RAJAH OF PANAGAL, Kt.

THE LATE RAJA OF PANAGAL.

In the death of the Raja of Panagal, on the 16th December last India has lost one of the greatest statesmen of modern times. He was in apparent good health till the week previous to his death, when an atttack of Pneumonia of a very severe type carried him away inspite of the best medical assistance.

The career of the Raja of Panagal marks the political progress of Southern India after the reforms, for he was at the helm of the administration on the transferred side for the first six years during which rapid strides were made in various departments of which Medicine and Public Health constitute the most important. If there be any one personality to whom our service owes more than to any other, it is to this far-sighted administrator. The liberal measures inaugurated during the regimes of Sir Alexander Cardew and Sir P. Rajagopalachariar reached their practical fruition during the administration of the Raja of Panagal. With his keen insight, sobriety of judgment, and sympathy with Indian aspirations, he effectively drew successive Surgeon-Generals to his side and with his remarkable capacity of making the Government see eye to eye with him and convincing them of the soundness of his views he succeeded in carrying his ideals into practice more effectively than any other statesmen would have done under the critical conditions of the first few years of the new administration under the Reforms.

One of the finest traits of the late Raja of Panagal's administration was the great keenness he always evinced in grasping the details of the administration in all its branches especially Medicine and Public Health so much so that Medical men who met him in personal discussion were often surprised at the mastery of several medical questions possessed by a layman of his type. It was due to this genius of grasping professional subjects that the series of reforms in the Medical administration were effected in the minimum of time

and with the maximum of benefit to this Presidency. The Indianisation of the Medical and Public health services, the large increase in the cadre of Civil Surgeons, the throwing open of the Professorships and other higher appointments to the deserving members of the Provincial service, the rapid strides in the expansion of Medical education by the opening of the Vizagapatam Medical College and the Medical Schools in different parts of the Presidency, the institution of rural dispensaries all over Southern India, throwing open to the villages the benefits of mordern medicine and surgery, the recognition of the importance of specialisation in various branches of Medicine and Public Health by the appointment of specialists, the free facilities given to members of the service in specialisation by the systematic and liberal grant of study leave and deputation leave, constitute the prominent features of his medical administration. The Indigenous systems of medicine did not escape his fostering care and attention. The Government school of Indian Medicine rapidly sprang out of his creative genius in the face of a certain amount of official and non-official opposition.

Courteous to a fault and always anxious to meet every type of representation he has never been known to refuse an interview however unreasonable the applicant may have been considered to be by the public. The sobriety of his judgment, the remarkable patience and perseverence with which he handled difficult questions the noble qualities of the heart which kept him uniformly balanced in temper in his dealings with his adversaries either in private or in public life mark the late Raja of Panagal as one of the finest types of really great and good men that has ever played a prominent part in the administration of Southern India. His life and work will ever remain a beacon light to his successors in public life and the footprints he has left behind him on the sands of time ought to be a true guide to every sincere patriot and worker in the cause of this country.

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CEREBRAL APOPLEXY

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The term Apoplexy or "Stroke", though it is applied to several distinct pathological conditions, is sometimes used to indicate a sudden loss of consciousness due to a vascular lesion—hæmorrhage, embolism, or thrombosis within the skull. Apoplectic conditions at different seats, centres or according to cause are called by their respective names.

Bulbar (that due to a rupture of a blood vessel in the medulla oblongata).

Capillary (that due to punctiform hæmorrhages in the grey matter of the brain).

Ingravescent (that applied to large effusious developing slowly, *i.e.*, for a period of several hours or for a day or two and this is observed in hæmorrhage at the external capsule).

Pontine (that due to a rupture of a blood vessel in the pons varoli).

Symmetrical (that due to a double hæmorrhage, starting from corresponding points of the two hemispheres).

Meningeal and Ventricular (those due to hæmorrhages at these sites as such or starting from vessels in

the brain substance and their rupture through into one or the other of these spaces).

Traumatic (that due to violence or injury, in contrast to the general run of spontaneous cases).

Pulmonary (that due to escape of blood into the pulmonary parenchyma).

Simple (that found in cases of death from Coma in which no cerebral lesion is found).

Spinal (that due to a rupture of a blood vessel of the spinal cord).

Splenic (that due to a flow of blood into the splenic substance).

Cerebellar and so on.—The subject of this paper, *viz.*, *Cerebral Apoplexy*, indicates the condition of a sudden loss of consciousness due to a lesion within the skull either from hæmorrhage, thrombosis or embolism. As this will cover a vast space I shall confine myself to Cerebral Hæmorrhage touching merely here and there cerebral thrombosis and cerebral embolism. Hemiplegia and other conditions will also be considered in this connection synonyms for Cerebral Apoplexy Sanguineous Apoplexy, Cerebral Hæmorrhage Apoplexy.

Anatomy and Physiology.

The two essential points for study in such a widespread structure like the nervous system are (1) *Localisation of the seat of mischief.* Is it for instance in the brain, cord, nerves or autonomic system ? and (2) the diagnosis of the nature of the lesion. The necessity of localising a nervous disease before diagnosing its nature will entail an accurate knowledge of the anatomy and physiology of the nervous system.

The nervous system comprising the brain, brain stem, spinal cord, and peripheral nerves and the autonomic system is the mechanism whereby the various motor and sensory functions are co-ordinated and the individual is enabled to react to the environment as a unit. The integrative function is subserved (assisted or promoted) by a complex system of neurons. Each Neuron consists of (1) Cell-body—vital centre ; (2) Axis cylinder process—collaterals and arborisations ; (3) Dendrons ; (4) Neuroglia.

The Neurons are efferent and afferent and the terminals of the axis cylinder process of one neuron are related to the dendrites and cell bodies of other neurons by contact forming synapses. The function of the neuron is to conduct nervous impulses !

Cell systems. The cell bodies of the neurons are collected more or less closely together in the grey matter of the brain and spinal cord and in the ganglia of the peripheral nerves. Their processes, especially the axis cylinder processes run for the most part in the white tracts of the brain and spinal cord and in the peripheral nerves. In this way the different parts of the central nervous system are brought into relation with each other and with the rest of the body.

Brain.

Looked at in its simplest form the Cerebrum consists of a mass of white fibres (the corona radiata) spreading out towards the grey cortex. Unlike the spinal cord the grey matter of the brain is found chiefly upon its surface. But in the interior at its lower part there are three conspicuous masses of grey matter which from before backwards are the *corpus striatum, the optic thalamus* and the *corpora quadrigemina.* The corpus striatum

is divided into two grey masses (the caudate nucleus internally and the lenticular nucleus externally) by a most important band of white matter, the internal capsule which carries the conducting strands from one side of the brain to the opposite side of the body.—Internal to the lenticular nucleus comes the anterior end of the optic thalamus.

Medulla and Spinal Cord.

Those parts of the bulb and the pons Varolii which form the floor of the fourth ventricle and the iter a tertio ad quartum ventriculum contain a series of grey nuclei which give origin to the cranial nerves from the third to the twelfth inclusive.

These form as it were, a continuation upwards of the anterior and posterior horns of grey matter of the spinal cord.

