached)

The symtoms were stated to be paresis of the leg and great salivation. Every case ended fatally. The diseases is of short duration 5-6 days.

Diagnosis was based both on clinical features and bacteriological findings. It was also substantiated by distinguished Veterinary surgeons in the U.S.A., on receipt of the clinical features of the disease.

POST MORTEM

Post Mortem shewed congestion of the brain and petechiae on heart muscle.

HUMAN DISEASE - 1st CASE.

In July 1929, I was sent to Siparia in the Southern part of the Island to act for Dr.Grell, the District Medical Officer.

On the 12th July 1929 at 10 a.m., a boy was brought to my office with the following history :-

On 12. 7. 29 - He complained of severe abdominal pains which started about mid-day and were so severe that he did not return to school in the afternoon.

On 13. 7. 29 - He was given some salts by his mother and his bowels acted.

On 14. 7. 29 - At about 10 a.m., he suddenly cried out for pains in his right leg and he felt feverish. Shortly afterwards he noticed that his right leg was weak.

On 15. 7. 29 - Fever continued and leg became "dead".

On 16. 7. 29 - He was brought to me.

For details of case see Section B

The case died on the 20.7.29 at 4 a.m., ~~he died~~. My diagnosis was Acute Ascending Myelitis(~~Paralysis~~).

I discussed the case fully with Dr.Rostant, Resident Surgeon of the Colonial Hospital, San Fernando and other doctors. I also mentioned the fact that a mule had died in a savannah nearby, from Botulism. I made inquiries and learnt that the boy played in the same savannah.

On 22. 7. 29 Dr Grell reported two cases - On 25.7.29 ; 30. 7. 29, two more cases. In August 6 cases and in September two cases. Some of these cases were sent to Hospital where they were attended by Dr.Rostant and my self.

Case No.7 is of especial interest from the diagnostic point of view and the importance of clinical observations.

On July 27th, she complained of headache and cold feelings in her hands and feet. She had fever and was sweating a great deal.

She was first seen by Dr.Grell on 31.7.29, and on examination her temperature was 100. Lungs clear. Heart normal. Abdomen soft. There was no paralysis and no retention of urine. She was quite bright so a provisional diagnosis of malaria was made.

On the next day - 1.8.29, the fifth day of the illness, he visited her and found the disease well advanced. T=104. Complete paraplegia, difficulty with micturition, but

On 3.8.29.- "The respiratory distress and inability to swallow closely resembled the appearance of hydrophobia in which I had seen three cases at Port of Spain, Trinidad.

She died that evening.

evidently the fact that there was no history of a dog bite and inasmuch as rabies in dogs had not occurred in the Island since 1914 made him dismiss that diagnosis.

In October 1929 - Drs/Grell and Rostant read a paper on the cases which had been treated by them and finally concluded that the disease was Acute Anterior Poliomyelitis.

I did not agree with this diagnosis on the following grounds -

1- The mortality rate being 100% was against it being Anterior poliomyelitis; for there is no record of an epidemic of Anterior poliomyelitis with 100% mortality.

2- The bladder and rectum were paralysed producing retention of urine and constipation. In Anterior poliomyelitis the sphincters are generally in tact.

3- Salivation is not typical of Anterior poliomyelitis.

4- Temperature remained high throughout the disease.

5- It is true that an acute flaccid ascending paralysis may occur in the diffuse type of anterior poliomyelitis, but it is rare.

On 22. 7. 29 Dr Grell reported two cases - On 25.7.29 ; 30. 7. 29, two more cases. In August 6 cases and in September two cases. Some of these cases were sent to Hospital where they were attended by Dr.Kostant and my self.

Case No.7 is of especial interest from the diagnostic point of view and the importance of clinical observations.

> On July 27th, she complained of headache and cold feelings in her hands and feet. She had fever and was sweating a great deal.
>
> She was first seen by Dr.Grell on 31.7.29, and on examination her temperature was 100°. Lungs clear. Heart normal. Abdomen soft. There was no paralysis and no retention of urine. She was quite bright so a provisional diagnosis of malaria was made.
>
> On the next day - 1.8.29, the fifth day of the illness, he visited her and found the disease well advanced. T=104. Complete paraplegia, difficulty with micturition, but no retention. Great respiratory distress, great defficulty in swallowing. "catching at throat, swallows like an hydrophobiac". There was marked distention of the abdomen which did not go down even after the bowels had acted with an enema. She passed her urine. Bowels never acted. She was seen the next day with the symtoms only exaggerated.

His observations pointed to the disease being Rabies but evidently the fact that there was no history of a dog bite and inasmuch as rabies in dogs had not occurred in the Island since 1914 made him dismiss that diagnosis.

In October 1929 - Drs Grell and Kostant read a paper on the cases which had been treated by them and finally concluded that the disease was Acute Anterior Poliomyelitis.

I did not agree with this diagnosis on the following grounds -

1- The mortality rate being 100% was against it being Anterior poliomyelitis; for there is no record of an epidemic of Anterior poliomyelitis with 100% mortality.

2- The bladder and rectum were paralysed producing retention of urine and constipation. In Anterior poliomyelitis the sphincters are generally in tact.

3- Salivation is not typical of Anterior poliomyelitis.

4- Temperature remained high throughout the disease.

5- It is true that an acute flaccid ascending paralysis may occur in the diffuse type of anterior poliomyelitis, but it is rare.

The Official diagnosis at the end of 1929 was that the disease in man was Anterior poliomyelitis which was known to exist in the Colony for many years and the disease in animals Botulism.

I, not being satisfied with the diagnosis of Acute Poliomyelitis, discussed the disease with several of my colleagues, and the following arguments arose :-

1- The disease in human beings occurred only in districts where animals were dying of so-called Botulism.

The reply to this statement of fact was;
It is true that wherever the disease occurred in human beings, in the same vicinity, animals are dying from botulism but, it is also true that animals are dying in several districts in which no cases of the human disease have been reported.

2- The majority of the cases occurred in the labouring class of the population. During 1929, 13 cases had occurred and of these eleven were labourers of the African race who neither possessed nor worked among animals. The other two cases were East Indians. When one considered that the animals dying from "Botulism" were cared for by East Indians, some of whom slept near their cows and were in intimate contact with them, the hypothesis that, the disease in man and in the lower animals was one and the same was difficult to accept.

3- It was pointed out that the signs and symtoms of these two diseases resembled each other.

The view of the Public Health Department was that the weight of the evidence at present was against the two diseases being the same for the following reasons :

> The disease in the lower animals was diagnosed as Botulism by the Veterinary Surgeons both from the signs and symtoms and bacteriological findings and therefore should be accepted by Medical men as being correct.

The disease in animals occurred in 1925 for the first time and has been endemic since. It was only in 1929 that the

first case of this virulent type of disease was reported in man. The question arose as to whether this disease had occurred in man during those years 1925 - 1929 but escaped recognition, or, that the transmitting agent only existed in the Southern part of the Island.

4- The next point discussed was whether the disease in man and in the lower animals was Botulism. Enquiries were made as to the possibility of eating tinned food contaminated with botulinus toxin with negative results.

As I was in Scotland during the Loch Maree outbreak I had no difficulty in stating that the disease in man was not Botulism. For, although, there might be some superficial resemblance, the picture as a whole pointed to some acute infective ascending myelitis.

The explosive nature of an outbreak of botulism, the history of eating tinned food and several of a party becoming suddenly ill were all absent. There was no dizziness or early paralysis of the 3rd nerve or ptosis except late in the disease, no loss of light reflex and accommodation.
The temperature was never subnormal. True paralysis of skeletal muscles is seldom found in botulism but was always present in these cases. The late stage of the disease when there is difficulty in swallowing and articulation with paralysis of the tongue resembled botulism.

TO SUMMARISE THE SITUATION AT THE END OF 1929 *

1- A new or at least, a rare disease was recognised in 1929 and diagnosed as acute ascending myelitis.

2- Other cases occurred and were diagnosed as Anterior poliomyelitis which was accepted by the Public Health Department.

3- The suggestion that the disease in man was botulism was discarded.

4- Animals were still dying from a disease called botulism.

5- The cases of the disease occurring in human beings were not, as was to be expected, if the diseases were the same, among those who attended and lived near to the animals dying from botulism.

6- The distribution of the disease in man did not correspond with the disease in the lower animals.

7- If the diseases are the same, how is it transmitted ?

Let us see what the different departments of medicine had contribute towards the elucidation of this disease.

The Clinician

He had recognised a clinical entity which was diagnosed as Acute Ascending Myelitis (a type of Landry's disease) on July 12th 1929.

On July 22nd 1929, Dr Grell another clinician had observed another case and was struck by the resemblance of the symtoms to Hydrophobia.

In September after several cases had been seen Dr/Grells and P. A. Rostant came to the conclusion that the disease was Anterior Poliomyelitis (Landry's type).

No clinician had investigated the disease in animals during 1929.

The Laboratory:

The Laboratory gave very little help - swabs from the throat was examined. Also, the blood and the Cerebro Spinal Fluid. Experiments on animals proved negative. But Bacillus Botulinus was found in some of the bodies of cows dying from the disease. P. M. changes did not help much.

PUBLIC HEALTH DEPT;

Public Health Departments were puzzled as to how the disease was spread especially as East Indians who attended the diseased animals seldom contracted the disease. The distribution of the disease in human beings was difficult to explain. Two cases occurred in one house after the lapse of 24 days. Also two cases occurred

on opposite sides of the street which suggested an infectious disease.

Veterinary Surgeons:

Veterinary Surgeons, were agreed that the disease in the lower animals was Botulism.

1 9 3 0.

As no Medical man had investigated carfeully the signs and symtoms of the disease reported as Botulism in the lower animals I decided to investigate the clinical features of this disease.

I discussed the disease with several Veterinary Surgeons who, with one exception, accepted the diagnosis of Botulism.

Practically in every case the signs and symtoms were very similar in my opinion to the disease in the human being.

Through the courtesy of the Gov't Farm I was shown a cow, which was demonstrated as an early case of botulism. The animal was made to walk a short distance which, it did with difficulty on account of paresis of the hind legs. After making a few steps it fell down, and it needed much persuasion and coaxing to get it up. It soon fell down again, this time never to rise ~~even~~ even though 4 men assisted it.

The clinical features of the disease were then demonstrated - Salivation, anorexia, constipation, retention of urine, paresis and paralysis of the legs. Afer this graphic demonstration I was convinced that the clinical features of so called Botulism in the lower animals and Anterior Poliomyelitis in human beings were clinically the same.

We then repaired to the office and there discussed the subject.

I suggested that the clinical features of the disease in the cow were more or less identical with those of the disease occurring in the human beings. To this the Vet. Surgeon, naively replied that, he did not know medicine and

had not seen any of the human cases and therefore could offer no opinion as to the disease in human beings, ~~but, he added, the disease in human beings~~, but, he added, the disease in the cow was Botulism. He then showed me the opinions of eminent authorities in the Veterinary World confirming his diagnosis on the clinical features he had supplied to them. And,he added, the fact that the bacillus Botulinus had been found in the soil at the Farm and in the organs of two cases dying from the disease left no reasonable doubt as to the nature of the disease.

Inasmuch as Botulism is always acquired by man as a result of the ingestion of the pre-formed toxim with some article of food i.e. tinned meat, how is botulism acquired by the lower animals I asked ?

His reply was that the toxim was produced by bacilli living on grass or old fodder and subsequently the rains washed down the toxim into the drinking pools where the animals obtained their water.

I was not convinced by this explanation as I felt that our hot tropical sun would destroy the botulinus toxin very quickly, nevertheless, in as much as, several Veterinary Surgeons had informed me that the disease was more prevalent at the beginning of the rainy season and gave the above explanation for the higher incidence, I accepted the hypothesis.

Another interesting observation made by the Veterinary Surgeons was that animals kept in stalls generally escaped the disease, while those left in pastures acquired the disease. The obvious conclusion was that the animals acquired the disease in the pasture and this fact strengthened the above explanation.

Not satisfied with the above explanation I read the chapter on Botulism, and the following passages have been taken from a system of Bacteriology in order to illustrate the unsettled condition of Veterinary science with regards to Botulism.

PATHOGENESIS OF BACILLUS BOTULINUS.

Van Ermengem states that botulism is an intoxication produced only by ingestion of the toxin.

Reputed manifestations of Botulism in animals (p.383 Vol 3 System of Bacteriology).

Apparently there are several diseases of animals with nervous manifestations which have been incorrectly grouped as being due to Botulism.

