Reports and Commentaries on Medical Cases Taken in Wards 93-4-24 by permission of Dr. Fleming Nightman, Rege in Clinical Medicine 1922.
I.

A CASE OF ENCEPHALITIS LETHARGICA.
Charles S.  Aged 36.  Married.

Occupation.  Joiner.

Complaint.  Muscular weakness and double vision.

Duration.  Three months.

**History of Present illness.**

During the past few years the patient has noticed that he was becoming more and more easily tired by any exertion or strain to which he was subjected, but this weakness has never been sufficient to lay him up from work.

In January of the present year this weakness became still more marked - so patient states. On the 17th of that month patient indulged in more alcohol than he had been accustomed to, and the effects of this caused him to retire to bed early.

From this time patient knows little about his illness. He is told that he became delirious and unmanageable, also that the Doctor who was summoned gave him an injection of morphia.

When he came to himself he was very weak and confused and also complained of double vision.

On February 2nd he was admitted to the R.I.E. still feeling very weak and confused.
Three days after admission patient became noticeably lethargic and his mental activity began to deteriorate. This lethargy steadily increased in spite of treatment and at the beginning of March patient could not be roused, though he was able to swallow when fed. His breathing became stertorous and at this time he developed incontinence of his bladder.

This state continued until the end of March when the patient began to take slightly more interest in his surroundings and by now he would sit up to be fed. Since this date his condition has been slowly improving, the lethargy becoming less pronounced and his intelligence and mentality more active.

At the present time though he is still lethargic, his intelligence is fairly good. His speech however is still very slurred and indistinct.

Previous Illnesses.

Patient cannot remember any particular disease of childhood. In 1901 he had a slight attack of smallpox but he recovered completely. Also chicken-pox when he was young. He says he has had no other illnesses. He has had several small accidents while at work, including one rather severe blow over the left eye. He has suffered no after effects from any of them.

Surroundings./
Surroundings.

At home patient's surroundings are entirely satisfactory, patient having an attentive wife and comfortable house. He has no family.

His work also is not very arduous and patient's surroundings here are satisfactory. He is a joiner.

Habits.

Food. Patient has not a heavy appetite but takes his food well. His food is of a plain and ordinary character. He has meals at the usual times.

Drink. Patient states that he is a moderate drinker, usually not taking very much as it leaves him very weak.

Tobacco. Patient smokes about 3 oz. per week - cigarettes and tobacco.

Family History.

Patient's parents are both dead, each having died at the age of about 60. Cause is unknown in either case.

Three sisters are alive and well. None dead.

One brother died in infancy from an unknown cause.

Patient's wife is in poor health and suffers from "Tuberculosis". Patient has no family.
State on Examination.

28.4.21. Patient has a drowsy and lethargic appearance as if ready to drop off to sleep at any moment. A drooping of his right eyelid furthers this appearance. When spoken to however, his intelligence is found to be under average, and a defective memory and difficulty of speech embarrass him considerably.

Height. 5 ft. 7 ins.

Weight. Now is 8 st. 2½ lbs. On admission was 8 st. 12 lbs.

Development is good - the tone of his muscles however being slightly increased.

His attitude is one of indifference to his surroundings and he lies with his eyes closed unless spoken to.

The only obvious morbid appearances are a drooping of the right eyelid and lateral strabismus of the right eye.

A scar over his left eye is seen from his injury there.

Temperature 98°. (on admission was 99.2°)

SYSTEMATIC EXAMINATION.

NERVOUS SYSTEM.

Intellectual functions. (Patient is right-handed)

Patient's general intelligence is under average and the patient frequently makes contradictory statements.
His emotional state is one of drowsiness and indifference to his surroundings.

His memory is very poor. The memory of recent events is almost negative, especially of events of the last three months. The memory of older occurrences is also impaired but not so much.

Patient's speech is that of a man who is almost asleep. The power of producing speech is unimpaired but the power of distinct articulation is very much diminished. The syllables are slurred together as in a state of intoxication and the speech is almost incomprehensible owing to slurring of dentals and labials. There is however no stammering, lalling or dysarthria.

There is no word deafness and patient can name common objects. The power of written speech is unimpaired.

At the time of examination there is no delirium, no delusions or hallucinations.

**GRANIAL NERVES.**

1st Olfactory.

The power of recognition of volatile substances is not impaired but the distance at which an odour can be appreciated is slightly below normal.

2nd Optic./
2nd Optic.

Visional acuity was tested with Snellen's types. Each eye was found to be deficient, the right more than the left.
Right Eye \( \frac{6}{24} \). Left Eye \( \frac{6}{18} \).

Extent of field of Vision. In the left eye the field of vision was normal except for a slight diminution at the nasal side.
In the right eye the field of vision is diminished at the nasal side and to a slight extent below. There is no central scotoma in either eye.

Colour sense is good and patient can recognise and match all colours which he is shown and there is no central colour scotoma.

Both eyes are presbyopic, the right being more so than the left.

The intra-ocular tension as judged by the finger is normal in both eyes.

There are no subjective visual sensations such as specks or lines in front of the eye.
Also no hallucinations of sight.

3rd Oculomotor.

There is marked Ptosis of the Right eyelid, the Left eyelid being unaffected.

Paralytic strabismus is present in the Right eye, observable when the patient follows the finger to the left side, the right eye/
eye being unable to accompany the left past the middle line and also being unable to converge.

Diplopia is present, due to the right eye and is greatest towards the left side of the field of vision. It is horizontal in character and there is no vertical diplopia.

Nystagmus could not be elicited in the right eye, but a slow rhythmical horizontal nystagmus was brought out in the left eye.

**Pupils.** These are equal in size and shape. The right pupil reacts sluggishly to light - the left normally.
The right pupil also reacts sluggishly to accommodation, while the left reacts normally.

**Movements.** The left eyeball moves freely in all directions.
The right is restricted in its movement and cannot move beyond the median plane towards the left side, otherwise movement is unrestricted in all directions.

4th *Trochlear.*
Is quite unaffected, the Superior oblique muscle functioning perfectly.

6th *Abduces.*
The lateral Rectus Muscle is unaffected.

5th *Trigeminal.*
The motor and sensory divisions are unaffected,
the Pterygoid, Temporal and Masseter muscles being strong and functioning.

Also light touch, pain, heat and cold being fairly well appreciated over the face, any mistakes being due to patient's sleepy condition, not to affection of his sensory nerves.

Sensation and taste are unaffected in the mouth, patient being able to discriminate between various tastes.

7th Facial.

Facial muscles supplied by the 7th nerve are unaffected.

Patient has no hyperacousis.

Taste in anterior \( \frac{2}{3} \) of Tongue is good.

8th Acoustic.

Patient is deaf in both ears, more so in the right than the left. A watch could be heard ticking by him at a distance of 6" from his right ear and 9" from his left, when heard 36" by a normal ear.

Weber's test indicated middle ear obstruction in both ears. Rinne's test was referred to the right ear.

Patient has a small amount of wax in both ears.

He states that he has not any ringing in the ears, nor has had since his illness commenced.

He has no vertigo at the date of examination but had attacks of giddiness at the commencement of his illness.

He/
He states that he has never had any discharge from either ear.

**9th Glossopharyngeal.**

Is unaffected: no disturbance of taste in the posterior \( \frac{1}{3} \) of tongue and no dysphagia and sensation is present in the pharynx.

**10th Vagus.**

**11th Accessory.**  are all unaffected.

**12th Hypoglossal.**

**Cervical Sympathetic.**

This is unaffected. The Pupils dilate to shade. There is no Enophthalmos or Exophthalmus and flushing or sweating of the neck or face.

**MOTOR FUNCTIONS.**

**Intention Tremors** are present, becoming more marked when patient carries out an action against resistance. Patient has difficulty in touching his nose with his finger and also in touching the fingers of each hand with his eyes closed.

**Tremors.** A slight tremor of each hand is observable when his are is stretched out, becoming very marked when patient attempts to write.
A slight tremor is present in the tongue when protruded.

A distinct lateral tremor of the whole head is present, brought out better when patient closes his eyes. When patient stands with his eyes shut he is very unsteady but can keep erect on two feet. He is unable to stand on one foot. Patient's gait is spastic in type and he drags each foot after the other. The foot is everted and the toes shuffle over the floor owing to imperfect flexion of the foot.

Patient is prevented from writing clearly by the shakiness of his hand.

**REFLEXES.**

**Superficial.**

The Conjunctival and Pharyngeal reflexes are present and unaffected.

The Abdominal reflex was brought out with difficulty on the left side, but could not be elicited on the right side. Babinski's sign. On the left side this was definitely a flexor response: on the right side the result was indefinite. Patient's soles are covered with thick skin, very slightly sensitive.

**Deep Reflexes.**

The Triceps, Biceps and Supinator reflexes are equally augmented in both arms.
Both knee jerks are equally increased and patella clonus was brought out in each leg.

The Abductor response on each side is augmented and a crossed response is well marked.

Ankle-clonus is not marked but is present on both sides.

**Organic Reflexes.**

At the time of examination there is no interference with defecation or micturition. An incontinence of urine which developed in the course of patient's illness was apparently due to his sleepy condition and his inattention to the calls of Nature.

**SENSORY FUNCTIONS.**

**Subjective Sensations.**

Patient complains of pain in his back, due to his lying on it for a long period.

He has no other subjective sensations. At the commencement of his illness he had headaches but he has none at the time of examination.

**Objective Sensations.**

Patient's sensations with regard to light touch are unaffected in any part of the body.
His discrimination between heat and cold was fairly good; a few mistakes being made in different areas owing to his sleepy condition.

His discrimination between blunt and sharp objects is unaffected in any part of the body.

Patient is able to localize points of touch perfectly and can discriminate sharp points when close together.

His vibration sense is fair. (Tuning fork)

Joint sense not good.

**THE CEREBROSPINAL FLUID.**

A Lumbar puncture was performed on the 4th March 1921. The Cerebrospinal fluid was clear and was not under pressure.

Pathologist's report.-

"Numerous Red Blood Corpuscles.
Relatively large proportion of Lymphocytes.
A few polymorphs.
No organisms seen in direct film and no growth on culture."

**WASSERMANN REACTION.**

This was tested on two occasions (21.2.21 and 7.3.21) and found to be negative on both occasions (Blood & C.S. Fluid)

**VASOMOTOR FUNCTIONS.**

Are unaffected.

**TROPHIC FUNCTIONS.**

Are unimpaired.
OTHER SYSTEMS.

Alimentary System.
There is nothing to note about the Alimentary System.
Patient's teeth have all been extracted except one, and replaced by false ones.
Patient states that he has never had nausea or vomiting during the period of his illness.
Bowel are slightly constipated occasionally needing aperients. Abdomen examination reveals nothing of importance. no retraction - no tenderness - organs of normal size.

Circulatory System.
There are no subjective phenomena.
Pulse. Arterial wall not thickened and pulse is regular in rate and rhythm and is well sustained.
Arterial pressure as estimated by finger - rather low.
As estimated by the Sphygmomanometer Systolic - 107) mm. Diastolic - 87)
Heart. Is normal in size, shape and action.
The sounds are distinct and closed.
A small scar over the apex was due to a burn.
There is no pulsation over the chest or neck.

Respiratory/
Respiratory System.

There are no subjective symptoms: no cough, pain or dyspnoea.

Breathing partly abdominal and partly thoracic.

Lungs are perfectly healthy and breath sounds normal vesicular.

Urinary System.

No subjective phenomena.

Incontinence of the bladder as already mentioned.


No deposits or abnormal contents.

Reproductive System  there is nothing to note.

Integumentary System.  no rash.

Locomotory System.

The muscles are fairly well developed and equally strong on both sides.

There is no rigidity but the tone of the muscles, especially of the arm, is slightly increased.

There is no cramp, contractures or twitchings.

Bones and joints are unaffected.
PROVISIONAL DIAGNOSIS.

ENCEPHALITIS LETHARGICA.

TREATMENT.

Diet. Milk while extremely lethargic.
Now light diet.

Medicinal. Tinct. Card. Co. 3 ss T.I.D.
Urotropine gr X. T.I.D.

Bathing with Methylated Spirits to prevent Bedsores.

Progress

3. 3. 21 The patient has become more and more
drowsy and today cannot be roused.

4. 3. 21 A Lumbar Puncture was done - fluid not
under tension (see report of pathology).
Breathing is laboured at times.

Incontinence of urine developed.

24. 3. 21 At all times patient has been able to swallow.
Patient is beginning to take more interest in
his surroundings.

29. 3. 21 A second Lumbar Puncture done - very low pressure.
Repeat similar to first.

10. 4. 21 Patient is gradually improving mentally and
the paralytic symptoms also are improving.
From this date improvement was very slow almost
unnoticeable and finally patient was transferred to
Every Lockhart Poorhouse - the paralytic symptoms still
evident. The mental state still dull.
COMMENTARY.

INTRODUCTION.

By way of definition Encephalitis Lethargica may be described as a general infective disease which is characterised by certain manifestations originating in the central nervous system, of which the most frequent and characteristic are a progressive lethargy or stupor and a lesion in or about the third pair of cranial nuclei.

Although the disease in its present day conception may truly be called a "new" disease, it is probable that it has existed for many years although unrecognised. As far back as 1841 Marshall Hall had recognised certain conditions of inflammation of the cerebral substance and had differentiated these from the conditions due to inflammation of the meninges (Marshall Hall - Diseases and derangements of the Central Nervous System 1841).

But it was not until 1881 that Wernicke described cases of encephalitis of sudden onset and characterised by an equally sudden ophthalmoplegia and, pathologically, by an acute haemorrhagic inflammation in the floor of the 3rd and 4th ventricles, (Wernicke - Lehrbuch der Gehirnkrankeiten 1881) which he compared with poliomyelitis and called Poliencephalitis Superior. His views were regarded with favour and in the succeeding/
succeeding years much confirmatory work was done by various observers, the most prominent of whom were Strümpell (1884), Meidin (1890), Gowers (1893), McEwen (1893) and Murawieff (1897). Lack of material caused the advance in knowledge to slow down until in 1917 von Economo directed attention to the "new disease" in Vienna and called it Encephalitis Lethargica (Von Economo - Wien. klin. Wehnschr. 1917) and he was followed by Netter of Paris (1918) and Harris and Hall in England in the same year (Lancet p. 568, April 20, 1918). From that date up to the present time much investigation has been carried out on the subject and the modern conception of the disease as illustrated by this case was clearly defined and many of the mysteries of pathology and etiology unravelled.