If we imagine the spinal cord to be split from behind and as it is traced upwards to be opened outwards so that the posterior grey cornuæ come to be external and the anterior columns come to the surface beside the middle line, we should find that is actually the case, that the motor nucelii of the twelfth, eleventh, seventh, sixth, and the fifth cranial nerves (motor nuclei corresponding to the anterior horns) lie immediately on each side of the middle line and that the sensory nuclei of the tenth ninth, eighth and fifth nerves (corresponding to the posterior horns) lie in a more external situation. The nucleii of the other motor nerves (fourth and third) lie farther forward beneath the Iter.

The white matter of the spinal cord contains long ascending and descending tracts and shorter ascending and descending association fibres.

The Motor Tract.

It is important to remember that a motor impulse passing from the cerebral cortex to the periphery must pass through at least two neurons, brought into relation with one another by means of an " intercelary " neuron.

A motor impulse starting in the motor area of the cortex passes through the corona radiata in the central white matter of the brain through the anterior two-thirds of the hind limb of the internal capsule, through the middle two-fifths of the anterior aspect of the crus cerebri of the same side, through the pons in a band lying between the superficial and deep transverse fibres and through the anterior pyramids of the medulla. Here the bulk of the motor fibres cross to the opposite side to form the crossed pyramidal tract in the lateral columns of the cord. At the point of decussation in the medulla a few of the motor neuroxons instead of crossing over pass down the same side of the cord in the anterior column close to the anterior fissure forming the direct pyramidal tract, the fibres of which all eventually decussate. The neuroxons of the lower motor neurons (spino-muscular level) pass out through the anterior nerve roots or the cranial nerves into the peripheral nerve trunks and terminate in the muscles.

The fillet. The cells of the posterior column nuclei are of moderate size and their axons pass as *internal arcuate fibres* into the reticular formation between the two olivary bodies, as the Inter olivary layer. They cross the median raphe dorsal to the pyramids and then turn upwards towards the upper parts of the brain, and so constitute what is known as the Fillet or Lemniscus. In the higher parts of the bulbs and pons, the tract is

reinforced by fibres from the cells of the sensory nucleii of the cerebral nerves. The fillet becomes a longitudinal bundle, which passes upwards to the thalamus, which forms the next cell-station on the pack of the sensory impulses to the cortex and this passes into the tegmentum. In the mid-brain the fillet splits into three bundles termed the lateral, the upper and the mesial fillet.

1. The lateral fillet is chiefly formed by fibres derived from the accessory, acoustic, the inferior olivary and the trapezoid nuclei of the opposite side. Some of its fibres terminate by synapses around a new collection of cells (the lateral fillet nucleus); their axons pass inwards towards the raphe. The rest of its fibres can be traced to the grey matter of the Inferior corpora Quadrigemina.

2. The upper fillet consists of fibres which go to the Superior Quadrigamina and partly to the tegmental region of the mid-brain and Optic thalamus.

3. The Mesial fillet goes on through the tegmentum of the pedunculus cerebri, and its fibres terminate around the cells of the thalamus, and the Subthalmic region. From here fresh axons forming a new relay continue the afferent impulses to the cortex of the cerebrum.

The Sensory Path.

1. The lower or peripheral neuron has its cells in the ganglia of the posterior nerve root. These cells give off an apparently single process which shortly divides like a T into two limbs, one of which corresponds physiologically to an elongated dendron, communicating with the peripheral sense organ, and the other which forms the axon, passing into the cord. These axonic

processes enter the cord in two bundles of which the Mesial bundle passes into the posterior column and the lateral into the marginal tract of Lissaur.

The processes of both bundles divide on entering the cord into ascending and descending branches, both of which, give off collaterals to various cells of the grey matter.

The descending branches run a short course, some of them in the comma tract of the posterior column and then turn into the grey matter.

The ascending branch may end in the grey matter soon after entering or it may run in the dorsal fasciculi as far as the medulla to end about the nuclei there. In any case, it does not cross the middle line. The lower sensory neuron is direct.

2. *The central Neruons* are composed of two orders of neurons one above the other *and are crossed.* The second order of neurons commences partly in the cord and partly in the medulla oblongata. After the decussation, the two sets run together in the median fillet to the optic thalamus.

3. From the optic thalamus, a third order of neurons carries by means of its axons the impressions derived from the second order, through the posterior third of the posterior limb of the internal capsule and the corona radiata, to the cortical sensory centres in the post central gyrus of the parietal lobe.

The internal capsule, is, the knee shaped bend of white matter which is bounded on its outer side by the lenticular nucleus and on its inner side by the optic

thalamus and the candate nucleus. The motor fibres occupy the anterior two-thirds of the posterior limb of the internal capsule.

In the bend from before back are fibres for eyes, head, tongue and month.

In the posterior limb are fibres for the arm, hand, trunk, hip, knee, leg, toes.

The posterior one third of the posterior limb contains sensory fibres and visual fibres.

The anterior limb contains commissural fibres of various kinds.

The *Arteries* of the brain are derived from the internal carotids and vertebrals, which form the circle of willis. The middle cerebral is the most important artery of the brain. The middle cerebrel or Sylvian arteries are the largest branches of the internal carotid, and pass upwards and outwards in the fissure of Sylvius till they reach the surface of the island of Reil, where they ramify in the piamater, forming part of the cortical system of arteries. These branches supply the under and outer surfaces of the frontal lobe (in part) and the ascending frontal convolution, the outer surface of the parietal lobe and most of the outer surface of the temporal lobe. They anastomose freely with the anterior and posterior cerebral arteries. Other branches furnished through the anterior perforated spot to the corpus striatum, are all terminal arteries. They are (1) the lenticular; (2) the lenticulo-striate; (3) the lenticulo-optic. The lenticulo striate arteries are the most frequently ruptured in cerebral hæmorrhage and this was called by Charcot, as the artery of cerebral hæmorrhage. (The posterior olfactory lobule or anterior perforated space is

situated at the commencement of the fissure of Sylvius and bounded posteriorly by the optic tract.)

Cerebral veins.—There are two sets—a superficial set and a deep one.

The special characters of the cerebral circulation are :—

1. The free anastomosis at the circle of Willis, which provides a ready supply of blood from other vessels in case of the sudden blocking of any of the more direct channels.

2. The tortuous course through bony canals of the arteries as they enter the skull, thus mitigating the force of the heart's beat.

3. Their ramifications in the piamater before entering the substance of the brain.

4. The thinness of the arterial walls and the smallness of the capillaries.

5. The existence of venous sinuses which are without valves and which do not run with the arteries.

The three cerebral arteries pass vertically into the nerve substance and do not anastamose with each other or with the arteries of the cortical system.

The external capsule is that portion of the cerebrum lying between the claustrum externally and the lenticular nucleus internally. It is in close proximity to the Island of Reil.

Predisposing Causes of Cerebral Hæmorrhage.

1. It occurs in a certain type of build. It is frequent in stout, plethoric men, with short thick necks.

2. (*i*) Certain occupations among which are those of butchers and publicans, owing to arterial changes produced by excessive consumption of nitrogenous food and alcohol.

(*ii*) Carters, hammermen, etc., as the result of vascular strain.

3. Certain blood diseases—Leukaemia, pernicious anaemia, scurvy.

4. Vascular degeneration due to chronic renal disease, the rupture being aided by the forcible pulsations resulting from cardiac hypertrophy. Syphilitic degeneration may lead to hæmorrhage, but more frequently causes thrombosis.

5. Removal of the natural support of the vessels, as in cerebral softening.

6. Injuries from without the cranium, etc.

Exciting Causes.

The most important exciting causes are.—

Diseases of the vascular walls :

(*i.*) Miliary and other aneurysmal dilatations.