Bulbar paralysis and paralysis of the Locomotor system are the cardinal symtoms in all these diseases.

Forage poisoning

Grass sickness

Lumberneck

Bacillus Parabotulinus disease

Lamsiekte

"It is difficult to understand how botulism can be contracted by pasture fed animals."

STABILITY OF TOXIN:

" The fluid toxin exposed to light and air undergoes rapid deterioration. But if kept in sealed tubes in the dark, deterioration is less rapid. Dterioration is more rapid the higher the temperature at which the toxin is kept".

Early in July 1930, I was transferred to San Fernando Hospital and on 28. 7. 30, it was again my good fortune to record the first case of the disease for 1930. The early clinical features of this case caused me to make a provisional diagnosis of Enteric Fever, for there was, pyrexia T=99.6: P=108: R=24, extreme drowsiness, distended and tympanitic abdomen, constipation, difficulty with micturition and vomiting. The next day I recognised the disease as that of Acute Ascending Myelitis. Six days

afterwards the Uncle who lived in the same house was admitted to hospital and died within three days.

As I was convinced that the disease in man (diagnosed as Anterior Poliomyelitis) and in animals (diagnosed as Botulism) was the same, I decided to transmit the human disease to acalf. On 9.8.30, with the help of a veterinary surgeon, I injected 10 cc of the Cerebro Spinal Fluid from Case 2 - 1930, intrathecally into one calf.

Into another calf I injected 10 cc of the Cerebro Spinal Fluid subcutaneously and intramuscularly. Although my experiment was a failure in as much as I did not succeed in transmitting the disease and thus proving that my clinical observations were correct, nevertheless, I think, it accelerated research, and a reconsideration of the diseases became necessary.

In September 1930, I ~~sited~~ sailed for England to do Post Graduate work.

Let us see what progress had been made during 1930 with regards to the elucidation of these diseases.

1- Clinically.

During 1930 the clinical features of the disease in the lower animals was investigated by a Medical Practitioner who came to the conclusion that the diseases in man and the lower animals were the same.

2 - Epidemiologically.

In 1930, three cases occurred in St,Mary's Village - Oropouche, about 8 miles from Siparia where the first case was reported. Of these, two occurred in the same house.

		Date of Onset	Date of Death	Duration of Disease
A. B.	15.	28. 7. 30	1. 8. 30	5 days
A. G.	35	2. 8. 30	9. 8. 30	8 days

The wife of A. G., the other occupant of the house escaped. We were of the opinion that A. G. had acquired the disease from A. B. through the agency of some biting insect such as the stomoxys fly.

3 - Bacteriologically.

A great deal of work was being done but nothing of importance was published until 1931.

Experimental Investigations.

"

In 1930 a portion of a human brain from a fatal case was sent ot Dr.Hurst, Bacteriologist of the Lister Institute, London, and a part to the Rockfeller Institute, New York, U. S. A.

Dr.Hurst on account of the negative findings after injecting a monkey and then a rabbit was of opinion at first that we were dealing with the virus of Poliomyelitis though some doubt existed. He continued his experiments by carrying the infection through a second monkey. The effect was that the monkey became unusually excited. From the second monkey he inoculated a rabbit which developed paralytic symtoms, typical of the virus of rabies.

At the Rockfeller Institute similar experiments gave the same results, with the further experiment that the virus was carried from the rabbit to a dog and symtoms typical of rabies resulted,

In 1931 a second human brain was sent to the Lister Institute, London, and the Rockfeller Institute, N.Y.; both after passage through one monkey and a rabbit gave the same results confirmatory of rabies virus. "

Human and Animal Disease.

"

In February, 1931, Sir Wilfred Beveridge a member of the Medical and Sanitary Advisory Committee of the Secretary of State for the Colonies visited Trinidad and investigated this matter examining all the records and visiting the places where human and animal cases had occurred.

He considered that the disease in man had no resemblance to the usual form of rabies and that the symtoms and epidemiological conditions (including the absence of any rabies in dogs for many years) were against any such conclusions.

He considered the disease in man to be a transverse myelitis of unknown cause and was unable to find any evidence of casual connection between the human and animal diseases especially as the animal diseases had been declared as Botulism by the Veterinary Surgeons and as it had existed in the northern part of Trinidad for four years without any such disease in man. He approved of the general control measures then being used by the Sanitary Department.

After his return to London and after conferring with Dr.Hurst and others, Sir Wilfred Beveridge advised us that the problem was a very difficult one and probably new to science. He reported that Dr.Hurst was very certain of his results and he considered we must be prepared to accept the human and animal disease as due to the virus of rabies which Dr Hurst is inclined to regard as a new and unknown type of virus this virus.

In 1931, the diagnosis of Botulism was confirmed from the clinical aspects by Mr Montgomery, Veterinary Inspector of the Colonial Office, who visited Trinidad early this year and saw cases of the disease in animals. He had seen cases of Botulism in S.Africa. "

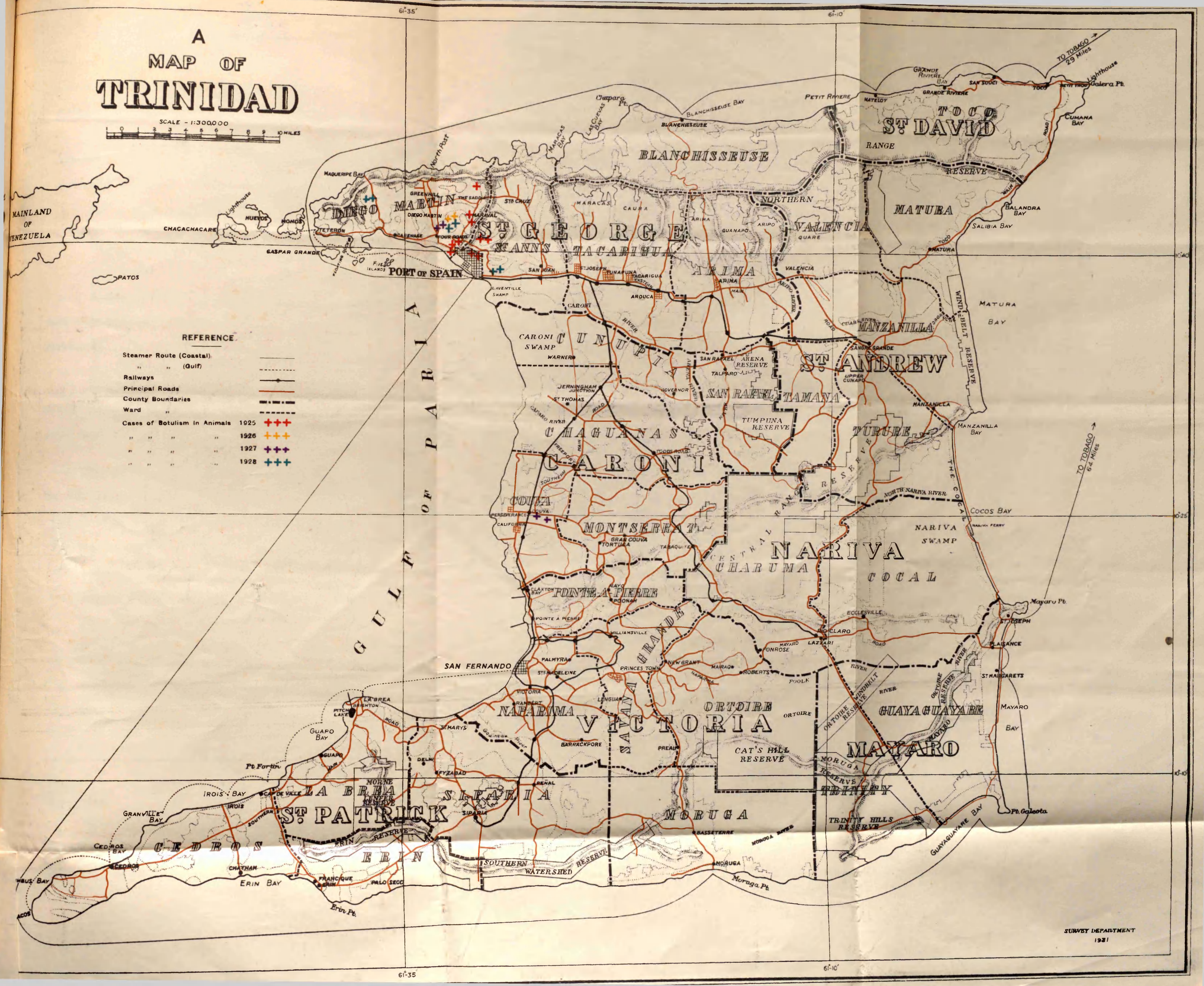
In July 1931 Dr Wise our Surgeon General read a paper in which he stated that both diseases were due to the virus of Rabies.

In Sept. 1931 negri bodies were demonstrated by Dr.Pawan in the brain of a bat with unusual habits.

In conclusion I would like to thank Dr Wise our Surgeon General for permission to write this thesis and my colleagues for making use of their clinical notes.

It is indeed a source of great satisfaction that the solving of this difficult problem was achieved by the co-operation of the members of our Medical Department in the course of their routine duties.


B
MAP OF
TRINIDAD
SCALE - 1:300,000
MILES
REFERENCE
Steamer Route (Coastal)
" " (Gulf)
Railways
Principal Roads
County Boundaries
Ward "
Cases of Transverse Myelitis 1929
" " " " 1930
" " " " 1931
Cases of Botulism in Animals 1929
" " " " 1930
" " " " 1931
MAINLAND OF VENEZUELA
CHACACHACARE
HUEVOS
MONOS
GASPAR GRANDE
PATOS
PORT OF SPAIN
GULF OF PARIA
DIEGO MARTIN
ST GEORGE
ST ANNS
TACARIGUA
BLANCHISSEUSE
ARIMA
VALENCIA
MATUBA
ST DAVID
TOCO
NORTHERN RANGE RESERVE
CARONI SWAMP
CUNUPIA
SAN RAFAEL
ST ANDREW
TAMANA
TUMPUNA RESERVE
TURURE
MANZANILLA
CHAGUANAS
CARONI
COUVA
MONTSERRAT
NARIVA
CHARUMA
COCAL
NARIVA SWAMP
CENTRAL RANGE RESERVE
POINTE-A-PIERRE
SAN FERNANDO
SAVANA GRANDE
NAPARIMA
VICTORIA
ORTOIRE
MAYARO
GUAYAGUAYARE
CAT'S HILL RESERVE
TRINITY HILLS RESERVE
MORUGA
LA BREA
ST PATRICK
SIPARIA
ERIN
CEDROS
SOUTHERN WATERSHED RESERVE
Galera Pt.
TO TOBAGO 29 Miles
TO TOBAGO 64 Miles
Mayaro Pt
Pt. Galeota
Moruga Pt.
Erin Pt.
ICACOS
COLUMBUS BAY
Pt Fortin
SURVEY DEPARTMENT
1931
LITHO BY THE GOVT. PRINTER


A
MAP OF
TRINIDAD
SCALE - 1:300,000
MILES
REFERENCE
Steamer Route (Coastal)
" " (Gulf)
Railways
Principal Roads
County Boundaries
Ward "
Cases of Botulism in Animals 1925
" " " " 1926
" " " " 1927
" " " " 1928
61°35'
61°10'
MAINLAND OF VENEZUELA
CHACACHACARE
HUEVOS
MONOS
GASPAR GRANDE
PATOS
PORT OF SPAIN
GULF OF PARIA
TO TOBAGO 29 Miles
TO TOBAGO 64 Miles
ST DAVID
TOCO
BLANCHISSEUSE
NORTHERN RANGE RESERVE
MATURA
VALENCIA
DIEGO MARTIN
ST GEORGE
ST ANN'S
TACARIGUA
ARIMA
CARONI SWAMP
CUNUPIA
CHAGUANAS
CARONI
COUVA
MONTSERRAT
ST ANDREW
MANZANILLA
SAN RAFAEL
TAMANA
TURURE
TUMPUNA RESERVE
CENTRAL RANGE RESERVE
NARIVA
CHARUMA
COCAL
NARIVA SWAMP
MATURA BAY
COCOS BAY
POINTE-A-PIERRE
SAVANA GRANDE
SAN FERNANDO
NAPARIMA
VICTORIA
ORTOIRE
GUAYAGUAYARE
MAYARO
MAYARO BAY
CAT'S HILL RESERVE
MORUGA
TRINITY
TRINITY HILLS RESERVE
GUAYAGUAYARE BAY
ST PATRICK
LA BREA
SIPARIA
CEDROS
ERIN
SOUTHERN WATERSHED RESERVE
ERIN BAY
IROIS BAY
GUAPO BAY
PITCH LAKE
Galera Pt.
Mayaro Pt.
Pt. Galeota
Moruga Pt.
Erin Pt.
Pt. Fortin
SURVEY DEPARTMENT
1931

B1

A FEW CASES OF ACUTE ASCENDING RABIC MYELITIS

by

JAMES ARNOLD WATERMAN
M.B. Ch.B. Glasgow, Gov't.Medical Officer,
Trinidad.