In the course of the investigation many names have been given to the disease but that of Encephalitis Lethargica would seem to be most appropriate in the present state of our knowledge as it indicates the pathology and most striking clinical feature of the disease.

**GENERAL AETIOLOGICAL CONSIDERATION.**

On the whole the case of the patient under discussion may be taken as typical of the condition and the lack of any definite/
definite cause in him illustrates the paucity of our knowledge concerning the disease from which he suffers.

With regard to sex and age incidence - males and females are attacked in equal proportions and our patient at the age of 36 is at the age typical for the onset of the condition. The disease is rare in childhood though cases are reported of quite young children being affected.

Seasonally again patient is typical. This illness commenced in the early part of the year and was at its height during the late Winter and this has been the case in the vast majority of cases. This point is interesting because the disease has been connected with Poliomyelitis Acuta by some observers and the seasonal influence of this is totally different - namely during the summer months.

As was to be expected a specific pathogenic virus was sought for and over this subject much controversy has raged - in 1917 von Weisner reported that he had transmitted the disease to monkeys, (von Weisner - Wein. Klin. Wehnschr. 1917; 933) and he also cultivated a Gram and diplo-streptococcus from the Central Nervous System of patients dying from the disease. His findings have been disputed by Netter and Kinnier Wilson (Lancet - July 6th 1918) and many others. Marinesco in the report on the disease to the Local Government Board (1918) adopts a conservative attitude with a leaning towards/
towards von Wiesner's views and suggests that there may be a filtrable virus similar in nature to that causing Poliomyelitis Acuta, gaining entrance by the mouth or nasopharynx and thence to the Central Nervous System - e.g. through the Cribriform plate of the Ethmoid.

Clinical evidence is decidedly negative as regards any help on this point. The patient under discussion comes from a district where no case has previously been reported and has never been in contact with a patient suffering from the disease. Bramwell in a report on the subject states that he has never seen two cases in the same family and has never yet had any evidence of direct transmission. Other reporters have quoted cases of the majority of the inmates of an institution being infected. (Report of the Chief Medical Officer 1919-20. p.357)

There is only one way of regarding these contrary views upon the subject, and that is to assume that many people harbour the organism as a harmless saprophyte, for example in the nasopharynx and that in certain persons for some reason at present unknown, an enhanced virulence of the organism or a diminished resistance of the body tissues results in the disease being produced.

Now/
Now the means of lowering the body vitality are numerous and among these, previous illness and high living stand prominent. In our patient previous illness can be excluded but the patient's habits may shed some light on the subject and here the patient's own statements must be taken as facts. He states that he is a moderate drinker and this his wife confirms; also that taking alcohol leaves him very weak. In addition he says that the night before his illness commenced he indulged in a large quantity of whisky in the presence of some friends, and from this time he knew very little more about the course of his illness.

Can these facts be connected up? The question must be left at that.

It should be mentioned that no organisms were found by the Pathologist in his examination of the cerebrospinal fluid.

THE DIAGNOSIS OF THE CASE.
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And now one must deal with the proof that this case is one of Encephalitis Lethargica and not one of the numerous and more common diseases which it so closely resembles - in other words the diagnosis must be established - this being the label attached which summarises best in accordance with present day knowledge, the history, manifestations, and the/
The elevation of the temperature during the first week in hospital (1st to 7th of the condition)
the course of the deviation from normal which the patient exhibits.

With regard to history Encephalitis Lethargica usually commences somewhat acutely with an attack of dizziness or fainting followed by mental confusion and a slight elevation of the temperature and here our patient is quite typical for in spite of his confused story it is clear that his illness commenced exactly in this way, and for some days after his admission to hospital he still had a temperature of above 99°F. and this was three weeks after the commencement.

Then after a varying prodromal period the typical manifestations of the disease develop; the muscular weakness becoming accentuated and the patient becoming somnolent and lethargic. The temperature now falls and may become subnormal and the pulse previously fast, falls to normal and is of poor tension. The patient lies on his back - his face expressionless and he is too weak or too drowsy to turn or make any voluntary movement.

Then the lethargy develops into stupor, even coma. And on glancing over the case of this patient one is struck by the fact that it is so typical as to be almost pathognomonic, but if further confirmation were wanted it is found on attempting to rouse the patient and listening to his slurred,
slurred, slow, monotonous speech, seeing his drowsy appearance, his dreamy smile, the drooping of his eyelid on the right side and the paralytic strabismus on the same side.

This is indeed a typical case of lethargic encephalitis for, although no single manifestation is pathognomonic when they are grouped together, they could hardly be construed otherwise.

DIFFERENTIAL DIAGNOSIS.

There are however several conditions which must be differentiated before this somewhat rare disease is diagnosed. Most of them can be dismissed in a word.

1. Poliomyelitis Acuta. In its typical form this presents no difficulties. Occasionally however cerebral symptoms are prominent and are very similar to those described above. However they are of sudden onset, briefer duration and speech changes are uncommon. The age of the patient, - the fact that there are no associated spinal cases in the same family certainly exclude the condition with the additional fact that the Cerebrospinal fluid of Polio-encephalitis shows many lymphocytes and few polymorphs, while the reverse is the case in the present patient.

2./
2. **Disseminated Sclerosis.** Before the lethargy became well marked the case was diagnosed as disseminated sclerosis owing to the intention tremor, spastic gait, and exaggerated reflexes. The profound lethargy which developed however differentiated the case.

3. **Cerebral Syphilis.** The Wassermann reaction of the blood and cerebrospinal fluid were negative and no other signs of syphilis can be found so this condition can be excluded.

4. **Tuberculous Meningitis.** The patient shows no evidence of Tuberculosis in his lungs or elsewhere, and no Tubercle Bacilli were found in the Cerebrospinal fluid. The ocular squint and the coma of Tuberculous Meningitis were the stumbling block but now that the patient is well on the way to recovery this can be differentiated.

5. **Botulism.** There is no history of a common source of food poisoning in this case and the patient's family who had eaten the same food as he showed no symptoms.

Other conditions which might be possible sources of difficulty but may be excluded owing to absence of typical signs - thus Cerebral Tumour (lack of vomiting), Typhoid (normal spleen), Uraemia (no oedema) and Cerebrospinal Meningitis (Cerebrospinal fluid) can be dismissed.
GENERAL RELATIONS IN THE MIDBRAIN AT THE LEVEL OF THE SUPERIOR QUADRIGEMINAL BODY
From anatomical observations in this case one can conclude that the affection under discussion is dependant upon an encephalitis which has shown a special predilection for the brain stem, and as the symptoms of this case are so typical and the condition of the Cerebrospinal fluid is also characteristic one is justified in assuming that the pathological lesion in this case is similar to that found in the many cases which have now been examined. For the purpose of completeness these may now be briefly mentioned.

**Macroscopically** it is the exception for any constant change to be found (McIntosh - Reports to the Local Government Board 121) and beyond some congestion of the vessels more especially in the region of the pons and thalamic region, together with an increase of cerebrospinal fluid, very little else can be found with the naked eye.

**Histologically.** - in all cases examined the presence of definite inflammatory lesions can be demonstrated, consisting of a cellular infiltration of the perivascular lymphatic sheaths and of certain areas of grey matter. The meninges escape damage or are only slightly involved. In the brain substance the changes have usually been found in the upper part of the pons and the basal nuclei. The infiltration consists mainly of plasma cells and large and small lymphocytes.

Perivascular/
Perivascular haemorrhages are often found around the aqueduct.

Armed with this knowledge one can say with a degree of certainty that in this patient the lesion is of a similar nature and that the infiltration has been more especially around the nucleus of the oculomotor nerve. The changes in the cerebrospinal fluid confirm the nature of the infiltration.

Bacteriologically it has been mentioned that some observers have demonstrated a diplo-streptococcus. These findings however lack confirmation and so will not be commented upon. In this patient no organisms were isolated by the pathologist.

THE SYMPTOMATOLOGY AND ITS EXPLANATION.

1. The Lethargic state.

Sleep is a condition which occurs when the afferent paths to the sensorium are inhibited and results if they are inhibited centrally or peripherally - the former being illustrated by opium narcosis and the latter by the effect of darkness in producing sleep.

Now the course of the sensory fibres passes through the crus cerebri close to the nucleus of the oculomotor nerve and thus a lesion situated in and around this nucleus is very liable to involve the sensory tract. This is all the more certain/
EXPLANATION OF THE MAIN SYMPTOMS

Sections through MIB BRAIN at level of 3rd Cranial Nucleus.

AQUEDUCT
OCULOMOTOR NUCLEUS
POSTERIOR LONGITUDINAL BUNDLE
MEDIAL LEMNISCUS
RED NUCLEUS
FIRES OF OCULOMOTOR NERVE

BASIS PLEUNCULI

I

The Immediate Relations.

Involving Post. Longitudinal Bundle causing nystagmus.

II

Involving Medial Lemniscus i.e. Sensory Fibres causing lethargy.

III

Involving Red Nucleus causing
Rhythmic Tremors & Immobile 5 dec.
certain when it is known that some of the fibres of the oculomotor nerve pass upwards in close relation to the fibres conveying afferent impulses to the Thalamic region. Thus because of both these reasons sleep results or somnolence or lethargy if the block is not total as in the case of this patient.

2. The Ophthalmoplegia.

This is due to direct action on or around the nucleus of the 3rd Cranial Nerve, only certain fibres being affected, as indicated by the paralysis of the right Internal Rectus muscle and partial paralysis of the right Levator Palpebrae superioris. This also indicates that the lesion is on the right side of the brain. Thus the Ptosis, Diplopia and Lateral strabismus in the right eye are explained.

3. The immobile face and the rhythmical tremors which the patient showed, more especially in the early part of his illness were probably due to interference with the function of the red nucleus, or the rubrospinal tract. (See diagram)

4. The Nystagmus which the patient shows is due to interference with the posterior longitudinal bundle.

5. Patient's difficulty in gait and his giddiness are apparently cerebellar symptoms and are explained by the researches of Marinesco who discovered a lesion in the cells of Purkinje which would account for them.
PROGNOSIS.

At the best this can be a mere guess for our knowledge of the condition is so incomplete that it is impossible to say which cases are going to recover completely and which are not. In this case the prognosis as regards survival is good, because the lethargy is definitely diminishing and the symptoms are slowly decreasing. The likelihood of a remission is not great for though these have been reported they form the exception to the rule. In addition statistics show that cases with a localised paralysis, as this case has, carry with them a much better prognosis than those without localising nervous signs. And it is said that if the patient survives the first three weeks the strong probabilities are that he will recover (Bramwell). The time necessary for recovery varies enormously in different cases, and in this case it is undoubtedly going to be a long process.

With regard to after effects the prognosis must be more guarded and the following points must be kept in mind.

I. The Mental effects occasionally are permanent and the patient may be left in a condition resembling melancholia.

II. Cranial Paralysis - in 75% cases a certain amount of residual paralysis is remaining after three months and among these our patient must be classed. The permanency of this cannot yet be stated.

III./
III. Occasionally a paralysis apparently of Spinal Chord origin appears - but this is rare and usually occurs in a younger type of patient than this.

IV. Sometimes the syndrome of Paralysis Agitans appears after Encephalitis Lethargica. This however carried with it a better prognosis than is usually the case with this latter condition.

TREATMENT.

"No therapeutic measures have so far proved to have a definitely beneficial effect upon cases of Encephalitis Lethargica. A specific therapy has yet to be discovered". (Bramwell)

General treatment adopted to this case was of course carried out. Thus the patient was in bed and while stuporose was kept on a fluid diet and when the stupor diminished he was sustained on a light diet.

Medicinally hexamine was given in ten grain doses three times a day, for it is known that this is excreted in the Cerebrospinal fluid. But the effects of this were extremely doubtful and the reason is not far to seek.

To exert any antiseptic power hexamine must be converted into Formaldehyde by the presence of an acid medium and as/
as Cushny has pointed out this is not present in the Cerebrospinal fluid.

Other drugs which have been suggested are iodides and arsenic, for the changes found in certain cases of basal syphilis and sleeping sickness are certainly similar in some results and these drugs have been found useful for such cases. In encephalitis lethargica however the effect is again extremely doubtful. Strychnine may be of use in some cases.

The value of repeated lumbar puncture which has been advocated by some authorities is usually small.

Apart from these measures little could be done beyond relieving constipation and preventing bedsores and thus in summing up the treatment one may say that the most important points are, in the present state of our knowledge, careful nursing and the prevention of complications.

CONCLUSION.

In conclusion one can say that the diagnosis has in this case been clearly established as a case of encephalitis lethargica. A study of the case has not cleared up any of the mist in which the etiology of the condition is placed, beyond indicating that the possibility of organismal causation cannot/
cannot be dismissed.

The symptomatology is to be entirely explained in the light of the pathology - namely an inflammatory reaction around the cerebral peduncles and the upper part of the pons.

The prognosis in this case while being very good as regards survival, is guarded, from the point of view of complete recovery.

The treatment is merely palliative.

However if the enquiry into the subject has brought out any point it has revealed the paucity of our knowledge on many aspects of the case, and it is quite possible that further research on the subject may fundamentally alter many of the views which have been set down in this commentary.
II.

A CASE OF ANEURYSM OF THE TRANSVERSE PART OF

THE ARCH OF THE AORTA.

Occupation. A weigher of goods at Leith Docks.

Complaint. "Cough and shortness of breath."

Duration. Almost three years.

History of present illness.

Nearly three years ago in March 1917, patient first began to be troubled with a cough of a dry, hacking nature and bringing up little sputum. At this time also patient noticed that he easily became breathless with the slightest cause. The cough was treated and disappeared in a fortnight and for a few weeks patient was free from symptoms. However during the Summer the cough kept recurring and disappearing at intervals. The intervals between the attacks became shorter and the cough gradually worse until in November 1918 patient became so weak that he had to cease work, which was of rather a heavy nature, as a weighman at the docks.

After four weeks' rest patient was sufficiently recovered to return to work (December 1918). During the year 1919 the cough and feeling of weakness returned; during the later part of the year it became more severe than it had previously ever been. Now/
Now the cough was accompanied by a "tight" feeling across the chest, which was only relieved on bringing up some thick sputum.

During the first week of 1920 another severe attack laid him off work again, and the weakness was more alarming and accompanied by marked breathlessness even on slight exertion.

Patient was advised to come to the R.I.E. and was admitted on February 26th although his symptoms at this time were much easier.

**Previous Illnesses and Accidents.**

Patient cannot remember any particular illnesses of childhood, but thinks he had the usual infectious diseases of childhood.

In 1913 he had a severe attack of Erysipelas and was ill for 10 weeks. He recovered completely however and has had no recurrences of the attack.