(*ii.*) Arterio-Sclerosis and atheroma, whether due to gout, renal disease, alcoholism or other causes. Independently of renal diseases, the blood-pressure tends to rise with advancing years.

(*iii.*) Vascular strain, as from exercise or causes such as straining at stool or violent coughing. It will be remembered that the brain expands during expiration and expiration with a closed glottis very greatly increases intracranial pressure.

Seats of Haemorrhage.

Scarcely any part of the brain is exempt from the risk of hæmorrhage but it is much more frequent at the base in the neighbourhood of the corpus striatum and optic thalamus which are mainly supplied by branches of the middle cerebral artery to which reference has already been made. Vessels may also burst in the lateral ventricles (ventricular hæmorrhage) ; or on the surface of the brain (meningeal hæmorrhage ; but when blood is found in these situations, it has often proceeded from a hæmorrhage primarily in the cerebral substance.

Anatomical changes, As to the blood thrown out.

There is less resistance to the overflow in the grey than in the white matter. It may vary in quantity from minute capillary extravasations up to those of several ounces. In the latter case, it tears up the cerebral tissue, destroying for instance, the great ganglia and the internal capsule and extending thence into the centrum ovale or it may burst through the optic thalamus or caudate nucleus into the lateral ventricle. Such cases are rapidly fatal and post mortem examination reveals a mass of dark red clot, filling the ventricle and occupying much of the hemisphere, surrounded by brain substance which is ragged and discoloured by blood. In cases which have lasted a few days there is the same dark-red clot and the tissue around is soft and discoloured yellow, from absorption of hæmoglobin (yellow softening).

The effect upon the parts of the brain not immediately destroyed by the hæmorrhage varies with the amount of effused blood and with the time which has elapsed since the hæmorrhage. If the clot is sufficiently large, it presses upon the sound cerebral tissue ; the

hemisphere in which it has taken place is enlarged ; the convolutions are flattened against the interior of the skull ; and the sulci are obliterated. After some hours or a few days, secondary oedema of the affected hemisphere may occur and the oedema may be so great that the healthy side of the brain may be compressed leading to Coma.

In later stages the clot becomes brown or brownish yellow, consisting of disintegrated blood and nerve structure ; and the surrounding tissue is frequently softened (white softening) and contains large mononuclear phagocytes full of granular debris. Finally, in patients who survive, the blood becomes absorbed and leaves a tawny or orange coloured spot, in which crystals of hæmatoidin can be found or a cyst may remain, containing serous fluid or a distinct, tough, fibrous scar, discoloured also by the remains of blood pigment.

There are finally, certain secondary changes of nerve tissue that may develop. These affect only such nerve fibres as have either been directly severed by the effusion or so much involved as to be unable to recover even their trophic and function. Then the portion of these neurons that have been cut off from their respective calls undergo degeneration the same as do severed fibres in peripheral nerves. In the case of the pyramidal or spinal motor tracts this degeneration may extend down the cord to the anterior horns ; but the terminal or spinal motor neurons being independent structures are not generally involved in this process. Of course fibres going to the other parts of the brain will degenerate in like manner if severed from their parent cells. While in the peripheral nervous system there may be a regeneration of severed or

degenerated fibres, nothing of the kind is known to occur in the central nervous system.

Course and symptoms.

Apoplexy. Cerebral apoplexy may be preceded for days or weeks by occasional giddiness, numbness or twitching of the fingers, headache, insommia, or some diminution of mental capacity ; but these are not so much indications of the severe attack to eome as evidences of existing disease of vessels, and perhaps due themselves to slight hæmorrhages. On the other hand, it may come on without any warning whatever. The attack may occur when the patient is resting asleep. Sometimes also it seems attributable to a definite cause, such as emotional excitement, muscular effort, violent coughing or straining at stool ; much more frequently it is directly attributable to an overloaded stomach or the exertion in running to catch a train. In numerous cases cerebral hæmorrhage causes a group of symptoms known as *Apoplexy*—that is, the patient is suddenly struck down unconscious, or he quickly becomes so. Patients have died in five or ten minutes from the first symptom. More often the symptoms come on slowly. The patient is seized with intense pain in the head, becomes faint or slightly collapsed, may be sick or have a slight convulsion, and then after half an hour or more, gradually sinks into a condition of coma. Or the first symptoms may show themselves in the motor system ; the patient Mumbles in his speech or his arm drops powerless and he gradually leans over to one side, falling if not supported, and then lapses by degrees into coma. Or the Coma may be developed in a few hours through stages of increasing drowsiness or the attack may begin with convulsions. In some cases, the collapse is

extreme and the pulse is barely perceptible ; but after an hour or two, it improves and becomes full and bounding as the patient assumes the condition described below with complete coma and paralysis.

When the patient is found by the friends alone, or is picked up in the street unconscious, or is unable to be aroused in the morning from sleep it is of course impossible to say what the onset has been. But, undoubtedly cerebral hæmorrhage may occur without apoplexy ; a very slight bleeding into the motor tract alone may give rise to paralysis without loss of consciousness, but this is uncommon.

The patient suffering from hæmorrhagic coma lies completely unconscious and cannot be aroused by shouting or any form of stimulation of his skin. The face is flushed and bloated, the pulse full and tense, the breathing stertorous and the cheeks puffed out. The snoring noise is due to the paralysed tongue and palate falling back and impeding the entrance of air. The pupils are dilated or irregular and do not respond to light. The limbs are at first flaccid, but may become rigid with the onset of reaction (so-called early rigidity). During the coma both the superficial and the deep reflexes are abolished ; later they return, first on the sound side and afterwards on the affected side where they are exaggerated. There are inability to swallow, retention or involuntary dribbling of urine, and involuntary evacuations. The head and eyes are frequently turned to one side, *i.e.*, the patient looks towards the lesion and away from the paralysed side (conjugate deviation) owing to the balancing action of the associated muscles being lost from paralysis. When the case is going to end fatally, the coma

increases, temperature falls, and Cheyne-Stokes respirations usher in the fatal end. In Pontine hæmorrhage, however, which is usually rapidly fatal, the temperature rises even to hyperpyrexia and the pupils are contracted. In favourable cases, the primary disturbance is not so grave, and the patient may regain consciousness in a few hours, or even after one or three days. " Reaction " then sets in, the temperature rises, as does the pulse, perspiration occurs and there is restlessness with excitement amounting sometimes to delirium. In this stage which may last from a few days to several weeks, trophic disturbances may occur, such as bed sores on the paralysed side and the case may end fatally but recovery is more common. It is rarely complete but usually attended by more or less paralysis or typical hemiplegia. In very severe cases the pulse and breathing are rapid, there is profuse sweeting and intense flushing of the face and skin generally then, after a time two to three hours or more, the patient becomes livid, rales occur in the larger bronchi and trachea the pulse gets weaker, the breathing slower and finally death takes place. The fatal termination may, however, be delayed for several days, during which the lungs are very apt to suffer from Oedema or pneumonia.

It is possible even in the comatose condition to make out the existence of paralysis. The cheek is more puffed out on the affected side, the naso labial groove is obliterated, and the angle of the mouth is lower, while saliva trickles from it. If the limbs on the two sides be raised alternately and then let fall, those on the paralysed side fall in an absolutely flaccid fashion, while upon the other side, some tone is maintained. The cremasteric and abdominal reflxes are absent on the paralysed side, on which Babinski's sign is often present. (When the

sole of the foot is stroked by a pointed instrument, the toes especially the great toe are over-extended towards the dorsum of the foot and abducted one from the other.