---00---

A few general remarks concerning the name of the disease and its characteristic features may be appreciated by ~~your~~ my. readers.

The cases reported below occurred during an outbreak of Acute Ascending Myelitis of a very virulent nature in 1929, 1930 and 1931.

The essential features of the disease may be summarised in the following definition ;-

> "An acute fatal febrile diease of short duration accompanied by severe nervous symtoms. It is characterised by a sudden onset with sensory disturbances, and early bladder and colon involvement. This is soon followed by an acute ascending flaccid paralysis commencing in the periphery of the lower extremities (at times in the upper) and rapidly spreading upwards, involving the muscles of the trunk, upper extremities, diaphragm and muscles of deglutition. The terminal symtoms are those of a bulbar paraylsis".

ETIOLOGY.

The disease is stated to be due to the virus of Rabies (Pawan and Hurst 1931). For details of experimental work see "Lancet 1931 Sept".

It is believed to be transmitted by the bites of bats which are suffering from rabies. The reservoir of the virus appears to be the domestic animals namely, cattle, horses, mules and asses. There has been No rabies in dogs for 16 years in Trinidad.

The disease is not mechanically transmitted, the transmutting agent first suffers from the disease and in its mad state attacks human beings.

> "In 1930 there occurred an unusual outbreak of rabies amongst herds in Brazil where about 30% of the cattle and 20% of equines died. The rabies virus was traced to bats which suffered from rabies and by biting these animals transmitted the disease to them. In this outbreak of rabies amongst cattle and horses in Brazil no human being nor dog was affected".

My interest in this disease started on July 16th 1929, when I saw the first case and diagnosed it as Acute Ascending Myelitis or Landry's Paralysis.

Since then, the etiology of the disease has been worked out and is stated to be due to the virus of Rabies (Pawan & Hurst, Lancet 1931). The name Acute Ascending Rabic Myelitis is therefore suggested as being scientifically correct, both from the etiological and clinical aspects.

The sites of election for inoculation of the rabies virus are the ends of the lower and upper extremities leading to two types of the disease. These sites are wisely chosen by the bat for they are frequently bare and therefore no virus toxin is lost as when bites are made thro clothing. In the 2nd place the bat can much more easily anaesthetise the part by flapping its wings and without touching the patient to arouse him.

Ins some cases. - the wound made by the bat could be recognised and in one case the patient was awakened and actually heard the bat flying about the room which was

covered with blood.

As negri bodies have been found in the brain of bats behaving queerly it is reasonable to assume that at any rate the bat is a transmitter of the disease.

It is the first time that the bat has been incriminated for conveying disease to man. Bats are indigenous to the Island and while in the past their bites which are superficial and contain only 2 small wounds were looked upon at most as a petty nuisance, now, they have to be treated seriously.

In two instances two members of a family living in the same house developed the disease and died. Two cases occurred in the same road in houses opposite to each other.

Although the fact that more than one individual in the same house acquired the disease, does not negative the bat being the transmitter, yet, it arouses the suspicion that the disease may be spread in other ways.

INCUBATION PERIOD.

In three cases in which a definite history of a bat bite was obtained the incubation periods were 21 days, 28 days and 33 days respectively.

The intervals between the onset of the disease occurring in the same house in two members of a family were :- 24 days in one case and 5 days in the other.

INCUBATION:

David Graham - 33 days. 25th Dec.1931 - 28.1.32.
E. G. (Tortuga) 6.7.31 - 3rd August - 28 days
Francis King - bite 6.9.31 - onset 27.9.31 - 21 days

From these cases the incubation periods were 21 & 28 days.

More than one member of a family in the same house suffered from the disease in three instances :-

In the first instance : - there was an interval of 24 days between the onset of the 1st case and the 8th case of the outbreak.

In the 2nd.instance :- one (Female) died on the 14.9.29 and the other (male) died on 17.9.29.

In the 3rd instance :- Onset 27.7.30 and the other 2.8.30.

The 5th case and the 11th case occurred in the same road in houses opposite to each other - at an interval of 19 days.

PROPHYLAXIS

Inasmuch as negri bodies have been demonstrated in bats advise should -

1- be given to the residents in the neighbourhood to protect themselves and animals from the bites of bats by the use of night lights in pens and nets for the beds.

2- As there may be other means of spread of the disease the affected person should be promptly isolated in hospital.

3- The next point of importance is the prompt vaccination with rabies virus of all persons in the immediate neighbourhood, who have been bitten by bats. Leaflets pointing out the advantage of obtaining efficient protection by this means may with advantage be distributed.

The question whether Rabies Vaccine should be administered to people before infection has taken place may be considered when the death rate among animals is very high and the disease is limited to a small area. The danger of neuroparalytic accidents although very small still has to be considered.

The vaccine may consist of living or dead virus. The modes of preparation of the vaccine are numerous and apparently each produces good results.

Mr Hendrick 1928 showed that the mortality was 1.16% in 84,844 cases treated with carbolised vaccines; 1.81% in 5,141 cases treated with dried cords vaccines and 1.61% in 8,435 cases treated with vaccines made from fresh nerve substance.

4- Disinfection of the house is of course essential and may be effected by the use of formalin spray. All bedding and other fomites must be sent to be sterilised.

5- Notification of Acute Ascending Myelitis; Transverse Myelitis, Anterior poliomyelitis, Encephalitis Lethargica and Cerebro Spinal Fever should be made compulsory.

6- The nursing of these patients should be entrusted to a senior nurse with experience in the nursing of infectious diseases. The duties of the nurse will be chiefly directed to the proper management of the saliva urine and faeces.

As Salivation is often profuse and inasmuch as the virus is said to be constantly excreted by the salivary glands the secretion should be received in a cup containing carbolic lotion 1-20.

As the secretion from the pancreas and intestinal glands often contains the rabies virus, the faeces of the patient should be mixed with 1-20 carbolic lotion to render it sterile.

Whether the urine contains the virus is not definitely known but no harm will be done if the urine is mixed with some 1-20 Carbolic Lotion before disposal.

The nurses in attendance on these cases had their hands examined carefully for wounds and scratches and were made to wear gloves.

T Y P E S.

There are two distinct types of the disease according as the virus enters by the upper extremity or the lower extremity. The latter type was by far the commoner.

S Y M T O M S.

The ~~source~~ Course of the disease may be divided into FOUR STAGES:

1- Onset of disease in generally sudden and acute.

2- Drowsy stage in which the patient lies quitely in bed suffering from pyrexia, distended abdomen, difficulty in micturition, constipation, furred tongue, pains in joints - closely resembling enteric fever.

3- Stage of general irritability, excitability and hyperaesthesia.

4- PARALYTIC STAGE.

The onset of paralysis is always preceded by certain premonitory symtoms which may last from a few hours to one week. These symtoms may be vomiting, constipation, diarrhoea, pains in the abdomen and joints, cold feelings in hands and feet, "pins and needles", headache. There is always a slight elevation of the Temperature.

The most characteristic symtom is a severe burning sensation in the sole of one foot and after a short interval in the other.

In a few cases the muscles of the extremities were very tender and the movement of the joints caused excruciating pains.

After the premonitory symtoms paresis of one leg occurs and shortly afterwards is followed by paresis of the other leg and difficulty with micturition. After a day or two an acute ascending flaccid paralysis of the lower limbs, bladder, large gut, rectum and trunk muscles would follow. The patient is now in the Drowsy stage - he lies quietly in bed with complete loss of power in the lower limbs - the abdomen distended partly from a distended bladder and partly as a result of paralysis of the large gut. His bowels are constipated, tongue coated.

After a varying period of hours to two days incoordination of the upper extremities can be detected and at the same time the patient begins to complain of

respiratory distress which ushers in the stage of excitement or general irritability. He is very restless tossing the upper part of his body about. He resents being disturbed. There is generally pyrexia and profuse perspiration through-out the illness. Soon there is dysphagia and profuse salivation due no doubt to paralysis of the muscles of deglutition, and, if perchance the patient should survive long enough, paralysis of the tongue and eye muscles may be noticed. Delirium or hyperpyrexia seals his doom.

The mind is perfectly clear almost to the end. Facies anxious, and as though conscious of impending dissolution, he begs that something should be done for him. Alas! a most unforgetable experience.

PARALYSIS.

The paralysis is of the flaccid type and is associated with a complete abolition of the deep and superficial reflexes of the affected areas.

In each case paresis preceded the paralysis and in some cases the patient died before paralysis had become fully established.

In the lower limb type, the paralysis gradually sprea upwards, segment by segment as though by contiguity.

In the Upper limb type the paralysis spreads from one limb to the other evidently the result of a transverse lesion in the cord and then ascends and descends pari passu. The distribution of the paralysis is at first asymmetrical but soon becomes symmetrical. In no case was there recovery of a muscle after paralysis had set in.

In two cases the paralysis ascended rapidly as far as the Costal Margin then there was a cessation in its advance for about two weeks and then recommenced to spread upwards and cause death in a few days.

The rapidity with which the paralysis became established varied: It was an index of the rate at which

the infection travelled up the cord. The following case gives a fair indication of the rate.

At 9 a.m., paresis of the lower extremities was noticed.
At 3.30 p.m., there was complete flaccid paralysis of the lower limbs.
At 9 p.m., there was commencing respiratory distress.
At 9 a.m., the next day breathing was very laboured and he swallowed with the greatest difficulty and pain. He died at 3.30 that afternoon.

In a few cases in which the paralysis was widespread and involved both upper and lower extremities and also the trunk it was noticed that the patient although lying helpless in bed was able to move either some of his toes or fingers. The last case I saw (June 1932) the patient could only move the fingers of his left hand.

Roderick Williams can move great toe right foot.
John Garcia can move great toe right foot.
Wilfred St.Rose can move toes and ankles slightly
Ethel Gluwalior slight movement of left toes.

R E F L E X E S.

The deep and superficial reflexes in the paralysed areas were absent. Early in the disease incoordination of the lower extremities could always be, demonstrated and its appearance in the upper limbs connoted that the end was not far off.

SENSORY PHENOMENA

It may be stated generally, that there is in varying degrees some disturbance with sensation. In the early stage of the disease there is frequently blunting of sensibility in the lower extremity, first attacked as compared with the other. In the case of (J.G.1930) on admission pain temperature and touch were less acute on the left lower extremity than on the right. On the other

hand in the case of (A.B.1930) sensation appeared normal the day before he died although flaccid paralysis was fully established in both lower limbs.

In two cases the anaesthesia (W.S.1929) and E.G.1931) involved the lower extremities and the trunk as far as the costal margin; but even in these cases small areas on the legs were ~~formed~~ found with normal sensation.

It must be remembered that a disease which produces drowsiness as one of its manifestations does not lend itself to the accurate determination of sensory phenomena especially when dealing with children.

Hyperaesthesia especially of the abdominal wall was noted in a few cases during the stage of excitement. Numerous and remarkable are the many different anaesthetic and paraesthic symtoms observed in this disease.

Often the patients complain of numbness, tingling, feeling of pins and needles.

P A I N.

A girdle pain is a frequent symtom and often they complain of pain in the muscles of the lower limbs, trunk and the neck and not unfrequently the muscles were very tender on palpation. In one case a slight amount of pressure on the muscles caused very severe pain and movements of his joints were likewise agonizing.

BLADDER.

The Sphincters:

The behaviour of the bladder was one of the most characteristic features of the disease and was observed very carefully. In the early stage of the disease the patient invariably complained of difficulty with micturition which gradually became worse until he could pass only a few drops of urine after much forcing.

Retention of urine then ensued and persisted through out the disease. In one case during the stage of

excitement I passed the catheter and found the mucosa hyperaesthetic, for although the catheter was passed unnoticed by the patient up the urethra nevertheless the moment it entered the bladder the patient made frantic efforts to remove it. A more detailed account of the bladder symtoms and a note on its nervous control is dealt with separately.

Rectal Sphincter:

As in the case of the bladder the poisoning by the rabies virus of the nervi enigentes produces paralysis of the walls of the rectum and large intestine and therefore constipation was usually present.