A year later he fell off a staging while at work and landed on his back. Although bruised and shaken no bones were broken and patient was laid up only a short time.

Patient gives a history of Syphilis.

**Surroundings.**
Surroundings.

At home patient's surroundings appear to be quite good and patient states that he has a very comfortable home.

At work. Patient's work is of an arduous nature at times, he being employed as a weighman. He has to carry many heavy sacks and states that it is very dusty and that he frequently inhaled the grain or dust from the sacks which he is carrying.

Habits.

Food. Patient has a good appetite and eats plain food at regular intervals.

Drink. He is a moderate drinker and has never drunk to excess.

Tobacco. Patient is also a moderate smoker, smoking one to two ounces of tobacco per week.

Family History.

Patient's parents are dead, his father dying at the age of 53 of "heart failure" and his mother at the age of 79 of "old age".

His brothers and sisters are all alive and well.

His wife is alive and healthy.

He has four children - all alive and well.

There is however a history of several miscarriages.
State on Examination.

Patient's intelligence is average.

Height 5' 9".

Weight (on admission) 11 stone 7 lbs.

His development is good and his muscularity above average.

With regard to his general appearance patient looks cheerful, has a high colour and a malar flush.

He has no morbid appearance and bears no evidence of previous disease.

His temperature is 97°.

SYSTEMATIC EXAMINATION.

CIRCULATORY SYSTEM.

Subjective Symptoms.

Patient has no pain but sometimes experiences a feeling of tightness across the chest.

On exertion patient suffers from marked palpitation but has no feeling of faintness.

He also complains of dyspnoea which is not troubling him at the time of examination.

His cough is of a dry, hacking character and has a distinctly/
distinctly brassy sound. It brings up a very small quantity of thick sputum.

**Pulse.** Rate (27.2.20) is 84.

The Arterial wall is thickened.

The Pulse is regular in rate and rhythm and the beat is fairly well sustained.

The left pulse, as estimated by the finger, is weaker than the right and slightly delayed.

As estimated by the sphygnomanometer the pressure on the right side which was required to obliterate the radial pulse was 20 mm. mercury more than that required to obliterate the radial pulse on the left side.

- **Right Side.** 105 mm.
- **Left Side.** 85 mm.

A Sphymographic tracing was taken on both sides but no differences in the arterial wave on either side could be discovered.

**General circulation.** This is rather sluggish in the lower extremities and varicosities are present in the saphenous veins in both thighs. Patient states that his feet swell up at nights.
Percussion of the Heart and Lungs
HEART.

Inspection.

The apex beat is not visible. Some dilated veins run across the left shoulder 2" - 3" below the clavicle.

No pulsation can be seen in the thorax or in the neck, but slight epigastric pulsation systolic in character can be made out.

Palpation.

After some time a diffuse pulsation of the anterior chest wall was appreciated but this was extremely slight and difficult to make out.

The apex beat could be palpated in the 6th Intercostal space just outside the mid-clavicular line.

No thrill could be detected.

Percussion.

There was some slight encroachment on the Superficial Cardiac dullness.

Percussion revealed a dull area over the lower part of the Manubrium Sterni and an increased dullness to the right and left of the sternum.

The heart is not enlarged laterally but is displaced downwards.

Percussion/
Area of dulness in the left intercostal region.
Percussion in the left Interscapular region, posteriorly revealed a slight dullness.

Auscultation.

In the mitral area the sounds are closed and distinct, and no lesion can be detected.

In the Aortic area a Systolic murmur can be made out faintly, not well propagated. The second sound is accentuated.

In the Tricuspid and Pulmonary Areas the sounds are closed and distinct.

The veins in the front of the chest are prominent, especially on the left side of the clavicle.

X-ray Examination.

Report:

"Very definite aneurismal dilatation of conus arteriosus".

Fibrosis of the Right lung was also observed.

RESPIRATORY SYSTEM.

Subjective symptoms.

Cough as has already been described, of a typically "brassy" character.

Breathlessness has become very marked recently and occurs even after slight exertion and severely incapacitates patient.

A/
A feeling of tightness across the chest occasionally troubles the patient but is relieved when patient gets up some sputum.

Patient has had no pain or haemoptysis.

Breathing. Frequency (27.2.20) 24 per minute. 
Type is chiefly abdominal.

Sputum. - is thick, viscid and mucoid.

Upper air passages exhibit nothing of note.
Tracheal Tugging was well marked, especially when the patient flexed his head backwards.

Thorax.
Inspection. The chest is well developed and there is no sign of rickets or other deformity. A slight flattening is apparent on the left side.
The chest movements are equal on the two sides. The apical expansion is small.

Palpation. Vocal fremitus is diminished more so on the left side.

Percussion. On the right side the percussion note becomes dull as the sternum is approached.
On the left side there is also a slight dullness at the side of the sternum - further out on both sides than normally.
At the back there is dullness in the left interscapular region/
region at the level of the first Dorsal Vertebra.

**Auscultation.** On the Right side over the upper part the sounds are Broncho-vesicular. Lower down they are less harsh.

On the Left side the breath sounds are diminished in the upper part.

At both bases the sounds are normal vesicular. Vocal Resonance is diminished over the upper part of the Left lung.

No accompaniments were detected.

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**INTEGUMENTARY SYSTEM.**

There are no subjective phenomena.

Slight scarring was detected on the legs, more marked on the left, and slight pigmentation.

Patient states that 18 years ago he had "boils" here which formed ulcers, probably specific in nature as they cleared up under treatment quickly.

Subcutaneous tissues show nothing abnormal. Patient states that his legs swell at nights after working.

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**URINARY SYSTEM.**

There are no subjective phenomena.

**Urine.** Specific gravity 10°S - no abnormalities in appearance or constituents.
Quantity passed averages 60 oz. per day.

REPRODUCTIVE SYSTEM.

No subjective phenomena and no abnormalities.

HAEMOPOIETIC SYSTEM.

There is nothing to note.

ALIMENTARY SYSTEM.

There are no subjective phenomena.

Teeth - in upper jaw they are false - in the lower jaw the molars are missing - the remainder fairly healthy. No signs of pyorrhoea.

Tongue is large, furred and tremulous.

Deglutition is normal. There is no dysphagia.

Bowels are regular and faeces normal.

Abdomen.

Inspection shows epigastric pulsation as already mentioned. Nothing else abnormal is observed beyond a slight pigmentation round the umbilicus.

Palpation, Percussion and Auscultation revealed nothing of note. No abnormality of any internal organ.

NERVOUS SYSTEM./
NERVOUS SYSTEM.

Higher Mental functions.

Intelligence is average, memory is good and there are no mental changes. Sleep is sometimes interfered with by his cough. Speech and articulation normal, and no alteration in patient's voice.

The Cranial nerves are all normal and are entirely unaffected.

Cervical Sympathetic.

The left Pupil is slightly larger than the right and it reacts sluggishly to light.

The right pupil is normal in size and reacts normally to light.

Patient has flushing of the cheeks but states that he always has had this.

Motor Functions.

There are no abnormal movements and no paralysis. Muscularity is good and patient's power of coordination is also good.

Reflexes. The Superficial, Deep and Organic Reflexes are all unchanged.

There are no Sensory Disturbances.

Vasomotor/
Vasomotor and Trophic functions are normal beyond the flush of the cheeks already mentioned.

**LOCOMOTOR SYSTEM.**

Patient has slight Rheumatism in the finger and shoulder. Otherwise there is nothing to note.

**WASSERMANN REACTION.**

Was strongly positive on both occasions on which the blood was tested. (28.2.20 and 13.5.20)

**PROVISIONAL DIAGNOSIS.**

"Aneurysm of the transverse part of the Arch of the Aorta".

**TREATMENT.**

Patient is kept in bed with light diet and little fluid. Potassium Iodide gr X T.I.D. increasing to gr XV T.I.D. 0.5 grm. N.A.B. 18.3.20 5.4.20 13.4.20 4.5.20 11.5.20 18.5.20. Six injections intramuscularly.

**PROGRESS NOTES.**

Patient steadily improved. This cough improved greatly and his weakness disappeared, together with all his subjective signs. On May 3rd he was allowed up.

A/
A second X-ray photograph showed that the dilatation had diminished greatly in size. Every day the patient reported improvement and on May 22nd he was discharged "much improved".
INTRODUCTION.

By way of definition an aneurysm may in the words of Osler be described as a tumour containing fluid or solid blood in direct communication with the cavity of the heart, the surface of a valve or the lumen, and the difficulty of giving a general definition is seen when it is found that even this general statement cannot be said to embrace the whole subject — such conditions as simple dilatations of the aorta or its branches and abnormal communication between two vessels being hardly included by the definition. However it must suffice for want of a better, and it certainly embraces the case of our patient as the unfolding of the commentary will show.

The case which has been described is a typical one in all respects identical with those first described by Galen in the distant past. The clearing up of the pathology, etiology and classification was a gradual process in which besides Galen, Antyllos (100 A.D.), Perusiaus and Vesalius (1500) and Ambroise Paré were the outstanding personalities, more especially the latter who was the first to suggest the relationship of Aneurysm to Syphilis which is so well illustrated/
illustrated by the present case. Since his time many observers have turned their attention to the subject, among whom should be mentioned Morgagni, Hunter, Scarpa and Thoma, so that to-day the subject has been almost completely unravelled and very little remains to be added.

THE ETIOLOGY.

There are two important factors which play a part in rendering an aneurysm a possibility.—

I. A localised loss of resistance of the arterial wall, together with diminished elasticity.

II. An increased blood pressure sudden or prolonged.

In normal conditions, notwithstanding the variations in this stress from moment to moment, and its maintenance up to the point of physiological efficiency during a long lifetime, the balance existing between the elastic resistance of the vascular walls (chiefly the middle coat) and the forces tending to expand them is wonderfully well preserved, and thus persons may attain an advanced age in whom neither the heart nor any of the arteries appear to have suffered in any degree. This is one of the most wonderful phenomena of the human body, more especially when the subject is considered from the physical point of view and, in the words/
words of Gairder "we remember how difficult, nay how impossible - it would be to construct an artificial machine of elastic and distensible materials which would not only resist indefinitely a constant mean internal pressure acting on it through the contained liquids, but also a sudden impulse and variable increase of that pressure repeated periodically at the rate of over 100,000 times a day or say 40,000,000 times a day unceasingly for all the seventy years of an average healthy human life". (Gairder - "Aneurysm of the Aorta" 1899)

Now considering the two factors, heightened blood pressure and weakened arterial resistance, there are certain predisposing factors which are conclusive to these, and it will be our duty to attempt to discover this in the case of our patient.

A. First of all increased blood pressure is caused for longer or shorter periods by violent efforts or prolonged exertion, more particularly efforts out of proportion to the strength of the individual and in those people who are out of training or those who have passed middle age.

This factor can be seen to have played its part in the case of our patient. His occupation was that of a weighman and entailed much sudden exertion in the form of lifting heavy weights on to the scales. This sudden exertion would cause an/
an equally sudden increase of blood pressure which if acting on an arterial system of normal elasticity would produce little effect, but if acting on a diseased artery and one of diminished elasticity would naturally tend to cause the latter to yield most at the part which received the greatest shock.

Thus the occupation of the patient rendered him particularly liable to aneurysm of the aorta and it is of interest that as far back as 1871 Sir Clifford Allbutt called attention particularly to certain occupations liable to sudden strain and which he described as "not only the cause but the commonest cause of aneurysm of the aorta" and his list included the occupation of our patient. (Allbutt - St George Hospital Report - 1871).

B. And now to take up the second determining cause, namely, localised loss of resistance of the arterial wall and diminished elasticity. In the words of Osler the most important single cause of arterial degeneration are the acute infections. In the case of our patient such acute fevers as scarlet fever, diphtheria, smallpox and typhoid fever can all be dismissed for the patient states that he has not had any of these. In any case they are unimportant as far as aneurysm is concerned. There is only one infection of any moment which is connected with aneurysm, namely, syphilis. This, as already mentioned, was recognised by Ambroise Pare and/
and since then multitudes of statistics have been compiled to prove this point, of which the quotation of one will suffice. Francis K. Welch in calling attention to the great frequency of syphilis in association with aneurysm in the British Army stated that in all cases he found that 66.1 were syphilitic. These figures have been confirmed and most observers regard them as rather low if anything.

Now the patient gives a history of having had syphilis, twenty years ago - his Wassermann reaction is strongly positive. So that the second of the two principles required for the production of aneurysm is present in our patient.

Many other subsidiary causes have been mentioned by various writers and it is somewhat disquietening to find that at least two of these are also exhibited by this patient. Osler states that patches of mes-aortitis produced by pneumonia or erysipelas are possible causes of aneurysm. In 1913 patient had a severe attack of erysipelas which lasted ten weeks. The significance of this in the presence of other causes cannot be decided.

Secondly since the time of Vesaluis external injury has been recognised as an occasional cause of aneurysm, more particularly a blow on the chest or a sudden fall. Our patient gives a history of a fall from a staging in 1914 and landing on his back.

Thus/
Thus in summing up it can be said that the case of our patient is a typical one illustrating all of the causes essential to the production of aneurysm - arterial degeneration due to syphilis and possibly to the acute infectious fever, and the sudden increases of blood pressure being provided by the occupation of the patient. In addition the arterial degeneration sets up a vicious circle necessitating an increased blood pressure to carry on the circulation through the narrowed arterial system.

PATHOLOGY.

It has been stated that in this case the cause of the arterial weakness is syphilis and it only remains to show how this disease affects the vessels. Syphilis may be said to affect the vessels in two ways in order to produce an aneurysm.

(1) A diffuse endarteritis.

(2) A localised syphilitic aortitis.

In the first case the pathological change is an endarteritis obliterans consisting of a progressive thickening of the tunica intima leading to a diminution of calibre of the affected vessels. This change affecting the nutrient arteries of the aorta, the vasa vasorum, would gradually obliterate them and cause/
cause degeneration of the wall of the aorta and thus loss of elasticity and weakening.

The more important change however is the localised syphilitic aortitis which is a definite and well recognised lesion. Macroscopically it is limited in extent involving the root of the vessel or a band an inch or two in width of the arch of the aorta.

The intima exhibits transverse and longitudinal puckering with bluish shallow depressions between.

Macroscopically there are found:

I. Foci of small celled infiltration in the adventitia and media sometimes so large as to resemble miliary gummata.

II. Necrosis fracture, separation and disappearance of the elastic and muscular fibres usually in patches.

III. The Spirochaeta Pallida may be demonstrated.

A patch of mes-aortitis may be present, due in this case to the acute infection erysipelas.