In Cortical Hæmorrhage.

1. Consciousness is seldom completely lost at the onset of paralysis.

2. The paralysis is of a monoplegic type.

3. The onset is attended with convulsions.

4. The prognosis is more favourable.

In a large proportion of cases, the patient is found to be suffering from hemiplegia, which may itself slowly recover or be permanent. The hemiplegia always results when the injury caused by the hæmorrhage has occurred to the internal capsule at the most common site, *i.e.*, in the vicinity of the corpus striatum, but hemiplegia follows injury to any portion of the motor tract, from the motor centres in the cortex of the brain to the upper part of the pons varolii. Certain peculiarities may however be noticed, depending upon the exact localisation of the damage whether it be (1) in cortex ; (2) Corona radiata ; (3) various points in the internal capsule ; (4) crus cerebri ; (5) pons; or (6) Medulla.

There is loss of power in the muscles of the face, arm and leg on the side of the body opposite to the brain lesion when the hemiplegia is said to be complete. It is obvious that other conditions than cerebral hæmorrhage produce identical symptoms, *viz.*, thrombosis, embolism, tumour, abscess, depressed fractures compression during birth, and menigeal hæmorrhage or inflammation.

The hemiplegia is said to be incomplete when only a leg or arm or the face or tongue is involved. This is

the case when the cortex or white substance of the heimsphere is involved. It is also termed *monoplegia.*

The paralysis of the face affects chiefly the lower zone, the upper region being only slightly weakened so that the patient can close his eyes and wrinkle his forehead-though he cannot blow out his cheeks, the angle of the mouth being drawn to the sound side. The tongue is usually affected ; when protruded, it deviates towards the paralysed side, being pushed over by the sound geniohyo-glossus muscle. The muscles of the trunk on the affected side are somewhat weakened, but never paralysed in the ordinary sense. This may be noticed in the inter-costals when the patient attempts a deep breath, and in the abdominal muscles when he coughs or attempts to sit up, though in ordinary quiet breathing the respiratory movements may be greater on the affected side. The explanat ion of the escape of the trunk muscles and the masseters is, that the muscles which are always bilaterally associated in their actions, as the intercostals of both sides are represented in both hemispheres (owing possibly to the existence of commissural fibres in the spinal or bulbar centres) while movements which are unilateral are only capable of being excited in one hemisphere, *i.e.*, that of the opposite side. This explains most of the features of hemiplegia. By it we can understand how the bilaterally associated movements of the upper part of the face escape obliteration ; and it also explains how the legs suffer less than the arms being much more closely associated with each other in action (as in walking) than the upper extremities are. It also affords a key to the explanation of the phenomena of double hemiplegia or pseudo-bulber paralysis ; in ordinary hemiplegia swallowing and articulation being

bilaterally associated movements, they escape, but when the hemiplegia is double, paralysis is marked as in true bulber paralysis. In cases of right sided hemiplegia, the hæmorrhage being into the left side of the brain, there is often aphasia, owing to interference of Broca's convolution; but in persons who are left handed, the aphasia may accompany hæmorrhage into the right hemisphere. Sensation is seldom as profoundly affected as motion, but complete hemi-anæsthesia is sometimes met with where the lesion affects the sensory path in the internal capsule, in the posterior third of its posterior limb. It may occur also to a limited extent in cortical lesions, and as a rule it is more complete in softening from emboli and thrombosis than in rupture of a vessel. The loss of sensation to touch or to pain may be scarcely observable and as a rule it is most marked in the extremities of the paralysed limbs and may be associated with hemianopia or hemianopsia, *i.e.*, blindness of one half of the visual field. As a rule both these rapidly pass away.

When the hæmorrhage is into the crus, medulla or pons, there may be paralysis of a cerebral nerve on the side opposite to the hemiplegia—a condition known as crossed hemiplegia.

Thus in lesions of the lower part of the pons the facial is damaged on the side opposite to the hemiplegia and in lesions of the crus the eye muscles opposite to the hemiplegia are involved. There is usually slight impairment of the muscles on the sound side in most cases of severe hemiplegia; this is generally most marked in the leg.

The paralysis of the face, arm, and leg may pass off rapidly in a few days if the damage has been very slight;

but as a rule weeks elapse before marked improvement sets in, even in the most hopeful cases. The leg recovers before the arm and the shoulder muscles before those of the hand, and it is not unusual to see a hemiplegic patient walking with his arm helplessly hanging by his side.

Recovery first begins in certain groups of muscles in the paralysed limbs; the facial muscles as a rule rapidly recover. The extensors of the foot and the knee-flexors are more deeply involved than the other leg muscles and they are more or less permanently phralysed, so that the toes cannot be raised above the ground, and progression is accomplished by a semi-circular movement of the leg. Recovery is slower and less complete in the hand and fingers, which often remain permanently paralysed.

In severe cases which do not recover, certain changes are generally observed, chief among which is rigidity. Rigidity may often be observed during the Coma—The so-called early rigidity is often noticed during the first week or two; it is caused by some irritation of pyramidal fibres or by an inflammatory action around the clot; it may pass into the late rigidity which is probably the result of the descending degeneration which follows the injury to the motor tract and it may last for years or pass into permanent structural contracture. In the upper extremity the spastic condition of late rigidity is seen by the adduction of the shoulder, flexion of the elbow and wrist, and of the finger into the palm; in the lower extremity the knee is straight, and the foot is in the position of equino-varus. The heel is drawn up and the anterior half of the foot adducted and drawn inwards. The muscles do not show wasting for some

weeks and then generally it is very slight and is owing to inaction. Their reactions remain for a long period but the deep reflexes are exaggerated. During the coma they are all abolished, but soon return after consciousness on the sound side when they are somewhat increased generally. Later on they appear in the affected limbs, where they are always much exaggerated after the end of the second week. On the affected side the knee-jerk is very excessive and ankle clonus is easily elicited and the great toe on tickling the sole is extended and a tap on the front of the wrist causes active flexion of the finger. The superficial reflexes on the weak side are greatly diminished. With the rigidity is often associated much pain, and though the flexion may be overcome by extension it immediately recurs. The contraction at first disappears during sleep, and may be removed by Esmarch's bandage ; later on when the structural contracture supervenes the joints become permanently immovable. Tremors, spasms, and irregular movements of various kinds occur chiefly in the arm in many of the cases which recover or partially recover.

So far we have considered the symptoms of cerebral Hæmorrhage, *viz.*, the Apoplexy and the paralysis. Coma which is the chief thing in an apopletic fit requires some further consideration and we will take under this heading *coma and other disturbances of consciousness.* These are of great importance for both the positive and the differential diagonosis. But at the same time they are matters most difficult to describe or define with exactness and in accordance with facts.

Coma is a state of profound unconsciousness not due to sleep, syncope, hypnosis, or drugs. In practice, we

meet with all kinds and degrees of disturbances of consciousness. The eyes may be open and staring, yet the person fails to make any response to our interrogations and evidently fails to have any understanding of language or surroundings. More often there is a condition of stupor that admits of but partial and temporary recognition. We can then conveniently distinguish Coma, stupor and dazed conditions.