Vomiting occurred in some cases on the day of onset of the disease; in others on the day following. In two cases the vomiting was persistent during the whole course of the disease. At first the vomitus consisted of food then bile and finally of "coffee grounds". This persistent vomiting appeared to be due to the toxin elaborated by the rabies virus.

DEGLUTITION:

Difficulty in swallowing occurred in all cases sometime before death. Some complained of severe pains in the neck and others were unable to move their heads because the muscles of the neck were paralysed.

The following descriptions given respectfully by a patient - a nurse and a physician may be of interest.

"I feel as if I am choking when I try to swallow". (J.G. in '29)

" Whenever the feeding cup is brought to him he pushes it away". Nurse reporting the action of a patient 10 years old.

" Catching at throat - swallows like an Hydrophobiac The respiratory distress and inability to swallow closely resembled the appearnace of Hydrophobia in which I had seen three cases at Port of Spain" (Dr.Grell)

SALIVATION.

Salivation was very profuse and should be attributed partly to the inability to swallow and partly to excessive secretion due to the stimulation of salivary glands by the toxin of Rabies Virus.

PERSPIRATION.

In the majority of the cases, perspiration was very profuse and characteristic.

TEMPERATURE.

The history of pyrexia for a day or two before the onset of paresis was usually obtained. The pyrexia ranged from 99° to 104. It was generally irregular in character but at times continued or intermittent in type. Hyperpyrexia occurred in some cases immediately before death.

PSYCHIC FUNCTIONS.

The mind remains clear throughout the disease. Hallucinations and delirium heralded the end in some.

L U N G S.

Lungs: were normal. Towards the end râles and crêps were heard at the bases. Respiratory distress due partly to the distended abdomen and partly to paralysis of the intercostals increased as the paralysis ascended. The patient often could not speak on account of dyspnoea.

E Y E S.

In the early stages the eyes appeared normal, but in three cases there was definite involvement in the later stages. In one there was ptosis in the others paralysis of the IV and VI nerves

PATHOLOGY.

The virus of rabies can spread up, down and across the cord. The bat generally bites the ends of the upper or lower extremities for they are generally exposed, and there injects the rabies virus which travels up the nerves supplying the part bitten.

The mode of spread of the virus can easily be traced by correlating the patients symtoms.

[redacted] J. G. Age 35, stated :-

1- That pain started in the sole of his left foot on 2. 8. 30, pointing to irritation of L5 S1 S2.

2- On 4. 8. 30., the left leg and thigh began to pain him L3.4.5.

3- On 5. 8. 32, the sole of the right foot began to burn like pepper showing that the virus had reached the cord and producing sensory manifestations of the right foot.

4- Difficulty in micturition; the result pf the toxin having spread downwards to S2 S3 - the nervi erigentes.

5- Paralysis of bladder and rectum - the nervi erigentes were obviously paralysed

6- The constancy of the motor paralytic phenomena as compared with the variability in the sensory symtoms may be due to the fact that the virus in attacking the whole cord would naturally destroy the more delicate nerve cells before nerve fibres.

THE BLOOD.

White cells:- Leucocytosis in some cases otherwise normal.

C. S. F.

1. Was generally under pressure.
2. It was clear
3. Clotted spontaneously at times
4. Globulin increased
5. Cells increased -
   Polymorphs 4: Lymphocytes [illegible].

URINE.

In a few cases albumin made its appearance during the course of the disease. Sometimes granular casts were

found.
~~formed.~~ Sugar present in one case, otherwise normal.

P. M. Findings.

The brain and meninges were very congested but there was no evidence of any purulent deposit.

The spinal cord and coverings showed great congestion and the substance of the cord in some cases was extremely soft even diffluent. The gray matter could not be distinguished from the white.

The mesenteric glands were enlarged.

The internal organs were congested.

LAB. REPORTS.

Sections of Brain and Spinal Cord:- shewed

Degeneration of nerve elements and very marked perivascular round cell infiltration.

It may be stated that Glycerin is the best substance to use for the preservation of material for experimental investigation. It not only preserves the rabies virus but also prevents putrefaction and so contamination with organisms.

DIAGNOSIS.

In a typical case, the ascending flaccid paralysis, difficulty with micturition or retention of urine, the pyrexia, perspiration, salivation, respiratory distress and dysphagia make the diagnosis easy.

In an early case especially in children ENTERIC FEVER may be simulated.

Landry's Paralysis is distingusihed by the non-involvment of the bladder to any great extent, the age incidence of the disease; the absence of profuse salivation and perspiration; the mortality in Landry's paralysis is 50% only.

Intra Thecal Haemorrhage can be distinguished by a Lumbar puncture.

Acute Anterior Poliomyelitis is distinguished by

1- The temperature chart pyrexia subsides quickly.

2- The control of the sphincters is never lost.

3- Diarrhoea is common.

4- Sensation not interfered with

5- Age incidence - generally in infancy

6- Mortality varies rarely above 50%

7- Onset of paralysis is sudden and reaches its maximum effect at once.

8- There is no salivation.

Acute Transverse Myelitis is to be distinguished by the complete loss of power and anaesthesia below the lesion, the paralysis is not an ascending one . The girdle pain is always present, the flaccid type of paralysis after a time becomes spastic, bedsores are very common.

Prognosis:- is very bad. Mortality 100 %

TREATMENT:

General nursing treatment.

The bladder to be catheterised 4 hourly.

A purge for the constipation

Posture of Patient to be changed frequently

Diet fluid and nutritive.

Pyrexia relieved by sponging.

Drugs have been tried without any beneficial result

Urotropine; N.A.B.: Quinine Hydrochloride.

B 2 Cases

Name and Year	Age	Sex	Date of Onset	Admission to Hospital	Date of Death	Address	Duration of Disease
1929:							
1- C.F.	15	M	12.7.29		20.7.29	Siparia	8 days.
6- R.W.	15	M	27.7.29	1. 8. 29.	3.8.29	Siparia	7 days.
7- Y.B.	5	F	27.7.29		3.8.29	Siparia	7 days.
9- J.G.	29	M	6.8.29	13. 8. 29.	14.8.29	Fyzabad	8 days.
11- D.C.	10	M	16.8.29	19.8. 29.	20.8.29.	Siparia	4 days.
1930:							
1- A.B.	15	M	28.7.30	29. 7. 30	1. 8. 30.	Oropouche	5 days.
2- J.G.	35	M	2.8.30	7. 8. 30	9.8.30	Oropouche	8 days.
3- A.P.	38	F	16.8.30	20. 8. 30	24.8.30	Oropouche	8 days.
1931:							
2- E.G.	28	F	3.8.31	7. 8. 31	9.8.31	Mayo	6 days.
3- S.B.	8	M	15.8.31	21. 8. 31	22.8.31	Erin	7 days
4- F.K.	10	M	26.9.31	3. 10.31	4.10.31	Maraval	8 days
1932:							
1- D.G.	14	M	28.1.32	5. 2. 32.	5.2.32	Tunapuna Infected at Parry Lands.	8 days

The following cases I attended :-

1929 ; Nos: 1. 9. 11

1930 ; Nos: 1. 2. 3

The following cases I examined once during the illness and made notes.

1931 ; Nos 2. 4.

The following cases I did not see

1929 ; Nos 6. 7.

1931 ; No 3.

1932 : No 1.

CASES OF ACUTE ASCENDING RABIC MYELITIS.

No. 1.

This case is important historically as it was the first case recognised and described during the outbreak of Rabies in man in Trinidad. The diagnosis made was ACUTE ASCENDING MYELITIS, but now that the etiology is known, ACUTE ASCENDING "RABIC" MYELITIS appears more appropriate.

C. F. Age 15 - School boy.

HISTORY :-

On 12. 7. 29. - He complained of severe abdominal pains which started about mid-day and were so severe that he did not return to school in the afternoon.

On 13. 7. 29. - He was given some salts by his mother and his bowels acted.

On 14. 7. 29. - At about 10 a.m., he suddenly cried out for pains in his right leg and he felt feverish. Shortly afterwards he noticed that his right leg was weak.

On 15. 7. 29. - Fever continued and leg became "dead".

On 16. 7. 29. - He was brought to me.

ON EXAMINATION :-

There was flaccid paralysis of the right lower extremity.

Knee jerk absent, no plantar reflex, no clonus.

Sensation, although present, was less acute than on left leg.

Heart normal - Lungs normal - Spleen not enlarged.
Urine = transparent - No albumin.
T = 101, P = 96, R = 24.

His parents were advised to take him to Hospital, but they refused, saying, that they preferred him to die at home rather than at Hospital. I have often wondered since whether my gloomy prognosis frightened them or their own intuition helped them.

On 17. 7. 29. - I visited the patient and found to my great surprise that the paralysis had spread to the left lower extremity - a flaccid paralysis; that his bladder was greatly distended, for he had not passed urine that day, and his bowels had not acted.
T = 103, P = 100, R = 26.
His mind was perfectly clear, but he was very anxious about the welfare of his mother. He was intelligent and could carry on a conversation.

He complained of no pain.

On 18. 7. 29.- He was sitting up in bed with his legs outstretched, unable to move them. His breathing slightly laboured and sweating profusely. Abdomen distended. And he complained of difficulty in swallowing.

On 19. 7. 29. - T = 102 : P = 92 ; R = 30.

There was marked paresis of his upper extremities. Breathing very laboured. Abdomen distended. Constipation He was unable to swallow. His tongue heavy, speech slow and indistinct. Salivation profuse. Perspiring freely. He was drowsy, but his mind was quite clear. All sensation lost in Penis and mucous membrane of the Bladder.

On 20½ 7. 29. - At 4 a.m. he died.

No.6 . TEMP. CHART.

Roderick Williams: Male. Age 15.

School boy. Residence - Siparia. African race.

ADMITTED :- 1. 8. 29 at 5.15 p.m. Died 3.8.29 at 3.30 p.m.

HISTORY :-

On 27. 7. 29. - He woke up with a burning pain in the sole of his left foot, the pains extended up the left leg during the day and was not able to move the leg. He had fever during the day.

On 30. 7. 29. - He suffered from pains in right lower extremity and inability to move the limb.

On 31. 7. 29. - He could not pass his urine and was catheterised by his Doctor.

EXAM. ON ADMISSION:-

T = 99.4: P = 102: R = 20.
A moderately nourished youth. Bladder very distended. there is flaccid paralysis of both lower limbs but on effort slight contractions are seen in the quadriceps of both limbs. He can move the great toe of his right foot. Reflexes of lower limbs absent. Epigastric is present. There is hyper-aesthesia of the hypo-gastric area. Complains of numbness of the thighs. Pupils active and equal. He is conscious and alert. There is no dysphagia, no dyspnoea and no paresis of the upper limbs. Urine nil.

2. 8. 29. - T = 101.2 : P = 112. There is spasmodic difficulty with respiration and swallowing.

3. 8. 29. - He is restless. T = 103.2 : 120 32. Skin cold and clammy. Profuse perspiration. Quite conscious. Died 3.30 p.m. 3. 8. 29.

No. 7. (1929)

This was the 7th. case of the series and only shows what a clinician, who uses his power of observation, can do in the elucidation of the etiology of an unknown disease.

Y. B. Age 8 - School girl.

HISTORY :-

On July 27th, she complained of headache and cold feelings in her hands and feet. She had fever and was sweating a great deal.

She was first seen by Dr.Grell on 31.7.29 and on examination her temperature was 100. Lungs clear. Heart normal. Abdomen soft. There was no paralysis and no retention of urine. She was quite bright so a provisional diagnosis of malaria was made.

One the next day - 1. 8. 29, the fifth day of the illness he visited her and found the disease well advanced. T = 104 . Complete paraplegia, difficulty with micturition, but no retention. Great respiratory distress great difficulty in swallowing. "Catching at throat , swallows like an hydrophobiac". There was no marked distention of the abdomen which did not go down even after the bowels had acted with an enema. She passed her urine. Bowels never acted. She was seen the next day with the symptoms only exaggerated.

On 3. 8. 29. - "The respiratory distress and inability to swallow closely resembled the appearance of hydrophobia in which I had seen three cases at Port- of- Spain, Trinidad."

She died that evening.

NO. 9. (1929)

John Garcia. Male. Age 29. African Race. Residence - Siparia ; Occupation - Labourer.

ADMITTED :- 13th August 1929 12.30 p.m. Died 14. 8. 29.

HISTORY :

While at work 6. 8. 29., he experienced a tingling and feeling of numbness in the left lower extremity. The leg became so weak during the night that he was unable to move it.