Thus, understanding the nature of the lesion which has been produced in this patient, one can quite readily follow the series of changes which have followed this localised loss of integrity of the arterial wall; how that part which is most subject to pressure — namely the transverse part of the arch — being diseased has given way before the sudden increases of arterial pressure which have been showered upon it in the course of the patient's occupation.
REPRODUCTION OF X-RAY PHOTOGRAPH
THE DIAGNOSIS AND DIFFERENTIAL DIAGNOSIS.

In this case the symptoms and signs are very characteristic so as to be almost classical and thus when taken in conjunction with the X-ray appearance little doubt as to the diagnosis can remain.

Certain points may be emphasised however in favour of the diagnosis:

(a) The pulsation observed on palpation over the upper part of the chest in the region of the manubrium sterni.

(b) The dullness heard on percussion over the manubrium sterni, extending half an inch to the left side and slightly to the right; also the dullness on percussion observed in the left Interscapular region.

(c) The systolic murmur heard in the Aortic region.

(d) The pressure effects - such as the brassy cough, husky voice, dilatation of the left pupil, faint breath sounds over the left lung and the prominent veins over the left side of the chest.

(e) Finally the general signs such as weakness and breathlessness - tracheal tugging and weakened and retended left radial pulse, and above all the X-ray appearance.

These facts taken in conjunction with the history of two definite causes and the appearance of the patient - robust-looking/
looking and of good colour with what Osler describes as the "cardiovascular facies" leave little doubt as to the diagnosis, although certain conditions must be differentiated.

1. **Intra-thoracic tumour** transmitting pulsation from the aorta is the main obstacle though in this case it is not a serious one. The patient's extremely healthy appearance negatives a new growth. He is well nourished and has lost no weight recently.

   The expansile nature of the pulsation could not be made out in this case and so was of no help in differentiating the two conditions.

   The X-ray appearance is different. In this case the shadow is continuous with that of the heart and is quite regular, whereas that cast by a malignant new growth would almost certainly be irregular.

   The evidence of organic changes in the cardio-vascular system and the systolic murmur heard in the aortic region clinch the diagnosis.

2. **Dynamic dilatation of the aorta** may under certain circumstances greatly resemble aneurysm but again the cardio-vascular changes, the history of a cause, and the X-ray appearance rule out the condition - together with its persistence after prolonged periods of rest.

   The condition of dynamic dilatation of the aorta is usually/
usually due to aortic insufficiency. In this case there is no aortic incompetence, the aortic second sound being quite clear and distinct.

(3) ** Mediastinal Abscess ** is differentiated by the lack of pyrexial symptoms - by the length of the history and the lack of a course.

(4) A pulsating empyema has occasionally given rise to difficulty but in this case the difficulty does not arise for the clinical picture of the case is quite characteristic and if any lingering doubt remained the X-ray appearance would dismiss it.

THE LOCALISATION OF THE ANEURYSM.

This paragraph must be inserted more from the point of view of completeness than any clinical benefit which would be derived from studying it.

From an extensive co-operation of clinical cases and post-mortem examination it has been found that when an aneurysm is located chiefly in a certain portion of the arch of the aorta certain typical signs may be produced, although of course it must be admitted that in cases of the larger aneurysms these merge into one another.

Thus according to Gulland the following four parts of the arch give characteristic signs:

(a)
(a) The ascending part of the arch.
(b) The junction of the ascending part and arch.
(c) The descending part of the arch.
(d) The transverse part of the arch.

Taking these in sequence:-

(a) The ascending part of the arch - here aneurysms pass to the right and forward - pressing on the Superior Vena Cava, the Right Subclavian Vein and the Right Internal Mammary Vein causing typical signs. The right lungs may also suffer from pressure.

(b) The junction of the ascending and transverse part of the arch. Here the signs are similar - the pressure is found more particularly on the right bronchus with the usual train of symptoms - bronchitis, bronchiectasis, etc.

Both these positions are eliminated in the present case for in it the symptoms and signs are found on the left side.

(c) The descending part of the arch. Here an aneurysm passes to the left and backwards, causing pressure on the left bronchus, the left sympathetic trunk, the oesophagus and finally the vertebrae.

At no time have there been any symptoms of pressure on the oesophagus, so this part is not the part chiefly affected.

(d) The transverse part - this is the only remaining part, and an aneurysm here also causes typical signs - indicated by tracheal/
RELATIONS. OF THE
ARCH. OF THE AORTA.
tracheal tugging, alteration in the cough, husky voice, and also well marked general symptoms of aneurysm, including pressure more particularly on the left side. And on reading over this patient's symptoms, no doubt can be left as to the localisation - namely that it is chiefly present in the transverse part of the arch of the aorta.

AN EXPLANATION OF THE MORE OBSCURE SYMPTOMS.

1. Tracheal tugging.
This consists of a distinct tugging imparted to the larynx and trachea with each heart beat, brought out in certain positions more particularly and it is due to the infringing of the aneurysm on the trachea or it may be the left bronchus with each beat of the heart causing downward displacement.

2. Breathlessness.
This may have an immediate effect or a remote origin. By direct pressure on the lung itself the aerating space is reduced and dyspnoea results, as it always does under such circumstances.

Secondly, partial obstruction of the left bronchus would also cause dyspnoea and by the physical signs in this case this factor can be seen to have played an important part.

Thirdly, it is known that pressure on the vagus would of itself reflexly cause dyspnoea.
Osler regarded this as due essentially to compression of the air tubes, although other observers believed that it was reflex in origin and due to irritation of the left recurrent laryngeal nerve.

4. **Dilatation of the left pupil** is due to pressure on the left sympathetic nerve trunk causing irritation of the sympathetic. If there had been paralysis the pupil would have been contracted.

5. **The weakening and retardation of the left radial pulse.** This was believed to be due to the aneurysm acting more or less as a reservoir but more probably it means that the opening of the left subclavian artery into the aorta has become narrowed, consequently impeding the blood flow. This fact has been demonstrated in many cases of Aneurysm of the transverse part of the arch.

6. **The systolic murmur in the Aortic area.** The origin of this is not quite certain. It may be due to the fact that the aortic valves are affected by the same thickening process as the remainder of the arterial system, but it may also be due to a propagation to the aortic area of a murmur produced at the mouth of the aneurysm.
THE PROGNOSIS.

This consists of a mixture of certainty and uncertainty. Based as it is upon irremediable changes in the wall of the aorta it is practically certain that ultimately the aneurysm will rupture. But the aneurysm has certainly improved under the influence of treatment and a second X-ray photograph indicates that it has become smaller. Thus it can be concluded that the patient, so to speak, takes his life in his own hands and the immediate prognosis depends upon the manner in which he can mould his life and habits to his conditions, and if he can do this successfully the prognosis for a few years at any rate is quite good. The fact that the aneurysm is definitely syphilitic renders the prognosis better than it would otherwise be for such aneurysms respond better to active treatment. Thus there is no reason why the patient should not lead a useful life for a few years at any rate, if he can restrain his activities to a minimum.

The ultimate termination, having mentioned the remote possibility of cure by organisation, is usually by rupture into the trachea-pleura, pericardium, bronchus or externally. Otherwise the termination is by cardiac failure, compression of the trachea and suffocation or intercurrent diseases.
**TREATMENT.**

In this case the main lines of treatment consisted of

(1) Maintaining an equable blood pressure and preventing any sudden raising of it.

(2) Treating the cause.

(a) Rest in all cases of thoracic aneurysm is essential and thus the patient was kept quiet in bed for three months and undue exertion of any nature prevented. In addition conditions causing embarrassment of the circulation were treated - thus straining due to constipation or bronchitis were alleviated and flatulence prevented.

(b) **Diet.** Opinions differ as to how far one should go in dieting the patient. In this case the patient was given a light diet, avoiding excess of animal food. Fluid was given in restricted quantities. The believers of the Tufnell form of dieting claim very good result but this diet is not suitable for all cases for it is very Spartan in nature. It is literally a starvation diet, made up as follows.

- **Morning meal.** 2 oz. Bread and Butter; 2 oz. Milk or Tea.
- **Mid-day.** 3-4 oz Meat; 2-3 oz Potatoe; 3-4 oz Water.
- **Evening.** 2 oz Bread and Butter; 2 oz Milk or Tea.

Many cases cannot stand this and cases have to be suitably selected.
selected. A modified Tufnell diet however is very useful.

(c) Drugs. Having found a definite cause, treatment was directed along these lines, and the patient was given 6 injections of Novarsenobillon intramuscularly (0.3 gm.) together with Potassium Iodide in ten grain doses three times a day. The latter appears to do good even in non-syphilitic cases.

The response to this combined treatment was excellent, as indicated by the alleviation of the patient's symptoms, and the satisfactory diminution in the size of the aneurysm as indicated by the second X-ray photograph.

CONCLUSION.

In conclusion one may say that a study of this case has emphasised the importance of syphilitic infection in the causation of aneurysm and that when this is combined with an occupation which involves sudden increases of blood pressure an open invitation is extended to the formation of aneurysmal dilatation.

If a vessel is going to give way the part which has gone in this case, namely the transverse part of the arch of the aorts is particularly liable, receiving as it does the main shock/
shock of any increase in pressure.

Finally the prognosis is bound up in the patient himself. The patient of the "don't care" type cannot be given a long span of existence even with the most diligent treatment otherwise, but a patient as this one who is intelligent and will actively co-operate with the physician and who will submit to a long and wearisome regime of treatment - this patient although feeling physically very fit, has submitted to lying on his back continuously for 3 months - can be given a much better prognosis for one knows that when he leaves the hospital he will "cut his coat according to his cloth" and thus will quite probably live a comparatively long and useful life.
III.

A CASE OF PARALYSIS AGITANS.
Robert W.  Aged 45  Married.

Occupation.  A miner.

Complaint.  (a) "Weakness of the left leg and left arm.
           (b) Weakness and pain in the back.
           (c) Shaking of the left arm."

Duration.  Five years.

History of Present Illness.

Patient is not quite certain when he noticed any pain first, but the first time he can remember was about five years ago when he was conscious of a slight pain in the lumbar region running up into the left shoulder. This troubled him but slightly for two years, but in 1917 the pains became more noticeable, and were of a burning nature. They now also affected his left arm and leg. The pain was now followed by a definite weakness in the limbs affected, so that patient was unable to continue with his work as a miner.

After ten weeks rest he had improved slightly and resumed work again, but after a short time he was again troubled by the weakness which was more marked on the left side/
side of his body than on the right.

This condition progressively became worse and in addition to the weakness the left limbs became blue and cold.

Six months ago he had an attack of lumbago and after this he noticed that his legs became much weaker and "shaky", more especially on coming home from work at night and in the morning when he woke he found his legs were stiff.

Three months ago he could not stand up properly and often fell, so he was compelled to give up work. About this time he noticed that he was having difficulty with his speech and as the condition was becoming progressively worse he came to the R.I.E. on the advice of his doctor, and was admitted on October 29th.

Previous Illnesses.

Patient has no recollection of any illnesses until five years ago when the present one commenced. (40)

At this time he had bronchitis following influenza. Two years later he had pleurisy on the left side and each Winter since then he has suffered from Chronic Winter Cough.

Accidents.

When he was 35, ten years ago, a portion of the roof of the mine in which he was working fell on him and crushed his/
his pelvis. He was off work for three months and since then has had slight weakness in the left leg and a limp.

General Surroundings at work.

(1) At Work. When his present illness commenced, five years ago, his occupation was that of a miner. He states that the pit in which he worked was very damp and after working hard and sweating he used to return for a good distance on the top of a cable car.

Recently he has worked as a labourer in a linoleum factory and here, the patient states, his surroundings were fairly good beyond the fact that he usually worked in a draught.

(2) At Home. Surroundings at home are quite satisfactory.

Habits.

Very occasionally he indulges in a glass of spirits, usually when he is suffering from a "cold". He is a non-smoker.

There is no history of syphilis.

Family History.

Patient has been married for 20 years. His wife and three children are alive and healthy.

Four/
Four children are dead (one dying of jaundice aet. 3 months, another of Diphtheria aet. 4 years, and two were still-born, one due to a fall of the mother, and the other from unknown cause.)

His father died (aged 44) of Chronic Bronchitis and Asthma. His mother is alive and well, as are also his two brothers and sisters.

Examination.

Patient's intelligence is difficult to judge owing to his slowness in answering questions, but it is obvious that he has no mental defect and his memory is quite good. He takes an intelligent interest in his condition and is not at all apathetic, indeed on occasions he becomes very worried and dejected. He does not suffer from any rapid change of emotions.

His height is 5 ft. 6 ins. and his weight 10 stone 2 lbs - he has not lost much weight recently.

Development. Patient's muscularity is fair and there is no obvious wasting of muscles. There is a marked increase in the tone of the muscles generally, more particularly of those of the left arm and leg, but also to a slight extent on the right side.
General Appearance. Patient's face is dull and expressionless and typically mask-like. His eyes however are bright and when he smiles, the smile although slow in coming lingers for a considerable time. On standing patient adopts the following position - the head and body are bent forwards, the elbows are flexed nearly to a right angle and stand out a little from the sides - the fingers flexed and the thumb resting against the forefinger. The legs are slightly bent at the knee.

Patient has a severe form of Acne Rosacea - very marked over the forehead, nose and cheek - otherwise he has no obvious morbid appearances.

His temperature is subnormal, being 97°.

EXAMINATION OF THE NERVOUS SYSTEM.

Higher cerebral and mental functions.

As already mentioned patient's higher cerebral functions are up to the average and his emotional state shows nothing beyond a desire to worry very much over his condition.

His memory is good and he sleeps well.

Speech. This is slow, monotonous and indistinct and the words have a tendency to be rushed out but with mumbling articulation. Patient takes a long time to answer any questions put to him.
CRANIAL NERVES.

1st (Olfactory) and 2nd (Optic) are unaffected. Patient can read Snellen's chart with ease at the standard distance.

3rd (Oculomotor) The External Ocular muscles are functioning normally and there is no squint - diplopia or ptosis.

Nystagmus is present in both eyes - more marked in the left eye. It is horizontal in type and slow and rhythmical in character.

The Pupils are rather dilated but are equal in size and regular in shape.

They react to light and to accommodation but react more sluggishly than normal.

The remainder of the cranial nerves were carefully tested but were found to be functioning normally.

The Cervical Sympathetic also presents no abnormalities.

MOTOR FUNCTIONS.