The duration of these states is next in importance. They may be of such transitory nature as to pass unnoticed or they may last several hours or days the lighter degrees being of course, as a rule, of shorter duration. The time in the attack when coma supervenes is also to be noted ; if at the start it may be partly a shock-effect ; if later and more gradual it indicates that the effusion has reached a large volume.

The size of the out-put requisite to produce this symptom varies much with its location. A small clot in the pons, for instance, will produce a much deeper impression on consciousness than one of far greater size in the hemispheres. It has been shown that the involvement of the sensory tracts has little or no influence on consciousness, while other cases with equal sized foci involving certain parts of the motor path show as a rule very marked impairment of consciousness. This fact is cited here to prove that much depends on the part involved as to the effect on consciousness.

It has been shown that Embolism—that involving only parts above the central ganglia, does not cause coma. Inasmuch as in many of these cases of Embolism a large patch of brain tissue is involved, and as further, the suddenness of the attack must be equal, whatever the

part involved, it follows that here again much must depend upon the particular structures included, for smaller infractions, if only they involve the ganglic, do bring on coma.

It can consequently be stated that whatever accessory influences there may be, there are but two important governing factors in the development of coma; the size of the hæmorrhage and the particular part of the brain implicated. They deserve a little further consideration.

As to the amount of hæmorrhage that will of itself cause coma, experiments on animals, have led to the conclusion that in the human being one and a half to two ounces are about the extent of limitation of the brain space that can be borne without interruption of psychical functions. (More can be tolerated in a diffuse effusion like a meningeal hæmorrhage than in a confined focus.) The exact amount thrown out in a case of apoplexy is rarely, if ever, known, since some of the fluid is promptly absorbed or scattered. It is not possible to more than estimate the volume of these irregular foci. So far as such rough estimation goes, it corresponds fairly to experimental results and this applies to cases in the hemispheres. When the size of an effusion is stated to be that of a hen's egg, it may be considered to equal two ounces of fluid. Hence hæmorrhage of that bulk should be and in practice is found to be on the border line. It may be expected to at least produce stupor and frequently some coma. When of greater volume, coma very generally results. In the basalgayglia, however, a much smaller amount may suffice.

The principle here is that the effusion by its volume, exerts such a general pressure on the whole cortex as to

obtund consciousness. Of the sufficiency of the factor there is no question. It may act by producing an anaemia or by more direct mechanical effect.

As to the susceptibility of different parts, injury below the oblongata (*i.e.*, in the cord) does not cause coma. The syncope of shock or even sudden death may result, but not real coma. It is uncertain whether hæmorrhage of the oblongata has much tendency to produce coma. Such cases are few and stupor is masked by respiratory and other phenomena. In the old case of Fabre some loss of consciousness attended a small hæmorrhage of the left pyramidal body. But in several other cases of small effusion in other parts of the oblongata no distinctly comatose condition has developed.

At the other brain pole—we have already seen that coma is essentially a consequence of general brain compression. In this major portion of the brain there is little difference between the various parts. Apparently the occipital lobe tolerates infringement better than the frontal and parietal lobes; but there is no decisive difference.

Regarding the cerebellum, the general finding is that uncomplicated hæmorrhage when moderate in amount does not invoke coma. But in these rather rare cases either rupture occurs or if much size is obtained, there is so much pressure on subjacent structures as to obscure the bearing of the case.

There still remains the region of the central ganglia the cerebral crura and the pons. Hæmorrhage of the caudate nucleus is prone to bring on coma. That in the lenticular nuclei and in the thalamic is somewhat less apt

to do so. When in a cerebral crus, there is commonly some coma, or at least, stupor, though these hæmorrhages are rarely voluminous. Those of the pons are most inclined to cause coma, though usually small unless they have already ruptured. A comparison of this last group of cases (involving the brain stem) brings out forcibly one fact already referred to, *viz.*, that hæmorrhages in the sensory path show but little tendency to cause coma, while those in the motor path have a marked tendency in that direction. This fact stands out quite as clearly when they are compared by volume.

Secondary factors in the causation of coma.—There are, of course, various other influences that affect this result. The person's susceptibility is one; Carbonic acid poisoning due to superficial respiration is another. But most important of these is the rapidity with which the effusion occurs. The effect on consciousness depends somewhat on the rapidity with which the compression is produced. But it is rare in clinical work to meet cases where a hæmorrhage has taken place with any such rapidity. It is observed that considerable time is taken up before the bleeding stops. We also know that in the slow, ingravescent form though a day or two elapse in the process, coma just as certainly supervenes when the volume of the focus becomes adequate.

The disappearance of coma is attributed to a re-establishment of the circulatory balance, to reduction of pressure from lessened cerebro spinal fluid, and perhaps a gradual tolerance to the focus. The shock-effect passes off and some of the fluid of the focus is absorbed.

(To be continued.)

THE RELATIONSHIP OF THE RED CELL SEDIMENTATION RATE TO VARIOUS CLINICAL CONDITIONS IN PULMONARY TUBERCULOSIS.*

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The work of Fahraeus[1] on the suspension stability of the blood in various physiological and pathological conditions first drew the attention of the clinician to the importance of the red cell sedimentation test in the diagnosis of certain diseases. The suspension stability of the blood is now known to vary within fairly wide limits, being greater in the child than in the adult, greater in women than in men, and greater in pregnant women and during the catamenia than under ordinary conditions. Fahraeus considered the test so valuable that he thought that by its means the different stages of pregnancy could be differentiated from other conditions similar to it. He attributed the phenomenon to the increase of the fibrinogen content of the blood in the various conditions giving impaired suspension stability. Amongst the pathological conditions showing increased blood sedimentation rate are malignant tumours, acute

* Read before the Indian Science Congress, January, 1929.

and chronic fevers, tabes, arteriosclerosis, syphilis, icterus, etc.

Later observers have proved that there is no definite suspension stability specific for different diseases. Artificially the speed of sedimentation can be increased by the addition of cholestrin to the blood and decreased by the addition of lecithin and there are a number of reagents that either retard or increase the speed.

In laboratory work it has also been proved that the sedimentation rate is decreased by storing the blood in sample tubes or bottles after extraction from the body so that the figures for the sedimentation reaction are unreliable unless the blood is put up in the tubes immediately after extraction. Westergren[2] does not seem to hold this view and considers that storage up to 6 hours makes no difference in the sedimentation reaction. This may be true in the climate where Westergren did his laboratory work.

Under the tropical conditions of Madras with an air temperature of 30°C to 37°C and sometimes higher, we have found that the sedimentation rate can decrease considerably by storage, instances of such decrease from 51 mm. to as low as even 1·5 mm. occurring after 6 hours' storage especially in samples where slight lysis has taken place. It is thus very essential in getting correct figures for comparative study that the blood should be put up quite fresh for the sedimentation reaction.

Again the temperature of the room in which the test is carried out is of considerable importance. As pointed out by Frimodt-Moller and Benjamin[10] the sedimentation rate in the tropics is higher than in temperate climates. It is thus necessary in recording the results to note the temperature of the air during the experiment.