On 7. 8. 29. - He could not move his right leg.

On 8. 8. 29. - He experienced some difficulty in passing urine.

10. 8. 29. - He could not breathe nor swallow properly.

EXAM. ON ADMISSION:

A well nourished man somewhat excitable and with an anxious expression. Heart - Nil; Lungs - Nil; Abdomen - slightly distended.

Can move upper extremities but there is obvious in-coordination. There is flaccid paralysis of both extremities; all reflexes are absent, but patient can move the great toe of his right foot. Knee jerk, plantar reflex, cremasteric reflex absent, but epigastric is present.

The cranial nerves are in tact. Bladder is distended T = 101.2 ; P = 96 ; R = 20. He obviously shows some respiratory distress and dysphagia. He is very restless. Quite conscious and mentally alert.

(9 P.M.) Skin ~~is~~ cold. Very restless, delirious at times. Coughs occasionally. Dyspnoea comes on in spasms. Unable to swallow.

14. 8. 29. - 6 a.m. T = 100.6.- Skin cold and clammy perspiring profusely, breathing very laboured. Died 9.35 a.m. 14. 8. 29

P.M. Brain and Meninges congested.
Liver and Kidneys congested.
Cord soft as butter.
Mesentery gland s enlarged.
Lungs - old pleuritic adhesions.
Bladder - peteclual haemorrhages on its surface.

P. A. R.

NO. 11 - 1929.

D. C. Male 10 years. Schoolboy.
African Race. Residence - Siparia.

ADMITTED - 19. 8. 29 at 3 p.m. Died 20. 8. 29.

HISTORY :

On 16. 8. 29, fever started in the morning.

On 17. 8. 29. - Fever continues and abdomen is painful Difficulty with rspiration.

On 18. 8. 29. Breathing much worse and he complains of difficulty in swallowing. Extremities are weak. Difficulty in passing his urine. Sweating profusely.

EXAM. ON ADMISSION.

Fairly well nourished child. Heart - nil. Lungs - nil. Abdomen very distended. Profuse sweats. Stiffness of neck muscles. Difficulty in breathing and swallowing - he pushes away feeding cup. Bladder distended. Lower limbs ~~have~~ paresis, reflexes are absent. Mentally confused. Has hallucinations. Lumbar puncture 6 p.m.

Died 7.10 a.m. 20. 8. 29.

- 1930 -

No. 1.

A. B. Male - Age 15. 28. 7. 30.

This was the first case reported in 1930. The important feature of this case was persistent vomiting.

HISTORY :-
Fever on 27th., and 28th., and difficulty with micturition.

ON ADMISSION :- 29. 7. 30 at 7.30 p.m. I found :

Bladder slightly distended.

No paresis of lower extremities.

Heart and lungs normal.

T = 99.6: P = 108: R = 24.

Shortly after admission he passed green coloured urine, the result of pills.

He had an enema and slept well during the night.

On 30. 7. 30 - 6 a.m. T=99; P=88; R=18
2 p.m. T=101.2; P=100; R=20.
6 p.m. T=101.6; P=104; R=24.
At 9 a.m., he was very drowsy with slight distension of the abdomen. He experienced some difficulty in passing his urine. There was no paresis nor paralysis of the extremities. He complained of pains in his joints. Blood was taken for the "Widal Reaction".

Evening: T=101; P=104; R=28. Pains in the knees much more severe. He was unable to move his lower extremities freely. Vomited twice dark material. Spitting very much. He is now restless. Passed his urine with increased difficulty.

31. 7. 30. - 6 a.m. T=103.8; P=96; R=24.
10 a.m. T=103; P=108; R=40
2 p.m. T=101; P=120; R=40.
9 a.m. there is definite paresis of both lower extremities. Vomited Knee jerk absent - Heart and Lungs normal.

3.30 p.m. Vomited coffee grounds looking material. There is motor paralysis of the flaccid type involving both lower extremities. Urine has to be drawn off. The patient is very restless. Respiratory rate is now 40.

9 p.m. T=102.6; P=148; R=44. Very restless. Some hyperaesthesia of abdominal wall. Vomited the same dark material several times during the night.

1. 8. 30. - 6 a.m. T=101.2; P = 124; R = 40
10 a.m. T=103.4; P=136; R=36
2 p.m. T=101.4; P=124; R=36.
There is some respiratory distress. He swallows with pain and difficulty. Perspiration is profuse. Mental condition is clear. Retention of urine continues. Lumbar puncture was performed and the fluid flowed out under increased pressure but it was quite clear. The patient died at 3.30 p.m.

No. 2 (1930)

J. G.:Male: Age 35.

J. G. - Male - Age 35, was the uncle of the last case (A.B.) and lived in the same house. In this case I noticed for the first time involvement of the 3rd., 8th, and 12th cranial nerves. I shall describe this case in greater detail.

HISTORY:-

On 2. 8. 30, he walked to the mortuary of the Hospital a distance of about 12 miles - in order to arrange for the funeral of his nephew, and while there his left foot began to burn him very much.

On 3. 8. 30.- Pain in left foot became worse.
His bowels acted that day.

On 4. 8. 30. - The left leg and thigh began to pain him and he therefore remained in bed during the day. He had a slight temperature.

On 5. 8. 30. - Sole of right foot began to burn him "like pepper". The lower bowels felt as though they were being twisted and the left leg and thigh felt paralysed. He had to force to pass his urine.

On 6. 8. 30. - Right leg began to get weak.

ON ADMISSION;

On 7. 8. 30 - 5.40 p.m. He complained of weakness of both lower extremities and a girdle pain around his waist. His bowels have not acted for four days and they feel as though they are being twisted.

ON EXAMINATION:

Paresis of both lower extremities with marked incoördination. Reflexes absent. Sensation - pain - touch - and temperature less acute on left lower extremity than on right.

Hyperaesthesia of abdominal wall.

He has to force a great deal to pass his urine.

Heart normal - Lungs rhonchitic - Abdomen tender.

Pupils normal in reaction.

NIGHT; Enema given, fluid returned discoloured.
Passing urine by drops and with difficulty.
Perspiration is profuse.
Complained of a burning pain over his bladder.

8. 8. 30.- 6 a.m. T=98.4. P = 86. R = 20.
10 a.m. T=99.8. P = 104. R = 24.

2 p.m. T=100.6. P = 108. R = 24.
6 p.m. T=100.6. P = 108. R = 24.

9 a.m. Bladder distended.21 ozs of urine drawn off. Bowels acted - constipated stool after Ol.Ric. Profuse perspiraton. He is very drowsy. Breathing very irregularly. Complains of the palms of his hands feeling cold and feels the chest stiff as if something is tightly drawn around it.

Breathing very irregularly. Complains of the palms of his hands feeling cold and feels the chest stiff as if something is tightly drawn around it.

5 P.M. - Lumbar puncture performed C.S.F. escaped under high tension. No pain felt during the insertion of the needle. He complains of difficulty in swallowing, especially with the right side of mouth, due to paralysis of the right half of the tongue. Salivation is copious. Extreme paresis of lower limbs. Speech indistinct. Incoordination of the movements of the upper extremities. He complains of pains in the muscles of the neck and chest. Ptosis of both eyelids. Mental condition good. No headache.

10 P.M. - T=101: P=120: R=32. Urine drawn off. Apparently there is no sensation in penis or bladder. Swallowed some milk with the greatest difficulty. Hiccough occurred during the night. Vomited curdled milk. Salivation and perspiration profuse Throat very painful.

9. 8. 30.-

6 a.m. T=102.8: P=128: R=36.

Lower extremities are paralysed. Respiratory distress very great - unable to speak for he needs every breath of air. Mentally clear. On protrusion of tongue is deviated to the right. Both ears are painful. Paresis of the upper extremities. Perspiration and salivation still profuse. He is having severe dysnoeic attacks. He dies shortly afterwards.

No. 3.(1930)

A. P. Female Age 38. Domestic servant.
Residence: St.Mary's Village, Oropouche. African Race.

ADMITTED TO HOSPITAL - 20. 8. 30 - Noon. Died 24.8.30. 7.15 pm

HISTORY:-

Of pains and burning in left foot for four days.

EXAM. ON ADMISSION:-

A well nourished woman who complained of a burning sensation in left foot. Heart - nil: Lungs - nil; Abdomen - soft: T=100: P=80. There was no paresis nor paralysis of her lower limbs. Knee jerks were diminished. No bladder trouble.

21.8.30.- Complains of numbness of the left leg. There is slight incoordination of lower limbs. Knee jerks are absent. There is some loss of sensation in left foot to pin pricks. T=98.4: P=100.

22. 8. 30. - Perspiring profusely. There is paresis of lower limbs. Able to extend and flex hip and knee joints. There is loss of sensation in left lower limb as far as the ankle. T=102.2: P=104; R=28. Lumbar puncture was performed, cerebro-spinal fluid was under pressure and turbid.

23. 8. 30.- Delirious at times. Unable to move lower limbs well. Paresis much increased. There is loss of sensation up to the middle of the legs.

Continued: (23.8.30.)

9 P.M. Paralysis of legs and loss of sensation up to the hips. Very restless, sweaing profusely. Delirious off and on. Bladder distended. Urine drawn off.

10 P.M. - T=100: P=96: R=24. When spoken to answers rationally.

24, 8. 30: - Complete flaccid paralysis of lower limbs. Abdomen is distended due to paralysis of bladder and large gut. Some paresis of upper limbs. There is some Dysphagia and dyspnoea. Loss of sensation to umbilicus. T=100: P=88: R=28. Died 7.15 p.m.

Duration of disease about 8 days.

- 1931 -

NO. 2. 1931.

S. G. Female. Age 28. Housewife.
East Indian. Residence Mayo.

ADMITTED TO HOSPITAL : 8. 8. 31 at 11.40 a.m. Died 9.8.31 at 10.40 p.m. Duration of diease 6 days.

HISTORY:

Of being bitten by a bat on 6.7.31 on the left foot. On 3.8.31, she suffered from fever and loss of power in left leg; cramps and painful sensations in left leg and foot. On 5.8.31 - the same symptoms appeared in the right foot. No action of bowels for eight days. Menstruation is normal.

EXAM. ON ADMISSION. 7. 8. 31. - 11.40 a.m.

Well nourished East Indian woman. Heart nil; Lungs nil; Spleen and liver not enlarged.

Left lower limb:- flaccid paralysis all reflexes absent. Loss of sensation in left foot and leg.

Right Lower limb:- No paralysis. No paresis. No loss of sensation. Knee jerk is absent. Patient can stand on right foot. There is loss of sensation of the abdominal wall as far as the costal margin. Abdominal wall reflex is absent.

Complaining of difficulty with micturation. T=98.2.: P=88.: R=16.

3.20 p.m. Complaining of pain in right leg. Mental condition is quite clear.

11. p.m. Can move the toes of the left foot a little. There is loss of sensation on the dorsum of the right foot and definite paresis of the right leg. Urine had to be drawn off. Constipation.

8. 8. 31. Left lower limb flaccid paralysis and loss of sensation.

Right lower limb, extreme paresis and loss of sensation except for a small area on the medial aspect of the knee joint and a small distance proximal to it. Had to be catheterised. Mind is clear. Patient is not in pain.

2. 30 p.m.- Abdomen very distended and tympanitic. T=105.2: 132 24. Right lower limb can be moved a little. 18 ounces of urine with-drawn. Urine Alb +++ There is no sensation in urethra and bladder.

6 P.M. There is incoordination of the upper extremities. The grips of both hands are weak. Not perspiring - Lumbar puncture performed. The C.S.F is not under tension (1) very clear. (2) pleocytosis (3) Globulin increased (4) Cells - 13 per cc in the ratio of 4 polymorphs to one lymphocyte.

9.15. p.m. Abdominal distension has increased. Complaining of thirst and insomnia. T=103.6: 128 24. There is some respiratory distress.

9. 8. 31. - 9 a.m. T=100.6: P=124.: R=24.
Dysphagia and dyspnoea much worse. Perspiring now.
Urine Alb. +++ Granular casts. No deposit. Clear and transparent. No blood. No bile. No sugar.
S.G. 1020.

Blood Haemoglobin 90% Red Cells 4½ millions.
Leucocytes 5000. Differential cell count.
Polymorphs 74% Lymphocytes 22.3%
Large Mononuclears 1.2%
Transitional 2.4%

P.M.: Kidney and liver congested
Cprd and Membranes congested
Brain and membranes congested
Pelechial haemorrhages on the surface of the heart.