There is a marked tremor of the left hand and a slight tremor in the right hand. This is quite involuntary and continues during rest. It is rhythmical in character, and its frequency is about 3 per second. It spreads up the left arm and is also seen to a slight extent in the left leg. In addition patient exhibits a fine vertical tremor of his head.
head. The trunk muscles are free from tremor.

The fingers are slightly flexed and the thumb is resting against the forefinger and constant slight extension and flexion of the fingers and thumb result in a movement resembling pill rolling being produced. Muscular weakness is present in the parts affected by tremor. Thus patient's grasp is poor, more especially in the left hand. There is a loss of associated and automatic movements.

Thus when patient is told to close his hand and then open it, the opening movement is slow and is not accompanied by the associated movements of spreading of the fingers and abduction of the thumb, which is seen in normal people. On grasping an object there is no associated movement of extension of the wrist, which should be seen.

Rigidity of muscles is seen, more especially in the right arm and hand, but is also present in the left, causing the movements performed to be slow and deliberate. And on passive movement it is felt to be a rigidity that is almost wax-like but is interrupted by rhythmical sensations due to the intrinsic tremor tendency of the muscle substance.

Co-ordination is impaired, chiefly because of the rigidity and weakness of the muscles and a well marked intention tremor is brought out when patient attempts to touch his nose with either forefinger, more especially on the left side. Incoordination is also shown by the left leg muscles.
REFLEXES.

The Superficial reflexes are all quite normal. There is no Babinski sign.

The Deep reflexes. The Biceps Jerk is exaggerated on both sides – the other arm reflexes are present but not marked. The Knee jerks are brisk, more especially on the left side. The other leg responses are normal, and no clonus (ankle or patellar) was detected.

Organic Reflexes are unaffected – micturition and defecation being normal.

The Winking Reflex is sluggish, giving patient a profound stare.

SENSORY FUNCTIONS.

Subjective sensations.

Pain is now present in the right elbow. Patient occasionally suffers from tingling on the left side, first in the feet and then in the arm. This however is not now present.

There is a feeling of cold in the left arm which is sometimes accompanied by a feeling of numbness.

There is no headache or giddiness.

Sensibility to touch.

Patient is somewhat hypersensitive to touch. Otherwise there is no alteration in his sensibility in any part of the body.
Heat and cold sensation is in no way affected.
Tickling throws the limb into a series of tremors.
Localisation of touch and discrimination is unimpaired.
Muscular sense is good.

**VASOMOTOR AND TROPHIC FUNCTIONS.**

Since admission patient has developed a well marked Housemaid's knee which he is unable to account for, on the left side. Otherwise there is nothing to note.

**WASSERMANN REACTION - is Negative.**

**LOCOMOTORY SYSTEM.**

Patient's gait is of a shuffling character. He rises up slowly to the erect posture and presents the attitude already described - namely of flexure in all parts of the body and on attempting to walk commences slowly but, gaining more confidence gathers speed as he goes on, the steps being short and dragging on the floor. Thus there is a tendency to "propulsion" and this has caused patient to fall on several occasions.

There is no tendency to retropulsion.

Patient's/
Patient's walking is at present embarrassed by an effusion into the right knee joint, this being only a temporary phenomenon.

The musculature of the patient has already been commented on - the hypertonicity, causing the rigid feeling on palpation, and the "mask like" appearance and the muscular tremor in the left arm, left leg and right arm are quite characteristic.

**ALIMENTARY SYSTEM.**

Patient has very poor teeth in both jaws, many being decayed - no definite pyorrhoea is to be seen. His tongue is coated and his breath foul. He has no subjective symptoms. Patient has a good appetite and takes his food well. His bowels are regular. Abdomen - shows nothing abnormal on inspection, palpation or percussion.

**HAEMOPOIETIC SYSTEM.** - is quite normal.

**CIRCULATORY SYSTEM.**

Subjective phenomena - occasionally patient has slight palpitation and dyspnoea. No cough. Pulse./
Pulse. Arterial wall not unduly thickened.
Frequency 79 - regular and pressure normal.

Heart. Inspection - no pulsation, apex beat not visible.
Palpation and Percussion revealed no enlargement of the heart.
Auscultation - the sounds in all the areas are distinct and sharp - no murmurs.

RESPIRATORY SYSTEM.

No subjective symptoms beyond occasional dyspnoea.
Breathing chiefly diaphragmatic. Frequency 20 per minute.
Thorax. Chest is slightly "barrel shaped" and moves slightly on respiration.
Palpation - reveals that vocal fremitus is normal.
Percussion - gives a hyper-resonant note towards both apices.
Auscultation. Breath sounds normal vesicular no accompaniments and Vocal Resonance normal.

INTEGUMENTARY SYSTEM.

Patient has Rosacea on face as already described, and has had it for five years. He associates it with the state of his health and says it is worst when his pains are bad. Skin is rather oily about the face, otherwise presents no morbid appearances.
URINARY SYSTEM.

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No subjective phenomena - Micturition is normal. Urine is of normal specific gravity and shows no deposits or abnormal constituents.

REPRODUCTIVE SYSTEM is normal.

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PROVISIONAL DIAGNOSIS.

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PARALYSIS AGITANS.

TREATMENT.

Pot. Iodide XXX gr. ) T.I.D.
Sod. Bicarb. XX gr. )

Hyoscin Hydrobromide \( \frac{1}{200} \) gr. hypodermic

Rest, light diet, and muscular exercises.

PROGRESS NOTES.

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November 4th. Patient's tremor, appearance and general conditions are as mentioned in the report. He is very depressed.

November 9th. Patient is becoming more cheerful in his outlook and is taking interest in his treatment and following it out earnestly. There is no definite improvement in the signs and symptoms.

November 16th/
November 16th. Patient is becoming more confident in himself on doing many movements with much greater facility than he previously could.

November 23rd. There is now a definite improvement on the tremor and patient is able to control it better. He has now much more confidence in himself.

December 3rd. The patient is vastly improved, more especially with regard to his muscular disability but also with regard to his tremor. He has succeeded in educating himself in many of the movements he had lost, and he is being discharged in a very hopeful and cheerful frame of mind.
INTRODUCTION.

It is now over a century since James Parkinson contributed to medical literature his well known essay on the "shaking palsy" and described with great exactness the chief clinical features of the disease which now bears his name, (James Parkinson - "An essay on Shaking Palsy" London 1817) and the closeness with which the features enumerated by him in 1817 correspond with those shown by this patient is most striking.

Since the time of Parkinson many contributions to the symptomatology of the subject have been made, and much pathological work done in efforts to get at the seat of the lesion and its cause: and the theories held on the subject were extremely divergent, the lesion being placed by different observers in such widely dissimilar structures as the cerebral cortex, cerebellum, basal ganglion, the brain stem, the spinal cord and even in the muscle itself. And even to-day when the main seat of the lesion has been localised with comparative certainty, divergent views are still held by various observers.

Now the reason of all this is probably that Paralysis Agitans is a syndrome with a definite and characteristic complex/
complex of symptoms which may be caused by a variety of pathological lesions. Thus our views on this subject may be compared with our views on the symptom complex described by Ménière and known as Ménière's disease which was afterwards found to be dependent on a number of distinct pathological entities. Another illustration is found in the subject of "Chronic Rheumatism" whose mysteries of origin are just beginning to be unravelled.

Thus for many years Paralysis Agitans was classed among the unknown factors in medicine, and it was only as recently as 1908 that Jelgersma in a masterly resume on the subject directed attention to the Corpus Striatum (Jelgersma - Nene Anat. Befund bei Paralysis agitans - Neurol. Centralb. 1908). For a time no advance was made but in 1917 Dr J. Ramsay Hunt of New York analysed many cases, and had the opportunity of pathological examination of some of these and at last succeeded in differentiating the types of the disease and proving that the main seat of pathological change was indeed in the Corpus Striatum (Hunt - Progressive Atrophy of the Globus Pallidus - "Brain" 1917 Vol. 40).

To-day though our knowledge is still far from complete it is generally recognised that paralysis agitans is a symptom complex having as its origin a degenerative change in the Corpus Striatum.

Anatomy.
A. HORIZONTAL SECTION THROUGH THE
RIGHT CEREBRAL HEMISPHERE
AT THE LEVEL OF THE
LENTIFORM NUCLEUS

(CUNNINGHAM)
Anatomy. Before discussing in more detail the Pathology of Paralysis Agitans it is first necessary to clear up certain points about the anatomy of the Corpus Striatum.

Cunningham describes the Corpus Striatum as a mass of grey matter embedded in the base of each hemisphere consisting of two parts:-

(1) A supero-medial part - the Caudate nucleus
(2) An infero-lateral part - the Lentiform nucleus.

The anterior parts of these are blended but the remainder are separated by a thick layer of white matter - the Internal Capsule.

Further the Lentiform Nucleus is subdivided into an external part the Putamen (Neostriatum) and an internal part the Globus Pallidus.

Philogenetically the Globus Pallidus is much older than the Putamen and is found as low in the animal scale as the fishes, while the Putamen is not found until one ascends the scale as high as the reptiles.

Histologically the two structures also differ. The Globus Pallidus contains aggregations of fusiform, pyramidal and large multipolar cells of motor type while the Putamen consists of two distinct types of/
of cells; firstly, similar large multipolar cells as seen in
the globus pallidus, and secondly small pyramidal stellate
or polygonal cells, the latter being much the more numerous.

The small cell fibres end in the Globus Pallidus and
constitute a short association and inhibitory system for
the Corpus Striatum (atrophy of these fibres causing the
syndrome of Huntingdon's Chorea).

The large cell fibres have longer axis cylinders and
pass in the ansa system to the thalamic and hypothalamic
regions.

Malone, on whose observations this description is based,
believes that the large type of cell is the homologue of
the Cells of Betz in the cortex and concludes that the Globus
Pallidus is the essentially motor part of the Lenticular

Other connections of the large cell system are with the
Red Nucleus and Substantia nigra, and hence with the Spinal
Cord. No connection with the cerebral cortex has been discovered.

Physiologically, - the Corpus Striatum and strio-spinal system
are to be regarded as constituting a
mechanism for the control and regulation
of automatic and associated movements contrasting with the
cortical system which controls isolated and discriminating
movements. When functioning normally it exerts a controlling
influence/
influence on the tone of all muscles concerned with automatic and associated movements. (Vogt Sydrome du Corps strié - Neurol. Centralb. 1911).

Pathology of Paralysis Agitans.

Since Jelgersma in 1908 described lesions in the basal ganglia much pathological investigation has been carried out, and the following observations are based on the findings of Lewy, Manschot, Auer and McCough.

I. Various stages of chronic atrophy are found in the large ganglion cells of the Putamen, more especially and to a lesser extent in the Globus Pallidus. Their bodies are shrunken, their processes atrophic, their nuclei contracted and occupying a lateral position in the cell body.

II. The small ganglion cells are well preserved and show no atrophy.

III. The vessels of the region are thickened and in the perivascular and pericellular spaces pigment is found and a yellowish lipoid material.

IV. The nerve fibres are reduced in number, more especially those of the ausa system (Strio hypothalamic) showing thinning and atrophy.

V. No other constant pathological change can be found in the Central Nervous System.

It/
It must be remembered that the type of Paralysis Agitans which is the subject of commentary - namely that occurring in elderly adults, is a presenile condition and concomitant senile changes will, no doubt, often be found in the Central Nervous System and in other parts of the body and this, no doubt has caused much of the confusion which has existed on the subject of Paralysis Agitans.

THE ETIOLOGY OF PARALYSIS AGITANS.

It is unfortunate that our knowledge as to the cause of Paralysis Agitans has not accompanied the advances in our knowledge of the pathology of the conditions and to-day very little light can be thrown on the subject.

This patient may be regarded as a typical case in all respects, for it is certainly usual for Paralysis Agitans to occur in males of advanced middle age.

Occasionally the disease shows a hereditary tendency. Berger in a series of cases found that in 151 of the cases the condition was also present in near relations of the patient and Gowers reports a similar percentage. Most careful inquiries into the case of this patient fails to bring out any facts which could be construed in this light, and the only points which might be of any interest are that in some of/
of the patient's relations there exists a neurasthenic tendency. This will be commented on later.

Again acute disease has been blamed in some cases and in about 30% of cases a history of acute disease occurring before the commencement of the tremor of Paralysis Agitans can be elicited. Stress might be laid more especially on acute disease of the Central Nervous System as a cause and interesting corroboration of this has been obtained in the recent outbreak of Encephalitis Lethargica. Since this epidemic many cases have been reported in the medical press of patients who have suffered from Encephalitis Lethargica, subsequently developing the signs of Paralysis Agitans. Purves Stewart reports cases of this (Purves Stewart - Diagnosis of Nervous Diseases p.137)

Our patient can recollect only one acute disease about the period of onset of his present condition and that was a sharp attack of influenza and although influenza has been blamed for many things, too much stress should not be laid on this. Nor upon the attack of Pleurisy which occurred two years later. Still it is possible that the virus of influenza which often does attack the Central Nervous System might have picked out the Pallidal System of cells as its victim.

Next comes a cause which the older writers laid much stress on, namely the part played by physical injury and emotion, more/
more particularly violent emotion, in the production of Paralysis Agitans. In many cases it is beyond all doubt that these factors are exceedingly important in the production of the disease. It is well known that sudden alarm may cause a general tremor - so much so that the verb "to tremble" is in the process of conversion into a synonym with "to fear". It is quite conceivable that in some cases the tremor may persist and develop into the disease. This fact is best driven home by the example quoted by Gowers (Gowers - Diseases of the Nervous System p.590) of a woman who, while sitting quietly at work was suddenly startled by a stream of water flowing from a tap on to her left wrist and who subsequently developed typical signs of the disease commencing on that part of the body.

Moreover in our patient there is a definite history of a severe accident occurring previous to the onset of his illness and followed by profound shock. Thus this factor cannot be dismissed lightly.

Many other undoubted causes of the disease have been described to combination of fatigue, cold and shock is an undoubted predisposing factor and this patient in his occupation as a miner was exposed to all three.

In certain cases Cerebral haemorrhage was undeniably the cause - in other cases Cerebral Tumour and softening were equally/
equally certainly predisposing or actually causative factors.

In a few cases Syphilis has been described as a cause, but the part played by it is extremely doubtful.