The importance of having a standard of calibre and height of column of blood has been sufficiently stressed by Frimodt-Moller and Benjamin. By putting up the same blood in tubes of the same bore but in different heights of the column of citrated blood, it has been found by us that the sedimentation rate expressed in percentages decreased with the height of the column. It is therefore quite incorrect to express the rates in percentages. Thus in table I blood 1 gives 45% sedimentation in 1 hour in the 200 mm. column, but 54% in the 100 mm. column and 62% in the 50 mm. column. The average of the figures for 12 cases gives 35% sedimentation for the first hour in the 200 mm. column, 45% in the 100 mm. column and 52% in the 50 mm. column. It is thus evident that the rate of sedimentation expressed in percentages cannot be considered accurate unless the same height of column and bore of tube are used. It might be possible however to express the sedimentation by a mathematical formula applicable to different heights of column and bores of sedimentation tube. The results of different workers are therefore not comparable unless a number of factors are identical, *viz.*, (1) the temperature of the air, (2) the time elapsed between the extraction and the experiment, (3) the calibre of the sedimentation tube, (4) the height of the column of blood ; not to mention other conditions like pregnancy and menstruation in females, the percentage of the citrate and the quantity used for the prevention of clotting, the time of the day the blood is extracted and so on.

TABLE I.

Showing rates of sedimentation in one hour for different heights of blood column in 12 cases, tuberculous and non-tuberculous—Bore of tube 2·5 mm.

Clinicial condition.	200 mm. column.	100 mm. column.	50 mm. column.
Bronchitis	90 mm.	54 mm.	31 mm.
Bronchitis	50 mm.	34 mm.	22 mm.
P. T. III	102 mm.	60 mm.	35 mm.
Bronchitis	30 mm.	23 mm.	15 mm.
Bronchitis	38 mm.	28 mm.	18 mm.
P. T. III	129 mm.	73 mm.	38 mm.
P. T. III	82 mm.	50 mm.	31 mm.
P. T. III	88 mm.	58 mm.	30 mm.
Bronchitis	16 mm.	15 mm.	13 mm.
Bronchitis	104 mm.	60 mm.	32 mm.
Bronchitis	22 mm.	18 mm.	14 mm.
P. T. III	105 mm.	62 mm.	35 mm.
Average	35%	45%	52%

In our work, on account of the laboratory being far away from the hospital, a period of 1 to 2 hours usually intervened between the extraction of the blood and the putting up for sedimentation and the atmospheric temparature was, as said above, 30° C to 37° C or higher.

The application of the red cell sedimentation test to tuberculous conditions originated with Westergren[2] who carried on a systematic research with the test in pulmonary tuberculosis. He found a great increase of velocity

of sedimentation in the disease, especially in cases with marked activity and tissue destruction. Westergren considered the test of very great value in prognosis. It is however very important as has been shown by Dreyfus, Hecht and others that in adjudicating the value of the test concomitant conditions like syphilis, cachexia, etc., should be taken into due consideration.

In the early stages of pulmonary tuberculosis, it has been found by various observers like Heaf,[3] Popper,[4] Freund and Henschke[5] and others that the sedimentation speed is slightly increased and that when cavitation and cirrhotic processes have set in, the sedimentation rate is markedly increased.

Grafe[6] considered that the test was very helpful in differentiating active from latent tuberculosis, and that injections of small doses of tuberculin caused an increaced sedimentation rate in latent cases, but not in healthy people. Injection of tuberculin also causes a further increase in the sedimentation rate in advanced cases.

Levinson,[7] by an analysis of over 400 cases at Chicago has shown that the rate of sedimentation increases with the advance of the disease in the lungs and that intercurrent diseases like Influenza, Purpura, etc., further increase the sedimentation rate. He considers that the test is a gauge of cell destruction in advancing lung disease.

Cummins and Acland[8] from an analysis of 200 cases conclude that the test is an additional method of measuring the constitutional balance of a case before, during and after treatment, standing on a line with the temparature, weight and general clinical progress.

Luzzatto Fegiz[9] whilst confirming the same view lays particular stress on the marked decrease in the sedimentation rate in artificial pneumothorax which denotes the effect on the fibrinogen content of the blood by a quick stoppage of the disorganizing process in the lungs.

Frimodt-Moller and Benjamin[10] working at the Arogyavaram Sanatorium in South India amongst the patients in the Sanatorium, with controls from amongst normal subjects and non-tuberculous individuals have shown that the sedimentation rates in the tropical conditions of Southern India, are higher than those of Europe in tuberculous as well as normal and non-tuberculous conditions. The same observation had been made experimentally by Westergren by incubating the tubes at higher temperatures.

Stubbe has shown by experiments that the rate of sedimentation in a thermostat at 38° C is about double that at 18° C. Collapse therapy and Sanocrysin treatment, he observed, had a decided tendency to lessen the rate especially in cases that benefited.

Frimodt-Moller and Benjamin consider that the test is valuable and delicate, though not specific and that it indicates the amount of tissue destruction and the fibrinogen content of the blood plasma. They conclude that the test has no specific value and cannot be used for distinguishing active from inactive cases except when considering its results along with other clinical signs and symptoms; that its special value lies in determining the prognosis especially in prognosticating brewing exacerbations or complications as in judging the results and prospects of artificial pneumothorax.

The work of Moral,[11] Salmon,[12] Weichsel,[13] Wachter,[14] Petre,[15] Bochalli,[16] Schmidt,[17] and others

goes to prove in general that whilst a high sedimentation rate indicates a bad prognosis and a low rate the reverse, exceptions to the rule are to be found in a certain proportion of cases; and that whilst there is no definite sedimentation rate for any particular disease or for different stages of pulmonary tuberculosis, a systematic record of the rate time after time in the progress of cases is of considerable value in determining the prognosis.

Though the amount of fibrinogen elements in the blood are now recognized to be the main factor concerned in the sedimentation reaction the possibility of the size and constituents of the red cell itself influencing the sedimentation cannot be overlooked, as has been considered by several workers.

Though the principle of the test as adopted by different persons, has been the same, the technique has, differed with different workers to a greater or less extent. Thus Fahraues used tubes of comparatively large calibre, Westergren used tubes about 30 cm. long and 2·5 mm. diameter, the blood column being 200 mm. in height. Frimodt-Moller and Benjamin used 1 c. c. pipettes graduated into 100 parts and read off the results in percentages.

Our technique has been the same as Westergren's. The ratio of 3·8 % citrate of soda to the patients' blood has been 1 to 4. The samples of blood obtained from the patients in hospital were transferred to the laboratory in specimen tubes and put up from 1 to 2 hours after extraction. The results were read after 1, 2 and 24 hours and the figures representing sedimentation at the end of 1 hour or half the sedimentation at the end of 2 hours whichever was greater was noted down as the sedimentation rate. We have already shown why the results

obtained by our method cannot be strictly compared with those obtained by Frimodt-Moller and others. They closely follow Westergren's results except that we worked in a much hotter climate.

The sedimentation rate in the various types and in the different conditions of pulmonary tuberculosis are set out in the subjoined tables. Table II gives the sedimentation rate in normal subjects, including patients suffering from bronchitis and other mild non-tuberculous conditions who attended the out-patients' department of the Tuberculosis Institute. It will be seen that the figures are very much higher than those of the temperate climate of Europe and those obtained in the hill climate of Arogyavaram. The normal figures for men as well as for women were 20 and 50 as compared with 17 to 25 for men and 25 to 34 for women at Arogyavaram and 3 to 5 for men and 5 to 10 for women obtained by Westergren, Katz, [18] and others in Europe respectively. Ten out of 46 men and 9 out of 36 women gave higher figures than 50 in our series.

TABLE II.

S. R. in normal and non-tuberculous subjects.

82 cases.