Died 10.40 - 9. 8. 31.

No. 3. 1931.

S. B. Male. Age 8. School boy. African Race.

ADMITTED - 21. 8. 31. 3.10 p.m.
DIED - 22. 8. 31. 9.15 a.m.

HISTORY:-

On 17. 8. 31, soon after waking up he complained of pain in both knees but was able to walk about as usual. In the afternoon he had a temperature but passed a good night.

18. 8. 31.- He could not move his right leg.

20. 8. 31.- Profuse perspiration; constipated. No history of a bat bite.

On 21. 8. 31.- at 6 p.m., patient was seen by Dr Grell who on examination found complete flaccid paralysis of both lower extremities and loss of sensation. Reflexes absent. Abdomen distended and tympanitic. Slight respiratory distress and some difficulty with micturation. T= 103.

ON ADMISSION TO HOSPITAL:-

We found a moderately well nourished boy with flaccid paralysis of both lower extremities and loss of sensation. Reflexes absent in both lower limbs. Abdomen- distended and tympanitic. Abdominal reflex absent. Loss of sensation of abdominal wall as far as 8th rib. Bladder distended. Incoordinaton of upper limbs - grip is weak Slight respiratory distress. No dysphagia. Pupils react well. Mind clear and answers questions intelligently. Urine - no alb; no sugar; T=100; P=90; R=18.

22.8.31:- 6 a.m.; T=103.4; P=124; R=36. Paresis of upper limbs more marked. Respiratory distress worse. Unable to swallow or speak.

Died 9.15 a.m. 22.8.31.

No. 21 (1931)

F. K. Male; Age 10. Onset 26.9.31.
Died 4.10.31.

This case has many important features.

1- Etiological- as it was the second case in which we obtained a definited history of being bitten by a bat three weeks previously.

2- I was able to recognise for the first time a definite "stage of excitement" as occurs in Hydrophobia due to the bite of a rabid dog.

HISTORY:

On 26.9.31 - he complained of pains in both knees and on 27.9.31, while playing marbles his knees pained him and felt so weak that he had to stop playing and retire to his bed.

On 28.9.31 - he suffered from pains in the abdomen and was unable to get out of bed. On 1.10.31, he had difficulty in passing his urine and faeces and suffered from fever.

On 2.10.31 - Pain in abdomen increased also the difficulty in micturition.

On 3.10.31 - at 11.30 a.m., he was admitted to Hospital of which Dr.Deane is in charge.

ON EXAMINATION, he found :-

A small wound which was pointed out as the site of the bat bite on the medical aspect of the interphalangeal joint of the right great toe.

T=101.2; P=130; R=30.

Flaccid paralysis of both lower extremities, all reflexes being absent.

Sensation - Pain touch and temperature absent as far as the knees.

Paresis of the upper extremities.

Speech heavy and deliberate. Some mental apathy- Heart and Lungs normal. There is some difficulty in swallowing and respiratory distress.

4.p.m., - Paralysis of R.VI. Nerve was noticed. 4.30 p.m.- I saw him and noticed that his breathing was very laboured and chiefly diaphragmatic in type. Abdomen distended due partly to bladder and partly to paralysis of the Colon. Flaccid paralysis of the lower extremities. Marked hyperaesthesia of the abdominal wall; a touch with a pin made him cry out with pain. His mental condition had changed completely. He was excitable, irritable and restless. Facies anxious looking. He was very talkative. No salivation. No perspiration. I decided to draw off some urine for investigation. The penis was insenitive to touch and the catheter passed up the penile urethra unnoticed by the patient.

continued.

As soon as the catheter touched the mucosa of the bladder, he made a frantic efforts to get at the catheter, and having been held by the nurses, he spat at them and attempted to bite them. This was a good clinical demonstration of-

1- the sensory nerve supply of the penis and penile urethra on the one hand and the bladder on the other;

2- that the toxin elaborated, apparently acts by increasing the excitability of the nervous system, and, no doubt, the patient at that moment corresponded to the stage of excitement of the rabid dog;

3- if stimuli of a certain intensity be applied during the stage of restlessness or excitement, they may call forth in these cases of acute ascending Rabic Myelitis the rabid dog's propensities.

The urine was straw coloured, transparent, acid, no deposit, no albumin, no bile, no blood, but sugar was present. S.G. 1028.

7 p.m.- T=102.6; P=130; R=30. Very talkative, at times delirious.

4. 10. 31.- 9 a.m. Salivation had commenced. There was a little incontinence. Apparently overflow incontinence, as he had to be catheterised. The nurse reported that the patient remarked that there was something in his throat sticking him, and at times, would grip his throat with both hands.

He died at 11 a.m.

1932.

No. 1. 1932.

D. G.; Male. Age 14. African Race.
Residence: Tunapuna.

HISTORY:

He spent Christmas 1931 at Parry Lands and while staying there was bitten by a bat.

On 28.1.32- he suffered from fever, pain and weakness in both knees.

On 2. 2. 32- he could not pass his urine.

On 3. 2. 32- he was unable to walk and pass his urine which was drawn off by a doctor. He was very constipated

On 4. 2. 32- at 11 p.m., he noticed his tongue was heavy and had much difficulty to speak.

ADMITTED TO HOSPITAL :- 4 a.m. 5.2.32.

Complete flaccid paralysis of both lower limbs with loss of sensation.

Bladder distended.

Abdomen distended and tympanitic.

Respiratory distress.

Dysphagia.

Died 2.p.m. 5.2.32.

HOUR CHART.

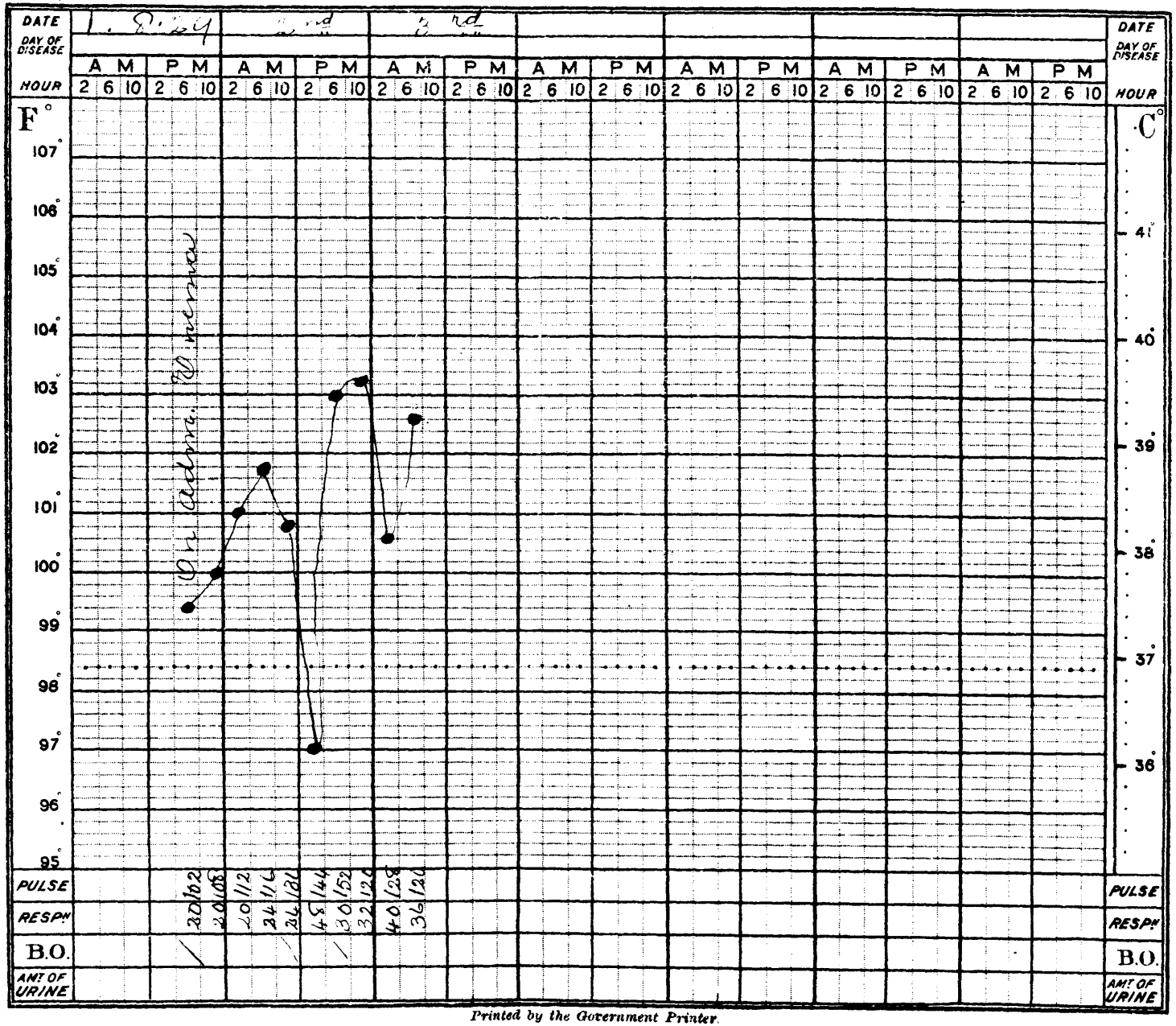
CLINICAL CHART.

Name Rodrick Williams Sex Male Age 15 Occupation School Boy

Diseases

Complications

Notes of Case.



Printed by the Government Printer.

HOUR CHART.

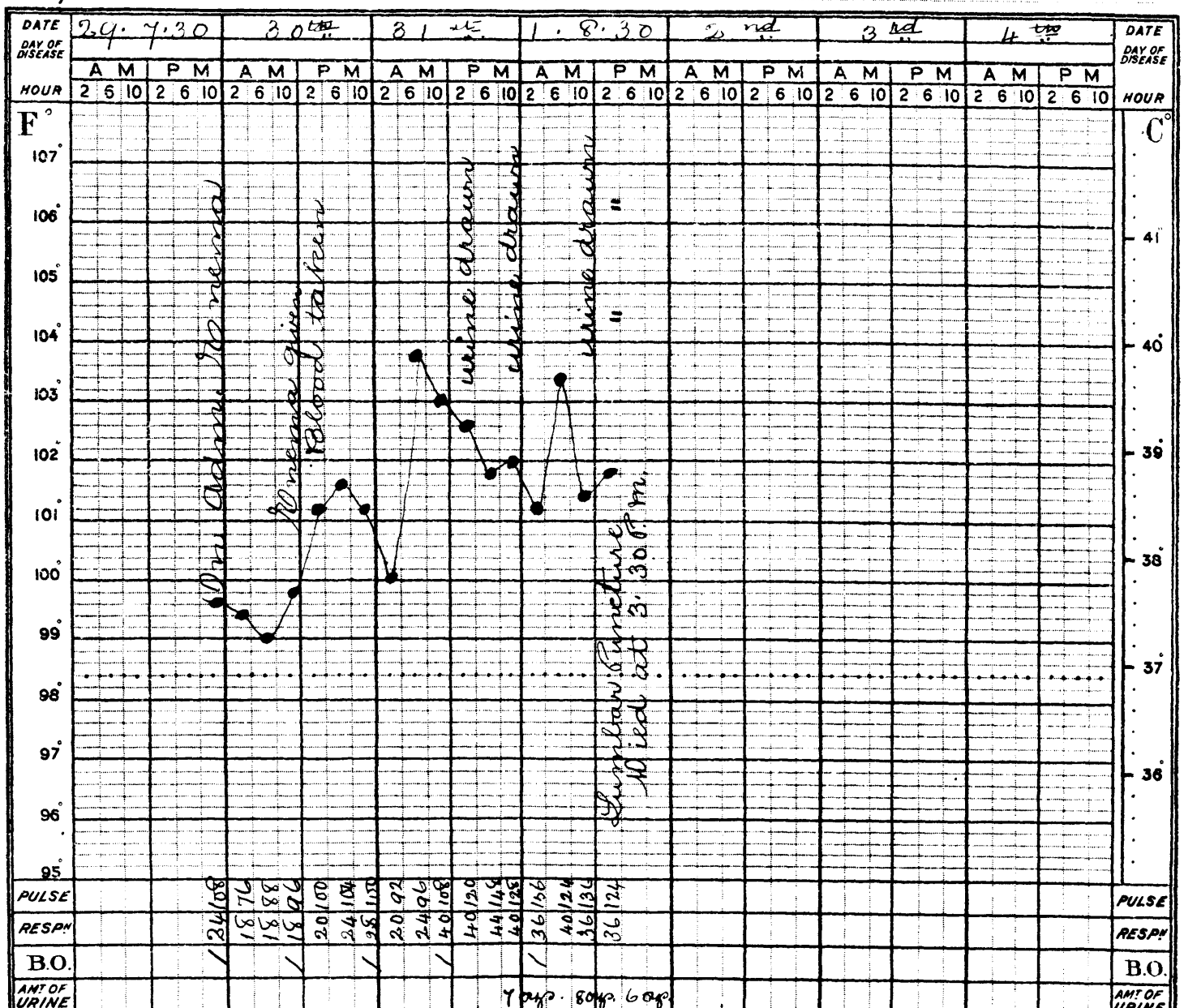
CLINICAL CHART.