And now in conclusion as regards etiology, how can these very varying factors be correlated? There is no doubt that in different cases all these factors mentioned had a bearing on the development of the disease and there is only one way of explaining this — a way which most observers seem to have overlooked. This has been mentioned earlier in the commentary, namely that Paralysis Agitans is not a disease caused by a certain specific factor but must be regarded as a symptom complex or a syndrome produced by a variety of pathological lesions acting on a certain part of the Central Nervous System, i.e. the Corpus Striatum. When looked at in this light the relationship of Acute Specific disease, of Cerebral tumour, of Cerebral haemorrhage and of senile changes hastened by injury or shock becomes quite clear. In our patient certain of the factors retire to the background and others become prominent. Thus one might say that the degeneration of the Pallidal system in him is due to commencing senile changes hastened by exposure to a severe mental shock with factors such as fatigue, exposure and cold being predisposing factors. And if this was inconclusive to/
to some then one could always bring forward the inevitable influenza, - that extremely useful straw at which the drowning theorist clutches!

**DIAGNOSIS AND DIFFERENTIAL DIAGNOSIS.**

The case is so typical as to be almost classical and very few words will suffice for this paragraph. Sufficient to say that it rests upon the development of the typical tremor followed in due course by the muscular rigidity and muscular weakness. The appearance of the patient, his bent position, his mask-like face, his mumbling speech, together with the history of onset and progression of the condition put the matter beyond doubt. If this were not so the proper place for the paragraph would have been at the beginning of the commentary rather than towards the end.

Certain points of differentiation with regard to the tremor must be mentioned however.

1. **Disseminated Sclerosis** causes a very similar tremor but this commences much earlier in life and the tremor is typically of the intention tremor type, i.e. is marked on performing any voluntary movement and is absent during rest, thus differing from that seen in this case.

   In addition the attitude of the patient and his facial appearance is quite distinctive.

2./
2. Toxic Tremors e.g. due to Alcohol, Mercury, etc. may be very similar, but there is no history suggesting them in this case, patient being a very moderate drinker and a non-smoker. In addition there is no muscular rigidity in toxic cases and again the attitude helps.

3. The Tremor of Senility. This is difficult to differentiate but is more marked about the head than in Paralysis Agitans and is coarse on a horizontal direction while this is in a vertical direction.

4. The pains which occurred in the early stage of the disease, together with the tremor, etc. made one think of Central Nervous Syphilis. However the negative Wassermann, the augmented deep reflexes and the lack of history excluded this point.

SYMPTOMATOLOGY.

It has been mentioned that the cardinal symptoms are three in number - Tremor, Rigidity and Paralysis, and these require a word of explanation in the light of the pathology which is now known.

(1) Paralytic Disturbance. This is partly dependent on the rigidity and partly on the palsy which is of a certain specific type, i.e. it is limited to automatic and associated movement.
movement. It is as if these movements which come instinctively to the normal person were entirely lost to the sufferer of Paralysis Agitans, and that to perform any movement he had to will himself to do it. Thus in the simple movement of rising up in a chair the different sets of muscles should associate themselves in the performance of the act but the associated movement is now gone and the patient has to think out which muscle will be needed for the movement. Thence the slowness on performing it. In the upper arm this is illustrated in the loss of such movements as swinging the arms when walking, which is an associated movement bequeathed to us by our quadriiped ancestors.

All this is explained by the fact that the centre which controls automatic and associated movement is in the Corpus Striatum and is destroyed by the lesion.

(2) Rigidity. This stiffness of musculature is a large factor in the production of the motor disability and causes the expression, the attitude and the gait which are so characteristic of the disease.

Its origin is central and is analagous with the spasticity accompanying lesions of the pyramidal tract.

"It is due to a loss of cerebral inhibitory function - or destruction of the higher centre which controls or inhibits spinal tonus" (Ramsay Hunt) and when this inhibition is lost hypertonicity/
hypertonicity of the musculature results. The connections of the centre with the spinal cord have already been illustrated.

(3) Tremor. This symptom is due to the loss of striated inhibition whereby one set of muscles becomes more hypertonic than the opposing set, and the stretching of the latter causes it in turn to be stimulated into contraction and so the circle continues.

As regards cure the prognosis is bad. It has never yet been conclusively demonstrated that, once the true syndrome of Paralysis Agitans has developed, cure can occur. The most that one can hope for is amelioration of the symptoms and signs and even this is usually temporary and of short duration, and the disease tends to progress slowly but surely and to involve as time goes on more and more muscle. And in the light of the pathology of the condition this is only to be expected.

In speaking of prognosis one is referring to the type which occurs in our patient and the other types are excluded - as for example the infective type following Encephalitis Lethargica where the prognosis is quite good.
The pre-senile type which our patient illustrates is founded on irremediable changes in the Central Nervous System.

With regard to life however the prognosis is better - the disease is extremely slow in its progression and hardly, if at all, does it tend to shorten life. The patients usually die of some intercurrent disease of the respiratory system. The older the patients become the less does the disease seem to trouble them and many of the cases of so-called senile tremor are probably due to undiagnosed Paralysis Agitans.

TREATMENT.

There is no conclusive evidence that any of the therapeutic measures hitherto employed have permanently arrested the disease and treatment must thus consist in attention to the general hygiene and avoidance of influences which have been observed to exert a deleterious effect, together with the administration of drugs and other therapeutic measures which have been found by experience to benefit the individual symptoms and perhaps to retard the progress of the disease.

Patient therefore should lead a quiet restful life avoiding as far as possible all cares and business worries. Fresh air and an open outdoor life are of first class therapeutic importance.

The distinct benefit which may accrue from exercises is well illustrated by this patient. This was first recommended/
recommended by Friedlaender, and patient should practice his power of voluntary control two or three times a day. This has a distinct effect upon the tremor. Bramwell recommends in addition to this slow passive movements of extension at the various joints followed by active movements of the extensor muscles against varying degrees of resistance to overcome the rigidity of the flexors.

The patient's attention is directed to the necessity of constantly trying to correct abnormal attitudes, for example, while walking.

Of the various drugs which have been recommended only Hyoscine can be said to have any distinct beneficial effect. Erb pointed out that it diminishes the tremor and general restlessness and causes the patient to sleep better. In this patient its use has certainly been justified, commencing with a dose of $\frac{1}{200}$ gr. Barbitone is of temporary use, and Iodides as given in this case probably help.

Possibly the most important of all is the psychological treatment. If the patient can be inspired with self-confidence remarkable results can be obtained. Sufficient emphasis has not been laid on this point. In Locomotor Ataxia for example it is now recognised that to instil self confidence into the patient is half the battle with regard to the ataxia, and Bramwell/
Bramwell lays much stress on this. Similarly with regard to Paralysis Agitans, if the patient can be made to believe that strict following out of the prescribed treatment will assuredly be followed by great improvement in his condition, then good results can confidently be expected.

The writer is quite convinced that the great improvement in this patient's symptoms was, to a large extent, due to this factor. On his admission patient was introspective, melancholy and despondent with a tendency to neurasthania inherited from his relations. Under hospital regime he soon began to cheer up and regain self-confidence and coincident with this was the improvement in his symptoms.

CONCLUSION.

And now, in conclusion, what points have been learned from a study of this case?

I. Firstly it is now recognised that Paralysis Agitans is a special type of central palsy due to loss of function of the Striospinal system.

II. Strictly speaking it is not a disease but a special type of palsy which may result from a variety of pathological lesions including Senile Atrophy, Vascular degeneration and/
A composite diagram indicating:

**THE STRIOSPINAL SYSTEM in relation to the CORTICO-SPINAL SYSTEM**

- Corpus Callosum
- Caudate Nucleus
- Paramedian
globus pallidus
- Lenticular Nucleus
- Corpora Quadrigemina
- Crus Cerebri
- Red Nucleus
- Dieter's Nucleus
- Pons
- Medulla

**Tracts**

1. Direct Pyramidal Tract
2. Crossed Pyramidal Tract
3. Vestibular Tract
4. Tectospinal Tract
5. Rubrospinal Tract
and various gross lesions of the Corpus Striatum such as softening, haemorrhage, tumours, and intoxications.

III. There are three varieties of nervous control over muscular function.—

(a) The segmental controlling reflex function and movements.

(b) The Striospinal controlling automatic and associated movements.

(c) The Cortico-spinal controlling discriminated and dissociated movements.

IV. The function of the Corpus Striatum, in addition to controlling automatic and associated movements also has a regulating influence on muscle tone.

V. Although cure of the condition cannot be expected in the presenile type - great amelioration of the symptoms may be expected from the prescribing of certain exercises and drugs, and more particularly by giving back to the patient the self-confidence which the slow and progressive course of the condition has taken away from him.
IV.

A CASE OF CHRONIC HYDRAEMIC NEPHRITIS

COMBINED WITH

VALVULAR DISEASE OF THE HEART.
Mrs McP.  Aged 28.  Married.

Housewife.

Complaint - "swelling of the legs".

Duration - "eight years".

History of Present Illness.

The patient states that she first began to be troubled by swelling of the legs eight years ago. At this time she noticed that her legs used to become swollen at night time and that the swelling was above the level of her boots. She had no other symptoms or signs beyond this and for two years this swelling appeared only occasionally after a heavy day's work and it entirely disappeared during the night.

A year after her marriage, (i.e. two years after the appearance of the first signs) the condition became more marked and after the birth of her first baby six months later she also suffered from pronounced breathlessness and she could not sleep unless she had her head propped up with pillows. After lying up for a few weeks the breathlessness passed away and the swelling of the legs disappeared.

Soon/
Soon however it commenced to return and patient states that practically every night on going to bed her legs were swollen but that the swelling had disappeared by the next morning.

After the birth of her second child the symptoms became more accentuated and in addition patient noticed that she was puffy under the eyes when she awoke in the morning. At this time she was admitted to the Infirmary and was treated for a fortnight after which she left at her own request (1916). She states that her stay in the R.I.E. did her temporary benefit, but that the swelling of her face and limbs soon returned. These however did not trouble her and the condition remained about the same for three years when she gave birth to her fourth child. After this she was very ill and states that she vomited blood. She also became very breathless.

In addition she noticed that she had to pass water more frequently, occasionally having to get up once or twice during the night to do so.

The breathlessness and oedema abated under rest and treatment but since this date patient has had occasional attacks of dyspnoea and the swelling has appeared in the limbs every night and in the face in the morning. In addition the patient periodically suffered from headaches.

Five months ago the strain of her fifth pregnancy caused an exacerbation of the symptoms and patient had marked breathlessness/
breathlessness and also palpitation. The oedema of the legs did not disappear during the night and only disappeared when she laid up for a week. About this time a pain in the back also troubled her and frequently she had to get up once or twice during the night to pass water.

As the condition did not seem to be improving patient recently came to the R.I.E. again and after examination was admitted to Ward 24. At this time she had marked oedema of the legs and puffiness under the eyes, but the subjective symptoms had disappeared with rest.

Previous Illnesses.

When the patient was eighteen months old she had "congestion of the lungs" she states and is told that she was very ill at the time. During infancy she had Measles and Whooping Cough.

When nine years of age she had Chorea and she suffered from this at intervals for five years.

She states that she has not had Rheumatic fever but that she had "growing pains" of a severe nature.

During the last eight years she has had "indigestion" periodically but not of a very severe character.

Previous/
Previous Accidents.

Patient has had no actual accidents but the front of her chest is badly scarred over a wide area due to too energetic poulticing when she was suffering from pneumonia.

Surroundings.

Her surroundings at home are fairly comfortable and her work as a housewife is of a somewhat heavy nature, she being on her feet practically the whole day.

Habits.

Patient has been accustomed to eat plain food at regular periods and has a good appetite.

Family History.

Living Members. Patient's mother is alive and well - her father is alive but has been in the R.I.E. suffering from Nephritis. Patient has two sisters alive; one has had Tuberculous glands and the other has Psoriasis. Three of her brothers are alive and well. One died in infancy from unknown cause.

Dead Members. Patient's grandparents are dead, one having died from Nephritis, the others from unknown causes. The ages of their deaths are not known.

Children/
Children. Patient had had five children, of which two are alive. The first died of Diphtheria aged 3 years, the third was still-born and the fourth died of "indigestion" aged 4 weeks.

The other two are alive. But the son aged 6 has swelling under the eyes every morning. The girl aged 1 year is healthy.

Patient gives no history of miscarriages.

General Examination.

Intelligence is average.

Height 5 ft. 2 ins.

Weight 12 st. 4 lbs.

Development is good. Patient has a good deal of adiposity.

Muscularity good.

General appearance. Patient's face has a pasty appearance and is swollen under the eyes and also slightly above the eye. There is also slight anaemia, but there is no cyanosis or jaundice and no other evidence of previous disease.

Temperature 98°.

SYSTEMATIC/
SYSTEMATIC EXAMINATION.

URINARY SYSTEM.

At the time of examination patient has no subjective phenomena of any kind. Occasionally she has an aching pain in the loins, chiefly at the left side. She says that this occurs at no particular period. She has never had attacks of pain shooting down into the groins.

She occasionally suffers from headache, frontal in character and bilateral.

Frequently on waking in the morning she notices that her vision is dim - this passes off however early in the day. This dimness of vision is often associated with headache.

Vomiting occurs, sometimes associated with periods of indigestion and has been very pronounced after the birth of each of her children, more especially with the first and the fourth, on both of which occasions there was blood in the vomit.

Dyspneoa. Patient has had periodic attacks of breathlessness for a few years and these became accentuated after child-birth, causing her to be unable to sleep unless her head is propped up.

Micturition./
EXAMINATION OF THE URINE

RENAL EPITHELIUM
NORMAL
FATTY
DISTURBED

EPITHELIAL TUBE CAST

BLADDER EPITHELIAL CELL

EPITHELIAL CAST

HYALINE CAST
Micturition.

At the time of examination the amount of urine passed in 24 hours is diminished and is 30 oz.

Patient has not to get up during the night now to pass it. For some time previous to this (patient is not sure of the time) she states that she has had marked frequency of micturition and has had on some occasions to get up two or three times during the night to pass water.

There are no subjective phenomena on passing water.

Urine. Quantity passed in 24 hours is 30 oz.

Odour is normal and the colour pale straw colour.

Specific Gravity is 1020.

Reaction is Acid.

There is a fluffy deposit of mucour - not phosphates or urates.

The urine has no opalescence.

Chemical Examination. On carefully testing for albumen a trace is found to be present. (27.5.21)

No blood is present.

No sugar or bile can be detected.

Microscopical Examination. Renal epithelial cells are found to be present and are numerous. There are normal cells, disintegrated cells and cells undergoing fatty degeneration.

A few epithelial cells from the bladder were seen.
casts. Some cellular epithelial tube casts were present but no blood casts could be found. Fine granular casts were seen in some parts. After prolonged search a few hyaline casts were discovered. No waxy casts could be found. Strings of Mucous were seen in parts of the field.