——	Men.	Women.
0— 5	3	0
6— 10	6	4
11— 15	4	2
16— 20	4	5
21— 30	8	7
31— 50	11	9
51—100	10	9

Table III gives figures for the three stages of pulmonary tuberculosis in 222 cases of men and women who were treated in the Madras Tuberculosis Hospital. It will be seen that whilst 3 out of 14 men and 9 out of 38 women give higher sedimentation than 50 in stage I, 11 out of 20 men and 8 out of 14 women gave the same rate in stage II and 75 out of 95 men and 35 out of 41 women in stage III. It is evident that the sedimentation rate has varied directly with the stage of the pulmonary implication.

TABLE III.

S. R. in Tuberculous patients according to stage of disease.

222 cases.

S. R.	Stage I.		Stage II.		Stage III.	
	Men	Women.	Men	Women.	Men	Women.
0— 5	0	0	0	0	0	0
6— 10	3	4	0	1	0	0
11— 15	2	4	1	0	0	0
16— 20	1	6	2	1	1	1
21— 30	2	6	2	1	4	1
31— 50	3	9	4	3	15	4
51—100	3	8	8	5	60	28
101—150	0	1	3	3	15	7
Total.	14	38	20	14	95	41

Similar figures classified according to the temperature as an index of constitutional disturbance and toxic absorption showed that 51 out of 126 men and 41 out of

90 women gave a sedimentation rate of over 50 when the temperature did not exceed 99°F. at any time of the day. When the temparature reached the maximum of between 99° and 100·5° F. daily, 73 out of 105 men and 51 out of 105 women gave a rate of over 50 mm. and when the temperature ranged above 100·5°F. 38 out of 40 men and 23 out of 27 women gave similarly high figures (*vide* table IV).

TABLE IV.

S. R. in Tuberculous men and women according to temperature.

493 Tests.

S. R.	Temp. Nor. to 99° F.		Max. 99 to 100·5° F.		Max. over 100·5° F.	
	Men	Women.	Men.	Women	Men	Women.
0— 5	2	0	0	1	0	0
6— 10	6	4	1	6	1	0
11— 15	7	4	0	5	0	0
16— 20	7	4	2	8	0	0
21— 30	16	9	7	18	0	1
31— 50	37	28	20	16	1	3
51—100	50	73	56	45	28	17
101—150	1	4	17	6	10	6

To find out whether the presence or absence of tubercle bacilli in the sputum made any difference in the sedimentation rate of the blood, figures from 490 experiments were classified with the result that 125 out of 165 men and 86 out of 116 women with positive sputa gave higher figures than 50 mm. whilst in the bacillus negative cases the figures were 36 out of 103 men and 29 out of

106 women. It is apparent that the bacillus positive cases with greater disorganization of lung tissue give higher figures than the earlier bacillus negative cases (*vide* table V).

TABLE V.

S. R. in cases of Tuberculosis according to whether T. B. S. were present or not in the sputum.

490 Tests.

S. R.	T. B. Positive.		T. B. Negative.	
	Men.	Women.	Men.	Women.
0— 5	0	0	2	1
6— 10	0	1	8	9
11— 15	2	1	5	8
16— 20	0	0	9	12
21— 30	4	6	19	22
31— 50	34	22	24	25
51—100	102	74	31	25
101—150	23	12	5	4
Total.	165	116	103	106

The next set of figures were obtained from 392 experiments with the idea of ascertaining whether there was any relationship between the tubercle complement fixation reaction and the sedimentation reaction in cases clinically tuberculous. The cases were divided into two sets those with a negative or weak complement fixation reaction and those with a moderate or strong reaction. It was found that 55 out of 108 cases of the former class (51%) and 168 out of 284 (59%) of the latter class gave higher figures than 50. The difference between the two

series was therefore not very well marked. This agrees with the observation of Katz and Kempner who consider there is no constant relationship between the two reaction (*vide* table VI).

TABLE VI.

S. R. in Pulmonary Tuberculosis according to the degree of complement fixation reaction.

392 Tests.

S. R.	T. C. F. R. negative or weak.		T.C.F.R. moderate or strong.	
	Men.	Women.	Men.	Women.
0— 5	1	0	1	0
6— 10	2	5	6	2
11— 15	3	5	2	2
16— 20	1	5	7	2
21— 30	6	8	15	16
31— 50	10	7	39	24
51—100	33	13	87	58
101—150	6	3	17	6
Total	62	46	174	110

The results laid out in table VII are interesting. In it, it is seen that in 75% of the cases general improvement is associated with decrease of the red-cell sedimentation rate and that in 25% cases only does an increase of the rate coincide with improvement in the patient's local and general condition. Again of cases that became worse 73% gave an increased sedimentation rate and 27% only a decrease of the rate.

77% of the cases that put on body weight gave a retardation of the sedimentation rate and 61% of patients who lost body weight gave increased sedimentation. This is as one would expect, considering that increase of body weight is as a rule a sign of general improvement (*vide* table VII.)

TABLE VII.

The Clinical Significance of the S. R. in Pulmonary Tuberculosis.

1.	Cases in which general improvement was associated with decrease of the sedimentation rate.	69 (75%)
2.	Cases in which there was an increase of the S. R. along with improvement in clinical condition.	23 (25%)
3.	Cases in which there was an increase of the S. R. with deterioration of clinical condition.	16 (73%)
4.	Cases in which decrease of the S.R. resulted, with progression of the disease.	6 (27%)
5.	Cases in which there was increase of body weight along with decrease of S. R.	61 (77%)
6.	Cases in which there was increase of body weight with increase of S. R.	18 (23%)
7.	Cases in which there was decrease of body weight with increase of S. R.	22 (61%)
8.	Cases in which there was decrease of body weight with increase of S. R.	14 (39%)
9.	Cases that improved under Artificial Pneumothorax treatment with decrease of S.R.	26 (72%)

10. Cases that improved under Artificial Pneumothorax treatment with increase of S.R. — 10 (28%)
11. Cases that improved under Sanocrysin treatment with decrease of S. R. .... — 21 (75%)
12. Cases that improved under Sanocrysin treatment with increase of S. R. .... — 7 (25%)

Of the cases that improved under artificial pneumothorax treatment 72% showed a retarded sedimentation and of those that benefited by Sanocrysin Treatment 75% showed a similar decrease in the rate (*vide* table VII.)

It is apparent from the above results that general improvement whether due to ordinary treatment, Sanocrysin therapy or collapse therapy is associated with greater red-cell stability to a more or less equal degree Whilst artificial pneumothorax brings on a more rapid decrease in the sedimentation rate due to quicker stoppage of tissue destruction, any other treatment that effects improvement in the patient's condition is apt to result in a similar inhibition of red-cell sedimentation though more slowly.

The most interesting of all the results are given in table VIII. 32% of the cases with a good prognosis, 82% of cases with a doubtful prognosis and 92% of those with a bad prognosis gave sedimentation figures of over 50. These figures have been obtained from an analysis of 229 cases. With the margin at 30, it is seen that 62%, 90% and 100% respectively represent the figures for the three classes of cases. It is thus evident that the sedimentation rate can be generally taken as a fair measure of the clinical condition and the prognosis of a case, though no definite conclusions can be drawn in individual cases.

The value of the test stands almost on the same level, as that of the pulse and the temperature in tuberculous conditions, and a graphic representation of the red blood cell sedimentation rate can be considered to be a very accurate index of the progress and the prognosis of a case of pulmonary tuberculosis.