Name A. Bruce Sex Man Age 15 Occupation School Boy

Diseases

Complications

Notes of Case.



Printed by the Government Printer.

HOUR CHART.

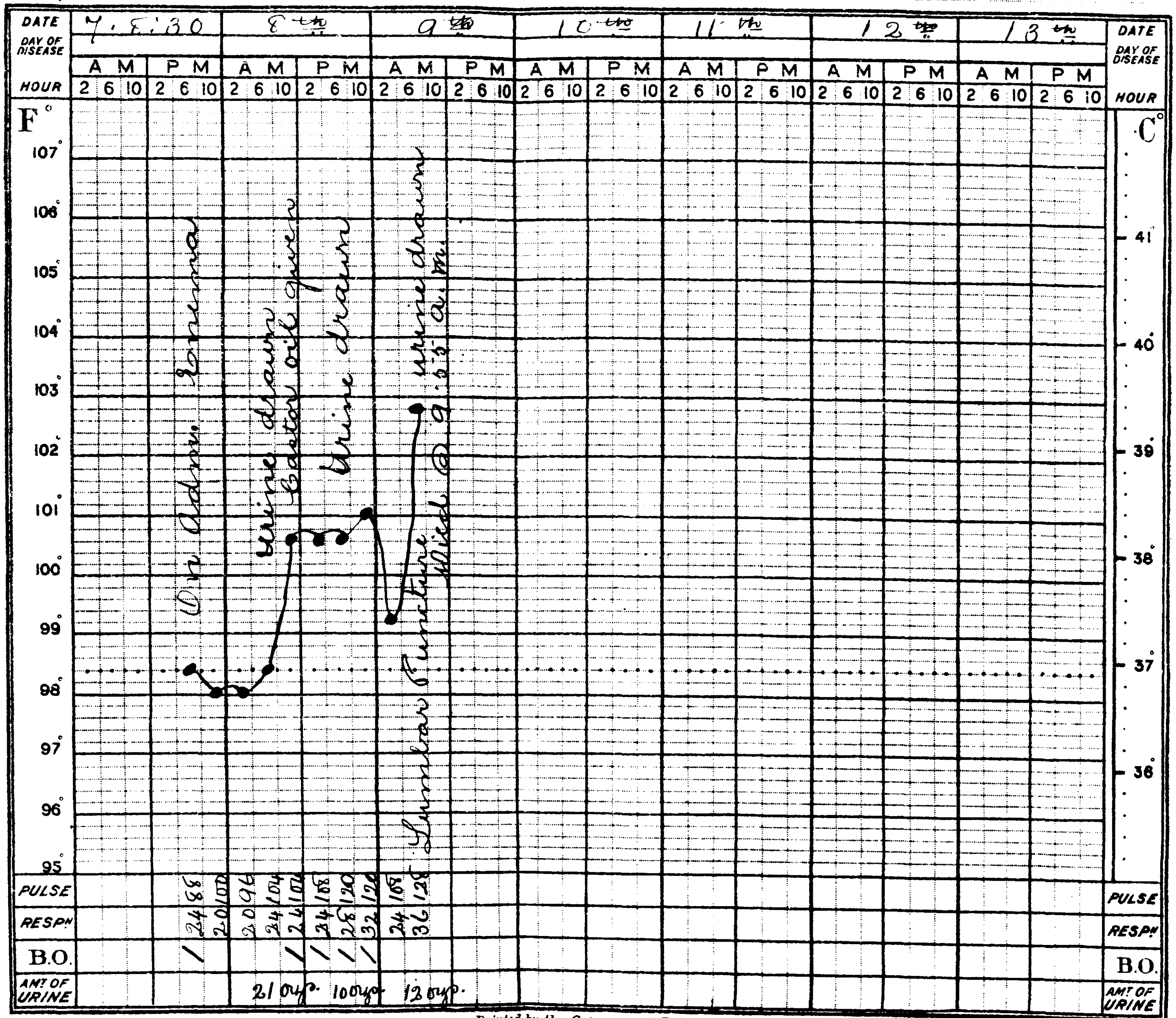
CLINICAL CHART.

Name Josiph Gooding Sex Male Age 45 Occupation Labourer

Diseases

Complications

Notes of Case.



Printed by the Government Printer.

4 HOUR CHART.

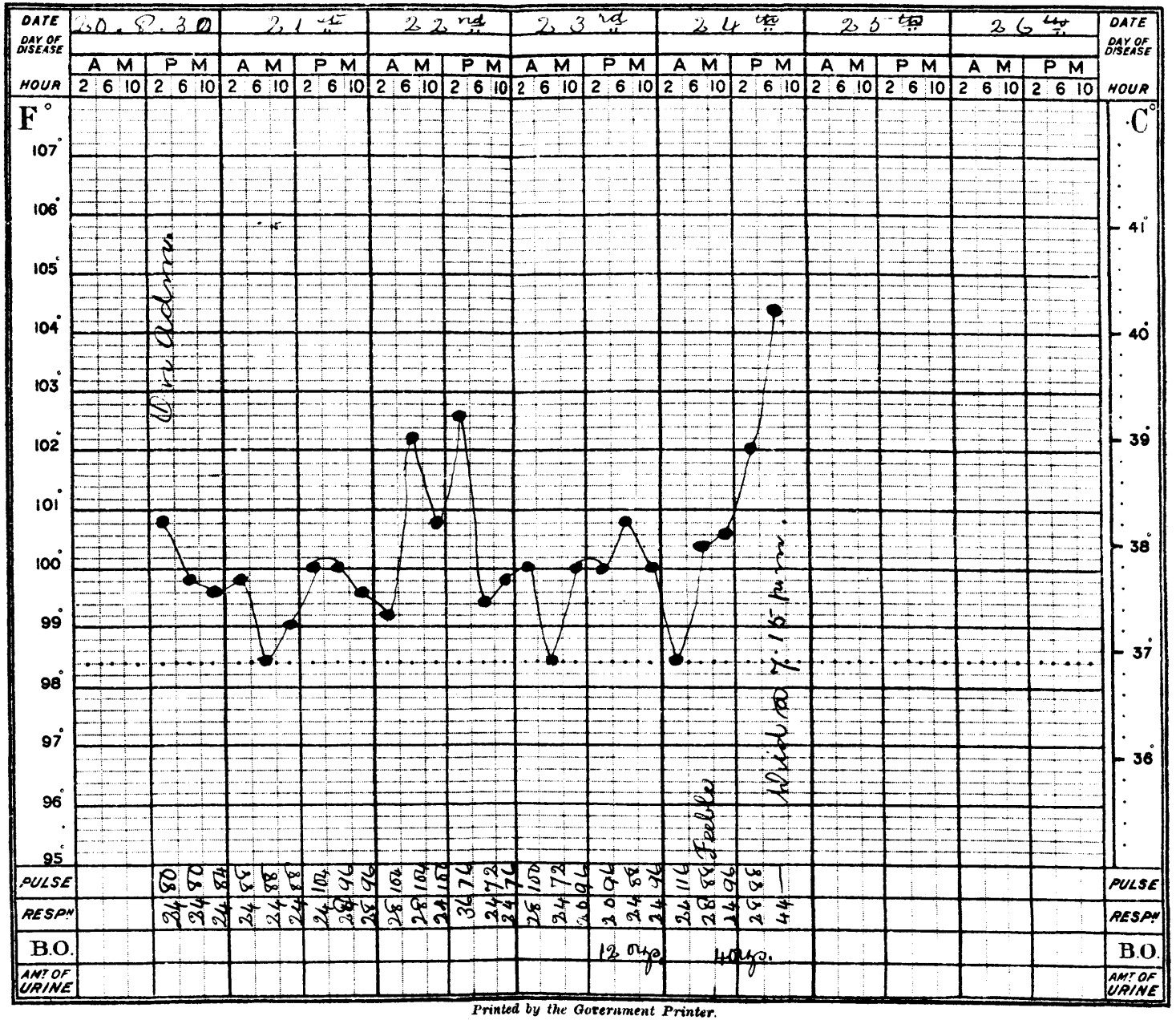
CLINICAL CHART.

Name Alice Phillips Sex Female Age 38 Occupation Labourer

Diseases

Complications

Notes of Case.



Printed by the Government Printer.

C

THE INFERIOR MESENTERIC GANGLION AS A REFLEX CONTROLLER OF THE BLADDER

BY

James Arnold Waterman, M.B. Ch.B. (Glasgow) Gov't. Medical Officer, Trinidad.

----------oooooo----------

The material contained in this paper has been derived in part from clinical and pathological observations on some rare cases of Acute Ascending "Rabic" Myelitis, a few of which I publish below.

I hope to bring forward evidence to prove that the Inferior Mesenteric Ganglion can act as a reflex centre. It is generally stated in books on physiology that a lesion in the Lumbar enlargement of the cord produces paralysis of the sphincter, and, therefore, incontinence of urine. In my series of cases, the whole Lumbar and lower dorsal regions of the Cord were destroyed by inflammation caused by the virus of Rabies, and in spite of this, the patients had Retention of Urine.

A very brief account of the embryology, anatomy and physiology of the Bladder and Sympathetic Nervous System will, I hope, be forgiven.

EMBRYOLOGICAL FORMATION OF THE BLADDER:

The Bladder with the exception of the Trigone and Sphincter is formed from the cloaca and its musculature originates immediately under the surface of the Entoderm. The trigone and sphincter are formed from the ends of the Wolffian ducts and the musculature originates immediately under the Ectoderm.

EMBRYOLOGICAL FORMATION OF THE SYMPATHETIC NERVOUS SYSTEM:

It is generally accepted by most embryologists that the ganglionic cells of the Sympathetic System are ectodermal in origin as the rest of the nervous system. These cells originate in the neural crest and then migrate in different directions where they aggregate to form ganglia. These cells should therefore possess the same functions as the anterior horn cells of the Cord.

NERVE SUPPLY OF THE BLADDER:

Motor Fibres are

1- Sympathetic to sphincter and trigone;

2- Parasympathetic to the wall of the bladder.

1. The Sympathetic:

The connector cells lie in Lumbar segments 1 and 2 and the connector or preganglionic fibres pass out in the Inferior Splanchnic nerves to the Inferior Mesenteric Ganglia. The excitor or post-ganglionic fibres arise here and pass in the hypogastric nerves to the bladder.

2. The Parasympathetic:

The connector cells lie in Sacral Segments 2 and 3, the connector fibres are found in the nervi erigentes and end in excitor cells in the bladder wall forming the pelvic plexus. The excitor fibres arise here and pass to the muscle of the bladder.

ACTION:

The SYMPATHETIC causes the sphincter to contract and the wall of the bladder to relax.

The PARASYMPATHETIC causes the wall of the bladder to contract and the sphincter to relax.

Sensory Fibres -
Sensory Fibres from the Bladder have been shown to pass up the hypogastric nerves, through the inferior Mesenteric Ganglia, inferior sp

splanchnic nerves, lateral sympathetic chain, white rami, and into the posterior nerve roots of Thoracic 12 Lumbar 1 and 2.

The cell stations for these afferent fibres lie in the corresponding posterior root ganglia".

The tone of the Bladder and its adaptation to its contents is controlled by a reflex:- the different impulses pass along the sensory fibres to the Cord in the Lumbar region and there the fibres synapse with the cells in the Lateral horn of grey matter and from here follow the path of the motor nerve to the bladder.

KUHNE'S EXPERIMENT:

It has been shown experimentally by Kuhne that impulses can be conducted along motor nerves in both directions.

SOKOWIN'S EXPERIMENT: or Bladder Axon Reflex.

Sokowin divided the Inferior Splanchnic nerve and so cut off all direct communication between the Inferior Mesenteric ganglion and the spinal cord. He then divided the left Hypogastric nerve and stimulated its central end and noticed:-

(a) Contraction of the trigone of the bladder on the right side;

(b) Contraction of the vesical sphincter;

(c) Ischaemia of the rectal mucosa.