**Renal function tests.**

<table>
<thead>
<tr>
<th>Test</th>
<th>Result</th>
</tr>
</thead>
<tbody>
<tr>
<td>Blood Urea</td>
<td>28 mgr. per 100 cc.</td>
</tr>
<tr>
<td>Urea Concentration</td>
<td>2.2%</td>
</tr>
<tr>
<td>Phenol-sulphone-phthalein</td>
<td></td>
</tr>
<tr>
<td>1st hour</td>
<td>40%</td>
</tr>
<tr>
<td>2nd hour</td>
<td>30%</td>
</tr>
</tbody>
</table>

**CIRCULATORY SYSTEM.**

**Subjective Phenomena.**

There is no pain over the heart, nor at any time has patient had any. Occasionally patient has attacks of palpitation on exertion and her attacks of dyspnoea have already been mentioned.

Patient has no cough at the time of examination but she occasionally has a typically bronchitic cough bringing up some frothy sputum.

**Pulse/**
Pulse.

The pulse-rate is 76 per minute.

It is fairly regular but occasionally there is an extra beat suggesting an extra-systole.

The beats are equal in strength and the pulse is equal on the two sides.

The pulse is difficult to feel but the arterial wall seems to be unduly thickened for a person of the age of the patient (28). The pressure as estimated by the finger is slightly high. As estimated by the Sphygomanometer the pressure is 126 mm.

No dicrotic wave could be felt.

The pulse was too inaccessible for a successful sphymographic tracing to be taken.

Heart.

Inspection. There is no praecordial bulging or flattening and the apex beat is not visible.

Widespread scarring over the chest was due to severe poulticing of the chest when patient was a child.

There is no diffuse pulsation of the chest and no pulsation of the epigastrium.

At the root of the neck there is a double pulsation - venous -
Case IV

Percussion of the Heart (Case IV)
near the middle line and arterial lateral to this.
The veins of the chest are not unduly conspicuous.

**Palpation.** The apex beat can be felt but weakly.
It is displaced downwards and slightly outwards to the 6th rib just outside the mid-clavicular line.
No thrill can be felt on palpation.
No pulsation beyond those already mentioned can be detected.

**Percussion.** The superficial dullness of the heart is not encroached upon.

The deep dullness shows that the right border of the heart is just under $\frac{1}{2}$" to the right of the border of the sternum, the liver dullness stretching up into the fourth right interspace. The left border is further out than normal (see diagram). At the apex it is just outside the mid-clavicular line (4" from the mid line.) At the level of the fourth left interspace it is also further out than normal, being about 3" from the mid-line.

**Auscultation.** In the mitral area a double murmur can be detected. (1) A presystolic murmur. (2) A diastolic murmur both conducted out towards the axilla.
The first murmur is rough and in a closed first sound and the second is of a blowing nature, merging into the first. There/
Indicating the Mucnurs and their propagation

A. AORTIC
B. MITRAL
There is no reduplication of either sound.
In the Tricuspid area the sounds are closed and distinct.
In the Pulmonary area the sounds are closed and there is no accentuation.
In the Aortic area there is a systolic murmur conducted down the chest and up into the neck. The second sound is closed and accentuated.

The heart sounds are not quite regular, an extra-systole occasionally being heard.

ALIMENTARY SYSTEM.

At the date of examination patient has no Subjective phenomena. Her appetite is good and she eats plenty of plain food. Occasionally she suffers from "indigestion" when she has a feeling of weight after eating and pain in the epigastrium sometimes followed by vomiting. This latter was very severe after the birth of her children, especially with the first and fourth when she also vomited dark coloured blood.

Bowels. These at present are quite regular. Patient occasionally has attacks of diarrhoea followed by constipation.
The faeces present nothing abnormal.

Abdomen./
Abdomen,
The wall is flabby and contains a good deal of adipose tissue and is marked with old-standing striae.
There is no prominence or retraction.

Palpation did not reveal anything abnormal. There is no tenderness on pressure over the region of the kidneys. The kidneys themselves could not be palpated and consequently no alteration in size detected.

Percussion showed that the liver was not enlarged nor the spleen. The stomach note was normal in area.

There is nothing else to note.

INTEGUMENTARY SYSTEM.
There are no subjective phenomena such as pain, itching or burning.
The skin has a pasty appearance. The scarring over the front of the chest has already been described.
The subcutaneous tissues contain a good deal of adipose tissue. There is still oedema to be seen below the eyes and slightly above the eyes.
Round the ankles the oedema has almost disappeared but there is slight pitting on pressure.
There/
There is no oedema of the wrists or hands, nor of the conjunctival sac though the latter becomes oedematous in the morning sometimes.

RESPIRATORY SYSTEM.

Patient now has no subjective phenomena. She sometimes has a bronchitic cough which brings up a good deal of frothy sputum. Breathlessness has already been mentioned. Breathing is chiefly thoracic. Frequency is 20.

Thorax.

There is nothing abnormal to be found beyond the scarring externally. The shape is normal, the movements equal on the two sides. The percussion note is resonant on both sides. On auscultation the breath sounds are vesicular with a tendency in parts to be broncho-vesicular, more especially on the right side. A few rhonchi were heard on the right side. Vocal resonance is unchanged on either side.

HAEMOPOIETIC SYSTEM.

Patient has a slightly anaemic appearance which is more apparent than real owing to the oedema of the subcutaneous tissues.

Otherwise there is nothing to note.
NERVOUS SYSTEM.

Higher mental functions. Patient's intelligence is average, her memory is good. She sleeps well unless suffering from breathlessness.

Speech and articulation is normal and she has had no fits and has not suffered from drowsiness or delirium. No hallucinations or delusions.

Cranial Nerves.

1st Olfactory is normal.

2nd Optic. Patient wears glasses sometimes. This is because on wakening in the morning she sometimes finds that her vision is dim and that there is a mist before the eyes. This usually clears up however early in the morning and then she can see quite distinctly. She has had this condition for about eight years (i.e. since the beginning of her illness). She wears concave glasses.

Ophthalmoscopic examination showed that the optic discs were quite normal.

3rd, 4th, 5th, 6th, 7th, 8th, 9th, 10th, 11th and 12th Cranial nerves are perfectly healthy and unaffected in any way.

Cervical/
Cervical Sympathetic is unaffected.

Motor Functions
Sensory Functions
Reflexes
Vasomotor and Trophic functions

LOCOMOTORY SYSTEM.
---------------------

is normal. No affection of the bones or joints. Muscles are rather flaccid.

PROVISIONAL DIAGNOSIS.
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Chronic Nephritis (with Mitral Stenosis and Aortic stenosis, the latter in a slight degree).

TREATMENT.
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Rest in Bed.
Milk Diet.
Purgatives (Calomel 2 gr.)

PROGRESS.
---------

The effect of the treatment was quickly apparent for the oedema soon began to disappear and in a short time entirely cleared/
THE RESPONSE TO TREATMENT
cleared up. This improvement was accompanied by a corresponding improvement in the subjective symptoms. The urine increased in amount and albumen was reduced to a trace and the patient was discharged considerably improved.
COMMENTARY.

It is an uncommon occurrence for anybody to be afflicted with two serious organic diseases which are totally independent of each other and therefore one's first duty in a commentary on such a case as this is to attempt to correlate the two lesions which the patient presents and either to prove that one is dependent on the other or to give adequate proof that they are independent of each other.

Thus considering the two lesions, Chronic Nephritis and Valvular disease of the heart, it is well known that a degree of chronic nephritis may be set up by a cardiac lesion for, as a result of valvular imperfection, more especially mitral, general venous congestion is produced and maintained. Now it has been proved beyond doubt that this condition can produce a granular condition of the kidney with chronic interstitial fibrosis. But to produce such a lesion the cardiac condition must be of a very serious nature with marked general signs, in fact much more serious than our patient exhibits, for on examination she shows no signs of cyanosis, no evidence of congestion of the liver or other organs, and only slight hypertrophy of the heart. In addition she states that she can do a day's hard work, also walk a wood distance without any/
any undue degree of breathlessness.

All these facts point away from the heart as being the primary cause of the renal inadequacy.

Chronic Nephritis is often primary to a cardiac lesion because the defective excretory powers of the kidney and the consequent retention of toxic products cause first a hypertonicity then a thickening of the arterial system. The increased work now thrown upon the heart to drive the same amount of blood through the narrowed vessels causes it to hypertrophy and, if it is unable to do so sufficiently, to maintain an efficient circulation, dilatation will result and valvular defects will become manifest.

To quote Professor Rose Bradford - "In some instances the cardiac enlargement leads to valvular defects such as relative mitral stenosis with a distinct presystolic murmur as the result of hypertrophy and dilatation of the left ventricle, or mitral regurgitation with oedema of cardiac type, especially marked in the lower limbs." (Chronic Nephritis by Prof. Rose Bradford in Allbutt's System of Medicine pp. 614-615)

In our patient's case it would be the former lesion that resulted and the patient's symptoms do not negative this hypothesis.

With/
With regard to the view that the two lesions were of independent origin, it has been proved that the lesion of the kidney is not dependent upon that in the heart, so that all that is needed is the proof that the cardiac lesion could be of primary origin. Now the commonest cause of endocarditis is Rheumatic fever and on this point the patient's history is uncertain. She says that she suffered very seriously from "growing pains" and this history always makes one suspicious of an unrecognised attack of Rheumatic fever.

Secondly it will have been noted from the history that the patient suffered for four years from a severe form of Chorea, and during one period of this attack she lost her voice completely and was for some weeks an inmate of the Royal Infirmary. This condition, it is now well recognised, is very frequently followed by endocarditis and in many of those cases which have proved fatal the organism which probably causes rheumatic fever, namely the micrococcus Rheumaticus of Poynton and Payne, has been isolated.

These facts together constitute a fairly strong case for the independent origin of the cardiac lesion, but they do not contradict the statements previously made that the cardiac lesion might be dependent upon the renal lesion, for it must be construed that the organismal infection which the woman/
woman had, probably attacked the endocardium, leaving the patient with a damaged heart and that when the kidneys subsequently developed an inadequacy the heart was less able to compensate for this than it normally would have been able to do, and consequently its damage was aggravated.

Thus, in conclusion, it has been demonstrated that the lesion of the kidneys is primary and independent of the cardiac lesion, while the cardiac lesion is probably also primary but has been aggravated by the renal condition.

The question now arises, "are both these lesions to be regarded as equally serious or is one relatively more important?"

At an earlier stage in the commentary facts were brought forward to show that the signs of heart failure were insignificant and that, indeed, the compensation on the part of the heart was very good. Hence the cardiac lesion must be regarded as subsidiary in importance to the renal lesion and the next part of the commentary will be devoted chiefly to a consideration of the latter condition.

THE MORE EXACT DIAGNOSIS OF THE RENAL CONDITION.

From the patient's somewhat disconnected lesion it is apparent that she has had some defect of the kidney for at least five years, and the statement that each step in the aggravation of the symptoms was dated to the birth of a child/
child is very typical. Thus one is obviously dealing with a chronic condition and the clinical classification of chronic lesions of the kidney is into Chronic Parenchymatous and Chronic Interstitial Nephritis. That these types cannot be placed in water-tight compartments is made obvious on studying this case, for its symptoms and signs do not completely conform with either.

McLean and Russel in an article on the subject tabulate the main points to be observed in coming to a conclusion as to which type is present (Lancet. Vol.1. 1920, p.1305) and it would be well to repeat this.

<table>
<thead>
<tr>
<th>Chronic Interstitial</th>
<th>Chronic Parenchymatous</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>I.</strong> Urine-Quantity.</td>
<td>Increased.</td>
</tr>
<tr>
<td>Sp. Gravity.</td>
<td>Diminished</td>
</tr>
<tr>
<td>Chlorides</td>
<td>Unaffected.</td>
</tr>
<tr>
<td>Protein</td>
<td>Not present or a trace</td>
</tr>
<tr>
<td><strong>II.</strong> Oedema</td>
<td>Little tendency</td>
</tr>
<tr>
<td><strong>III.</strong> Cardiovascular changes</td>
<td>Marked</td>
</tr>
<tr>
<td><strong>IV.</strong> Urea concentra-</td>
<td>Diminished</td>
</tr>
<tr>
<td>tion.</td>
<td></td>
</tr>
<tr>
<td><strong>V.</strong> Blood Urea</td>
<td>Increased</td>
</tr>
<tr>
<td><strong>VI.</strong> Tendency to</td>
<td>Pronounced</td>
</tr>
<tr>
<td>Uraemia.</td>
<td></td>
</tr>
</tbody>
</table>
Now on turning to our patient's symptoms difficulty confronts us - the quantity of urine passed per day is diminished and there is marked oedema, so one naturally inclines to the belief that one has to deal with a lesion affecting the parenchyma. But there is merely a trace of albumen in the urine and the cardiovascular changes are marked as indicated by the cardiac lesions and the thickening of the arterial wall. These latter facts are more indicative of an interstitial lesion. Further observation however would minimise the value of the latter two statements because a sufficient cause for the cardiac lesion has already been brought forward and though the wall of the artery does seem to be thickened the blood pressure as estimated by the Riva. Rocei sphygmomanometer is not unduly increased, the systolic pressure being 126 mm.

With regard to the question of uraemia the patient has bad headaches frequently, however she is slightly myopic and refuses to wear glasses so this may be a sufficient cause. She states that on waking on a morning her vision is dim but on ophthalmoscopic examination of the optic discs no change can be found. Occasionally she vomits and occasionally has paroxysmal attacks of breathlessness. These facts point to a degree of uraemia which is not marked however, and lend little/
little help in the classification of the condition.

Taking all the facts together the main lesion would appear to be in the parenchyma but it is evident that there is also a certain amount of Chronic Interstitial Nephritis also. The history that on previous occasions the patient has had to get up in the night to pass water would justify us in assuming that this was the case and that the present attack is more of the nature of a subacute parenchymatous lesion super-imposed upon a chronic lesion of the parenchyma and interstitial tissue.

THE ETIOLOGY OF THE LESION.

Nephritis has been attributed to a large number of factors, some acting directly on the kidney and others on the blood vessels.

Chronic nephritis is usually the result of acute nephritis and from our patient's history she would appear to have had an acute attack about five years ago, after the birth of her first child.

Causes or alleged causes of nephritis can be divided into predisposing and exciting and one must now endeavour to find if any of these are present in our patient.

Among the predisposing causes the following are the most/
most important.

(a) Climate - frequent exposure to damp and cold. It is quite possible that the congestion due to the cold might light up an old standing nephritis (Nestor Tirad) or throw strain upon a weak kidney. In our patient the part played by this is doubtful. She has lived in Edinburgh all her life but has never been unduly exposed to the inclemencies of the weather which are so characteristic of the Edinburgh climate.

(b) Alcoholic Intemperance.

(c) Mental or nervous exhaustion.

In our patient both these can be excluded.

(d) Heredity.

This raises a very interesting point in the commentary. The patient's memory can go back as far as her grandparents, two of whom she is certain suffered from chronic Bright's disease. In addition her father also has Bright's disease and finally we are told that her son aged six years has swelling beneath the eyes every morning, so this makes a complete picture of four generations, and very strongly supports the views of those who so firmly believe that heredity is a most important factor in the cause of Nephritis.

Coming to the exciting causes the following are worthy of mention.-

(a)/
(a) Chill - the effect of this has been commented on.
(b) Acute fevers - in childhood patient had measles and whooping cough but too much stress should not be laid upon this.
(c) Chronic Intoxications - there is no history or signs of such in our patient.
(d) Organisms. Day and Clark working on this subject isolated various organisms, more particularly the Staphylococcus Aureus, Streptococcus and E. Coli. In addition they injected them into animals and produced an identical lesion in the animals and from their urine recovered the same organisms and by means of an autogenous vaccine caused great amelioration of the condition (Day and Clark - Lancet 1920. 2. p. 550). Their work though very interesting still needs following up. In our patient no organisms were isolated.
(e) Pregnancy. In our patient this was probably the exciting cause, for the symptoms date back to the first child. It is probable that the pregnancy caused a great deal more work to be done by kidneys already weak through hereditary causes and proved too much for them, and that when the kidneys were picking up again afterwards, another pregnancy broke down their resistance again and aggravated the condition.
MORBID ANATOMY.

This is so well known that it does not require detailed study and can be dismissed in a few words as no useful purpose would be served by enlarging upon it.

Suffice it to say that the kidneys would probably be found to be small in size - the cortex showing marked fatty changes and the capsule perhaps thickened.

Microscopically the tubules would be found to be distended and the epithelium showing fatty and degenerative changes.

The glomeruli would be enlarged, their capsules thickened.

Interstitial change would be found also in various parts of the kidneys and the vessels would show thickening or hyaline change.

PROGNOSIS.

This being one of the most important parts of the commentary needs more detailed study. The progress of Chronic Nephritis alone is somewhat of the nature of a guess, but when complicated by a cardiac lesion it is even more difficult. It has been shown that the cardiac lesion is not of itself of serious/
serious import so our attention will chiefly be turned to that of the kidney.

Now the whole question of prognosis as far as a renal condition is concerned, evolves from our knowledge of the amount of damage which the renal function has received and to estimate this the main functions of the kidney must first be known. Following McLean's classification these may be said to be four in number.—

I. Removal of certain waste products of intragenous metabolism.

II. Removal of acid products from the body.

III. Maintenance of an optimum concentration of salts in the fluids and tissues.

IV. Excretion of toxic materials artificially introduced.

Disease of the kidney may affect any or all of these functions, and a sound classification of kidney disease would be according to which function was chiefly affected. Thus in cases of Chronic Parenchymatous nephritis where oedema is prominent and the function of the kidney controlling the secretion of salts and maintenance of an optimum concentration in the tissues and fluids is out of gear the name of Hydremic chronic nephritis is given. While in the interstitial variety where/
where excretion of waste nitrogenous matter is the chief
defect the name Azotaemic nephritis is given.
Each type carried with it its own prognosis.

Thus to come down from the general to the special we
have to find out to what extent this patient's kidney is
damaged, and this has to be done by estimating the value of
each of the kidney functions. Many tests have been devised
for testing this, some good, some bad and some indifferent.
Out of these we will select three which have proved generally
to be the most useful, namely:

(a) Estimation of Blood Urea.
(b) Estimation of Urea concentration.
(c) Estimation of excretion of Phenol-sulphone-phthalein.

(a) Blood urea.

Normally the blood contains a certain amount of urea
varying from 15 - 30 mgs. per 100 ccs. If the amount rises
much above that figure we may be fairly certain that the
kidneys are inefficient if such gross conditions as acute
vomiting and diarrhoea can be excluded. McLean is however
of the opinion that the test is only of use in very pronounced
cases and if positive indicates a very serious condition of
the kidney (i.e. if the Blood urea rises to 40-50 mgs.
per 100cc.) The rationale of the test is explained on the
accompanying/
accompanying diagram (see appendix). In our patient the amount was found to be 28 mgs. so that we can say with a degree of certainty that the damage to the kidney is not of an extreme nature.

(b) The Urea Concentration test.

De Wesselow working in conjunction with McLean introduced this test (Quart. Journal of Medicine 1919 - 12 - 347) and is believed by them to be more accurate than the Blood urea test and to indicate renal deficiency when the former does not. The test is very simple and depends on the fact that a patient with renal inadequacy, when given large quantities of urea is incapable of excreting them in normal concentration in proportion to the damage sustained by the kidney. 15 gr. are given by the mouth and two samples taken, one after each hour to nullify any diuretic effect of the urea.

The patient should excrete it in a concentration of about 3%; if much below this inadequacy is present. In this patient the test was performed and the concentration was found to be 2.2%, thus indicating a slight deficiency.

The urea is estimated by a modification of Gerrard's process with hypobromide solution.

(c)/
(c) Phenol-sulphane-phthalein test.

This is a dye which is completely and rapidly eliminated without chemical change by healthy kidneys, producing in alkaline solution a brilliant red colour which can be estimated by a colorimeter. The details of procedure are explained on the accompanying chart (see appendix). Normally 60% is excreted in the first hour and 25% in the second, making a total of 85% in the first two hours. This patient when tested was found to excrete 40% in the first hour and 30% in the second, thus making a total of 70% and again a slight degree of deficiency is indicated though it must be said that apparently healthy people may under certain conditions excrete a little and too much stress should not be laid upon it.

Many other good tests may be carried out, such as the urea concentration factor (i.e. mgms per 100 ccs urine - mgms per 100 ccs. blood) which should be over unity and in our patient was 1.3.

The diastatic activity of the urine may be estimated and is a good index. The estimation of chlorides is not necessary for the amount of oedema forms the best index for that.

In summing up we may say that our patient has at present no serious renal inadequacy though she shows indications of commencing insufficiency, for it must be remembered that the kidneys/
kidneys are 400% strong and to show any deficiency they must be cut down to the extent of at least one half their normal value. This fact must be appreciated to get any useful idea of prognosis. But taken in conjunction with the long history and the fact that the patient is not seriously incapacitated and can still do a hard day's work, gives the patient a much better outlook than the clinical features alone would lead one to suppose.

The cardiac lesion is an aggravating factor and makes the prognosis more guarded, but even with that it can be said with a fair degree of confidence that the patient will get over her present attack and enjoy some years of moderately good health provided that she takes care of herself and avoids aggravating factors such as cold, damp and irritating nitrogenous food. Another pregnancy would severely impair the prognosis. The fact that no albuminuric retinitis is present is of good prognostic significance.

As it can safely be assumed that the patient will not rigorously carry out the good advice which will be given to her, it is probable that she will have more of these subacute attacks, each getting worse than its predecessor and impairing the renal function more, until finally the kidneys will become insufficient to carry on vital functions. Whether the heart will give out before this is difficult to say.
TREATMENT.

The treatment of the present subacute attack is exactly similar to that of simple Acute Nephritis - namely diminishing the call upon the excretory power of the kidneys by rest in bed, a milk diet or a nitrogen free diet, and helping elimination by other channels. The chief problem arises however in regard to the chronic condition when the present attack has settled down.

The same broad principle is aimed at but it is quite obvious that the patient cannot or will not live the remainder of her life on a nitrogen-free diet. Nor is this desirable, for it has been shown during the course of the commentary that this is not the function which is affected; it is rather the elimination of salts and the maintenance of an optimum concentration of salts in the tissues and fluids. Epstein was the first to point out the fallacy of this line of treatment and he demonstrated that a diet which contains a moderate amount of protein relieved the oedema and the symptoms far quicker than a rigid milk diet. (Epstein - American Journal of Med: 1917 - 635) and clinicians are gradually coming round to his beliefs.

Epstein's explanation of the improvement was that the plasma content of protein was increased, causing a greater osmotic/
osmotic pressure to be exerted in the circulatory system, thus attracting fluid from the tissues. McLean doubts this and thinks that the beneficial action is due to the diuretic action of the urea which is naturally greatly increased by this diet, and supports his argument my getting the same result after treatment with urea alone.

Whatever the reason, Epstein's diet undoubtedly does a great deal of good though it does not reduce the albuminuria. The fact that the patient is much more happy on such a diet should perhaps be pointed out. Epstein's diet consists of the following proportions:

1. Total in calories 1280 - 2500
2. Protein - 120 - 240 grams.
3. Fat (unavoidable) 20 - 40 grams.
4. Carbohydrate 150 - 300 grams.
5. Fluid 1200 - 1800 ccs.

It can be recommended somewhat as follows.

Breakfast:- Porridge made from 2 oz dry oatmeal.
Lean bacon 3 oz. Toast and tea.

Dinner:- Vegetable soup or 3 oz. of fish, flesh or fowl.
3 oz. potato.
Milk pudding or fruit.

Tea: - /
Tea:— Tea or coffee. 2 oz. bread and butter. Jam.

Supper:— Similar.

The following should be excluded altogether—peas; beans, meat extracts, onions, rhubarb, alcohol, also tobacco, as containing too much nitrogen or being irritant to the kidneys. Salt should be cut down to a minimum.

Otherwise the treatment is on the lines of common sense—plenty of fresh air but avoiding chills and damp, and careful living.

Drugs play a secondary part—a periodic purge is useful—diuretics and tonics each have their sphere of usefulness though the former are used with caution. The patient is anaemic so that an iron tonic would certainly be useful.

The heart condition at present is not serious enough to require cardiac tonics, and the treatment of it is to avoid straining the damaged heart.

CONCLUSION.

In conclusion only two points are worthy of mention. The first is well illustrated by this case and that is that the clinical appearances of a case of nephritis may lead one very much astray in judging the damage sustained by the kidneys. The/
The only rational way of finding this is to estimate the renal function by one or all of the ways mentioned and many serious mistakes with regard to prognosis will be avoided.

The second point is that which has been brought out under the heading of treatment - namely the value of a diet such as that recommended by Epstein which is efficient in clearing up the gross physical signs and certainly adds much to the comfort of the patient. It is probable that the lives of cases of Chronic Parenchymatous Nephritis are cut short rather than improved by a continuous regime of an unsustaining diet and a modified nitrogenous diet improves such cases to an extraordinary extent both physically and mentally.

A. Container
B. Blood
C. 25 cc. N/100 H$_2$SO$_4$
(A) 5% H₂SO₄  (B) Blood 10/25ccs  Nₐ₀₀ H₂SO₄
A. 5 ccs Acid Sodium Phosphate are measured into the blood tube.
3 ccs Blood are added to the Sodium Phosphate.
6-8 drops Caprylic Alcohol are then added.
Finally .3 grm. Soya bean meal.
Replace stopper and close tube by clip.
Place tube in a bath - temperature 40 - 45° C. for 10 min.
Meanwhile add 25 ccs. N/100 H₂SO₄ to tube C and 1 drop Caprylic alcohol and 2 drops Methyl Red.

B. When hydrolysis is complete tube B is removed from the bath and connected with tube A containing 5½ H₂SO₄
and with tube C containing 25 cc. N/100 H₂SO₄.
Open the clips and draw air through the whole system for 2 minutes, by means of a suction pump.
Tube A removes any ammonia from the air.
Tube C takes any ammonia carried away from tube B.

C. After 2 minutes the Suction is stopped and the Blood tube B is opened and 4 ccs. saturated Potassium Carbonate are added and 3 gms. Anhydrous Potassium Carbonate, the stopper replaced and a current of air drawn through, the time required to remove the ammonia being 30 min.

D. The tube containing the standard is disconnected and the acid transferred to a small Erlenmeyer flask and titrated with N/100 A₈OH.
E. **Calculation.**

The difference between the 25 ccs. acid taken and the number of ccs. of Alkali used give the number of ccs. neutralised by Ammonia.

Subtract .4 cc. for traces generated by Soya Bean.

Each cc. neutralised = 10 mgms urea per 100 cc. Blood.

---

II. **The Phenol-sulphoric-phthalein Test.**

**Solution.**

\[
\begin{align*}
R & \quad \text{R} \\
\text{Phenol-sulphoric-phthalein} & \quad 0.6 \text{ gm.} \\
2 \text{ N. Sod. Hydrox.} & \quad 0.84 \text{ cc.} \\
\text{.75% solution Sod. Chlor. ad} & \quad 100 \text{ cc.}
\end{align*}
\]

**Procedure.**

(a) Patient is given a glass of water half an hour before.
(b) The Bladder is emptied.
(c) 1 cc. solution is injected into the lumbar region.
   Allow 10 min. for beginning of excretion.
(d) Then 1 hour later collect the urine (Sample I)
   Repeat after another hour. (Sample II)
(e) To sample I add 10 ccs. 10% Sod. Hydroxide solution
   and dilute to a given volume with tap water, and compare
   with/
with a standard solution prepared by introducing 1 cc. of Phenol-sulphone-phthalein and 10 cc. of 10% Na OH into a 1000 cc. flask and filling with water. To compare the colours some type of colorimeter such as the Dubosque is used. From the results the percentage can easily be calculated.

Normal Exertion.

1st Hour.  40 - 60 per cent.
2nd Hour.  20 - 25
Total.  60 - 80

III. Method for determining the amount of urea present in the urea for the Urea-concentration test.
Apparatus.

1. A 50 cc. graduated burette (A) which has a glass tap (R) fixed at its upper end.

2. Connected by rubber tubing (E) with a bottle (D) containing the urine and hypobromite solution which consists of 23 ccs. of a 40% sodium hydroxide to which 2 cc. bromide are added.

3. The lower end of the burette is attached by rubber tubing to a small bell shaped vessel (M) to hold the water displaced by the nitrogen.

Technique.

(a) Water is poured into M until three quarters full seeing that no air bubbles are in the tube.

(b) 4 ccs. urine are measured into the tube K 25 ccs hypobromide are placed in the bottle D.

(c) Open the tap R and place the rubber stopper tightly in the bottle D.

(d) Raise the bulb M until the water in the burette is at zero, close the tap and replace bulb.

(e) Thoroughly mix the two solutions and after a minute raise the bulb M so that the fluid is at the same level.

(f) Read off the number of ccs. of gas evolved; from this the amount of urea is calculated from a standardised table.