These observations were interpreted by him to mean, that the Inferior Mesenteric Ganglion was able to function as a reflex centre. But Prof.Langley and his school, who do not accept the theory that sympathetic ganglia can act as reflex centres, considered the reaction as due to a preganglionic axon reflex depending entirely on the fibres of the Inferior Splanchnic. Langley's explanation is as follows:-

That each Inferior Splanchnic nerve gives off a collateral branch, which synapses with a ganglionic cell of the Inferior Mesenteric Ganglion, which in turn gives origin to the Right Hypogastric nerve. Therefore, stimulation of the central end of the fibres in the Left Hypogastric nerve resulted in the impulse passing upwards to the bifurcation of the axon and then down the collateral to the ganglionic cell of the Inferior Mesenteric Ganglion. From here along the post ganglionic

fibres or right hypogastric nerve to the bladder to bring about the reaction. Langley's explanation is based on Kuhne's experiment, namely, that "an apparent motor reflex can be obtained by the stimulation of a nerve which is itself motor".

EXPERIMENT:

SPINAL ANAESTHESIA:-

Before the injection of Novocain into the spinal column the bladder was filled with boric lotion. The anaesthesia produced was very successful for amputation of the leg below the knee. The operation lasted about forty five minutes. In both lower extremities there was motor paralysis and loss of all sensation. In the hypogastric area sensation was dulled.

At the end of the operation a catheter was passed into the bladder, but this evoked no sensation neither in the urethra nor in the bladder. The sphincter appeared normally contracted.

This proved, that although the paths along which sensory stimuli pass from the bladder to the cord and cerebrum (i.e. The 12 Lumbar 1 and 2) may be physiologically interrupted, and the nervi erigentes temporarily put out of action, yet, the sphincter vesical maintains its tone and remains contracted. Thus showing that the Bladder centre in the Lumbar enlargement is not the only controller of the sphincter vesicae.

It is a well known fact that during general anaesthesia the sphincter vesicae remains contracted unless too much chloroformhas been administered when the pupils dilate and become fixed and the sphincters relax pointing to paralysis of the sympathetics.

The sympathetics are more resistant to poisons than the C.N.S., for they supply organs which function continuously and are of vital importance to the organism.

POST MORTEM FINDINGS:

Post Mortem findings in these cases of Acute Ascending Rabic Myelitis were very characteristic, but the changes were more marked in some cases than in others.

1- Loss of demarcation between the grey and the white matter of the Cord;

2- The Cord in the Lumbar and lower dorsal regions was soft and often diffluent.

In short total destruction of the Cord and therefore complete physiological separation of the Inferior Mesenteric Ganglion from the Cord.

CLINICAL FEATURES:

Signs and symtoms noticed in these cases of Acute Ascending Myelitis chiefly in connection with the bladder :-

1- Often sensory disturbances of the foot appeared very early in the course of the disease and pointed to irritation of nerves originating in segments L.5; S.1; S.2.

2- Difficulty in micturating as shown by having to force to pass urine although there was no mechanical obstruction. This showed that the nervi erigentes were being poisoned at their centre of origin in the Cord. S.2; S.3.

3- Paralysis of the bladder with retention of urine - The nervi erigentes were obviously paralysed.

4- Loss of sensation in the penis, S.2; 3; 4, destroyed.

5- Severe burning pain over his bladder and hypogastrium and hyperaesthesia of the mucous membrane of bladder and abdominal wall were due, no doubt, to the toxin irritating the cells in the Cord at the level of 11th. and 12th. L.1.

6- Bladder sensibility was lost in the late stages of the disease. From this I concluded that the toxin had destroyed that part of the Cord through which the sensory fibres pass to get to the brain, namely, Th.11 & 12. L.1 and 2.

Now I hope we are in a position to answer the following question.

Why was there a tightly contracted sphincter causing retention of urine although the centre for the bladder in the Cord was destroyed ?

We know that the Inferior Mesenteric Ganglion was physiologically separated from the Cord which had become disorganised by the toxin of the virus of rabies. Therefore it was not due to a reflex through the Cord.

What are the other explanations?

(a) That it was due to an axon reflex as in Sokowin's experiment and Langley's interpretation.

I mention this only to dismiss it. For there is no experimental evidence - as far as I know - that sensory impulses arising normally from a distending bladder will pass up a motor nerve.

(b) That it was due to reflex action through the Inferior Mesenteric Ganglion, the modus operanid being as follows:-

The sensory impulses generated by a distending bladder pass up along the sensory nerve via the Inferior Mesenteric Ganglion where a collateral is given off. This hypothetical collateral completes with ganglion cells of the Inferior Mesenteric Ganglion a low reflex arc which controls the tone of the sphincter of the bladder. Be this as it may, I suggest that whenthe Cord is separated from the Inferior Mesenteric Ganglion, either temporarily as in anaesthesia or permanently, due to morbid changes, as in these cases of Acute Myelitis, the following changes occur :

The impulses passing up from the Bladder become shunted off along the collaterals which synapse with cells in the Inferior Mesenteric Ganglion, and so complete a true reflex arc.

A NEW CONCEPTION OF THE NERVOUS CONTROL OF THE BLADDER.

The Bladder may be said to be controlled by three centres:

(a) Inferior Mesenteric Ganglion keeps up the tone of the sphincter of the bladder by reflex action.

(b) The Cord normally reflexly controls:

1- the relaxation of the wall of the bladder in order to adapt itself to the amount of urine present;

2- the action of the sphincter of the Bladder;

3- the contraction of the wall of the Bladder.

(c) The Cerebrum which is called upon to function when numerous and strong stimuli come from the bladder, as when there is an urgent desire to urinate, and micturition is inconvenient at the time.

Clinical evidence in support of the Mesenteric Ganglion (sympathetics) being a reflex controller - subordinate to the cord - of the vesical sphincter will be dealt with shortly.

A complete transverse lesion of the cord:-

During the War, injuries occurred in which the Spinal cord was completely severed resulting in a stage of "spinal shock" which may be divided into two phases:- " A." This is associated with flaccid paralysis, absence of of reflexes, loss of sensation, and retention of urine and faeces.

Explanation: As a result of the injury ~~of~~ the cord is thrown "out of action", leaving the sympathetics, masters of the situation with power to cause contraction of the sphincter of the bladder reflexly.

" B." This stage begins to pass off in from 7 to 10 days, and has disappeared as a rule, in about three weeks. It is followed by a return of reflex ~~action~~ function in the portion of the cord distal to the injury; the knee and ankle jerks return, and reflex emptying of the bladder and rectum takes place".

Explanation: The cord has been put back into gear - the nervi erigentes (S 3. S 4.) and ~~the~~ Th 12 L 1 and 2 are once more functioning and therefore the bladder empties reflexly.

The effect profuced on the bladder as the result of an injury to the lower end of the Spinal Cord.

S. J. - Male. Age 34. Stone Quarry Labourer. Admitted 12.7.32.

HISTORY: Of while climbing down a rope, he was struck on the head by a stone which rendered him unconscious as a result of which he fell down striking his back.

ON ADMISSION: He was conscious. He complained of a girdle pain about 3" below the umbilicus. Lower extremities were paralysed.

The only voluntary movements were slight reflexion at knee and hip joints. Knee jerks absent, no babinski.

Tactile and thermal and painful sensation in lower limbs dulled. Pain and tenderness at 1st lumbar vertebra.

"X" rays revealed a fracture dislocation of 1st Lumbar Vertebra -

What was the condition of the Bladder in this case ?

On 12.7.32. No urine was passed voluntarily - but there was a little overflow incontinence - Urine had to be drawn off.

On 13. 7. 32. He felt nothing on passing catheter up the penile urethra but on reaching the bladder he said that he felt a sensation of heaviness.

On 19. 7. 32. He began to pass his urine voluntarily that is, after a period of 6 days retention.

The explanation of the retention of urine is quite simple. The 1st Lumbar vertebra is at the level of the 2nd 3rd and 4th sacral segments, and therefore any injury to it may cause injury to 2. 3. 4 Sacral segments. Inasmuch as the muscular wall of the bladder is supplied by Sacral 3 and 4 paralysis of bladder wall will follow. But the motor supply of the sphincter which comes from Th 12. Lumbar 1 and 2, via the sympathetics, not being injured, the vesical sphincter remained contracted and thus produced retention.

M. F. Male Age 30. Admitted 29. 6. 32, at 1 p.m.

HISTORY: Of having fallen off a lorry while in motion.

ON ADMISSION:

He was unconscious. Respirations were slow, deep and stertorous. Pulse was slow. Pupils active and moderately dilated. There was some bleeding from a scalp wound and also from left ear. Very restless - Lumbar puncture revealed blood stained cerebro spinal fluid.
The Diagnosis was " fractured base of skull".

I am only concerned with the state of the bladder in such an injury, as books very seldom mention it.

The patient had to be catheterised daily until 7. 7. 32, that is for 8 days after the accident. From the 8th to the 10th 7. 32 he passed urine reflexly in bed.

Explanation:

The cord was put out of commission by concussion and perhaps small haemorrhages.

The Sympathetics - especially the inferior mesenteric ganglion having escaped injury therefore caused the tone of the sphincter to be maintained reflexly through the Inferior Mesenteric Ganglion and therefore retention of urine.

On 11. 7. 32, patient regained consciousness. Discharged three weeks afterwards.

PHYSIOLOGICAL AND ANATOMICAL EXPLANATIONS.

Of Clinical Conditions of the Bladder.

1- True Incontinence.

(a) Passive type - when the urine dribbles away continuously;

Lesions (1) Paresis of the muscular sphincter of bladder e.g., in cases in which the sphincter is overstretched as in dilatation of the female urethra.

(2) Very severe shock;

(3) In very deep anaesthesia.

(b) Active type of Reflex Incontinence - when the

bladder empties itself reflexly without the knowledge of the patient.

Lesion: Some injury to the spinal cord above the 10th Thoracic Vertebra.

Mechanism: The reflex takes place in the Lumbar enlargement.

(c) Overflow Incontinence:-
When the bladder is over-distended with urine and the sphincter urethrae becomes incompetent through mechanical pressure.

Lesion: (1) Obstructive as an enlarged prostate.

(2) Any lesion which causes retention.

2- Retention of Urine.

(a) When the muscular wall is paralysed.

Lesion: 1-Lesion affecting the 3rd and 4th sacral segments or paralysis of nervi erigentes.

Mechanism: The nerve supply of the sphincter vesicae being in tact it s tone is kept up. Reflex action through the cord is abolished because the nervi erigentes are paralysed.

The reflex through the Inferior Mesenteric ganglion is in tact and therefore the sphincter is kept tonically contracted.

2-Lesion affecting the lower end of cord from 10th Dorsal segment e.g. Rabies.

Mechanism:
The reflex through the cord is abolished and one might expect incontinence of urine for the nerves coming from the cord are paralysed.

The reflex through the Inferior Mesenteric ganglion being still in tact we get the tone of the sphincter maintained and even increased causing contraction of the sphincter and so retention of urine.

3- Concussion of the Cord or even shock may put the cord out of action and so cause retention. In these cases of retention of urine, the patients have no desire to micturate and no pain is experienced.

(b) Mechanical Causes:-

(i) Enlarged prostate

(ii) Stricture urethrae

In these casesthe patients attempt tp pass urine and suffer much pain.

In Conclusion, I may state that -

1- Embryologically - the Inferior Mesenteric Ganglia represent masses of cells which have migrated from the Central Nervous System, and it is only reasonable to assume that they still retain the same potenialties and can perform the same functions as those cells.

2- Anatomically- the sensory fibres from the bladder pass through the Inferior Mesenteric Ganglion and we should expect nature to make use of the ganglion cells there, in order to complete a low reflex arc.

3- Physiologically - Anerback's and Meissner's plexuses are recognised as local nerve centres capable of initiating reflex action independently of the Central Nervous System. Inasmuch as the Sympathetic Ganglia correspond to these plexuses of the Parasympathetic system, their actions should be similar.

4- Experimentally - Sokowin's experiment and simple explanation appears to be substantiated by my clinical findings.

5- Clinically and Pathologically - the explanation that theInferior Mesenteric Ganglion can act as a true reflex arc station appears the most reasonable in order to account for the retention of urine, although the Cord was physiologically out of action on account of the morbid changes.

References :

1- Applied Physiology. S. Wright 1931.

2- Visceral Disease. Pottenger 1925.