TABLE VIII.

The S. R. in Relation to Clinical Prognois.

229 cases.

S. R.	Good Prognosis.	Doubtful Prognosis.	Bad Prognosis.
1— 5	1	0	0
6— 10	8	3	0
11— 15	7	0	0
16— 20	6	2	0
21— 30	26	2	0
31— 50	38	5	3
51— 100	37	41	22
101— 150	4	13	11
Total ...	127	66	36

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DETERIORATION OF THE DIGITALIS GLUCOSIDES IN THE TROPICS

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AND

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A few months ago, a sample of tincture of strophanthus was sent to us for biological assay by the second Physician of the Madras General Hospital. This sample was found, on examination, to have completely lost its potency and exhibited no physiological effects when administered intravenously to cats in proper dilution. We tried to get another sample of tincture strophanthus from the General Hospital, but were told that this preparation was not usually stocked but was bought in retail as required from local chemists. So we collected a few samples of both Tr. Strophanthus and Tr. Digitalis from the dispensing counter of various local Pharmacists and tested their potency biologically. The work on Tr. Digitalis was started earlier, as the Surgeon General as well as the Superintendent of the General Hospital were sending us samples for standardisation. The results of the biological assay of these preparations are given below in tabular form.

A short description of the method employed in biological standardisation of these preparations may be given here. The experiments are done on animals; either frogs or cats or both may be used. We used Rowntree and Macht's modification of Hatcher and Brody's cat method in our assays. The animals selected were healthy cats weighing between 1600 and 2500 grammes. The anæsthetic employed was 0·18 gramme of Chloretone per Kilogram weight of body, dissolved in a few cc of alcohol and injected intraperitoneally. In about 15 to 25 minutes the animals go under deep anæsthesia. Then they are placed on the operation table and a cannula introduced into the femoral vein. The tinctures are usually diluted with 9 parts of physiological saline and placed in a burette connected to the venous cannula. The physical character of the solution are noted and the heart rate counted with the aid of a stethoscope. The solution is then allowed to flow into the vein slowly and at a constant rate, say 1 or 2 cc every 5 minutes. The heart is auscultated at regular intervals, the beats counted and any change in the character of the sounds noted. The preparation is run in until the heart stops. This gives the minimum lethal dose of the diluted Tr. for the animal. One-tenth of this is the M.L.D. of the undiluted preparation. The number of cc of this equalling 1 cc of standard tincture is then calculated. The standard is known as the cat unit. In a preparation of standard strength, 1 cc. is the M.L.D. for 1 Kilo weight of cat and is the cat unit. Therefore from the M.L.D. of the given tincture the percentage strength of the preparation can be easily calculated.

An example of this kind of assay is given below :—

Date of assay 29-11-1927.

Weight of cat 1950 grammes

Chloretone 0·18 grammes per kilo.

Dilution of Tr. 1 in 10.

Total solution required for the assay :—17 cc.

Time required to complete assay :—67 minutes.

Physical Character of dilute Tr :—Light greenish Opalescent.

Time.	Quantity of dilute Tr.	Ht. rate.	Remarks.
12— 35	0 cc.	155	clear, regular
12— 40	1 ,,	140	,,
12— 45	2 ,,	136	,,
12— 50	3 ,,	128	,,
12— 55	4 ,,	130	,,
1— 0	5 ,,	124	,,
1— 5	6 ,,	136	,,
1— 10	7 ,,	117	,,
1— 15	8 ,,	126	,,
1— 20	9 ,,	114	,,
1— 25	10 ,,	142	,,
1— 30	12 ,,	142	,,
1— 35	14 ,,	150	,,
1— 40	16 ,,	muffled	
1— 42	17 ,,	dead	

M.L.D.—1·7 cc. of undiluted Tr.

Cat Unit—0·88 cc.; % strength—114.

TABLE I.

(Tincture Digitalis.)

No.	Physical Characteristics.	Date of Manufacture.	Date of Assay.	Reduction in Ht. rate.	No. of cc. equal to 1cc. of standard Tr.	Comparative Potency with standard Tr. %
Good.—						
1 Digifortis P. D. & Co.	Opalescent greenish yellow.	7— 7—27	20— 2—28	20	0 61	164
2. Digifortis P. D. & Co.	do. ...	19— 7—27	17—12—28	28	0 72	138
3	Opalescent greenish	not known	9—11—28	16	0 80	125
4	do. ...	do.	27—11—27	41	0·87	114
5	do. ...	26— 6—28	21— 8—28	20	0·88	113
6	do. ...	not known	18—12—28	22	0·93	107
7	do. ...	1924	20— 2—28	40	1·03	98
Weak.—						
8 same as No. 1 but opened on 20—2—28.	Opalescent greenish yellow.	7-- 7—27	3—11—28	nil	1·03	98
9	Opalescent greenish.	12— 4—28	10—12—28	nil	1·14	87
10	Dark opalescent greenish	11— 4—27	17— 1—28	38	1·15	85
11	Light green.	not known	2—11—28	20	1·25	80
Toxic.—						
12	Cloudy greenish yellow.	do.	22—10—28	8	0·58	171
13	Dirty green cloudy.	do.	5—11—28	nil	0·88	113

TABLE II.

(Tincture Strophanthus.)

No.	Date of Manufacture.	Date of Assay.	Reduction in Ht. rate.	No. of cc. equal to 1cc. of standard Tr.	Comparative Potency with standard Tincture.
Fair.—					
1	not known	1— 8 – 28	30	1·33	75
Weak.—					
2	not known	3— 8—28	16	1·66	60
3	12—4—28	5—12—28	12	1·79	56
4	not known	20—12—28	nil	1·77	57
No Physical Result.					
5	not known	6— 8—28	no effect even with large doses		
6	do.	6— 8—28	do.		
7	do.	8— 8—28	do.		
8	do.	8— 8—28	do.		

Table I shows the results of assay of 13 samples of tincture of digitalis. Of these, 7 have been classified as good. The maximum slowing of the pulse was 41, obtained with a tincture prepared in Calcutta by an Indian firm (Union Drug Co.) The percentage strength of this was 114. The date of preparation is not known but was possibly a month or two previous to the assay. A sample of Tr. Digitalis prepared by the Madras Medical Stores Depot during 1924 but kept unopened in the Laboratory was assayed in February 1928 and found to be quite potent; the heart rate slowed by 40 beats and the percentage strength was 98. Nos. 1 & 2 are two samples of Digifortis, an overstrength tincture prepared by Parke Davis & Co. in July 1927. Both bottles were opened on the date of assay, *i.e.*, 7 months and 17 months after date of manufacture and found to be up to the potency claimed for them, *i.e.*, 150%. The first sample of digifortis which was opened in the laboratory on 20-2-28 was again assayed on 3-11-28 and found to have deteriorated. Although the strength had only deteriorated 33%, yet there was no slowing of the pulse. This means that no therapeutic effect would be noticed in patients. Tinctures, No. 9 & 10 which were certified fully potent at the time of issue had lost 15% potency in about 8 months. Tinctures Nos. 12 and 13 are toxic samples. These tinctures, when diluted for assay purposes, did not produce a uniform opalescent solution but showed cloudiness, and a blackish precipitate was visible. In these cases some changes had taken place in the active glucosides so that no therapeutic effect would be obtained, but at the same time the minimum lethal dose for animals was greatly lessened. To avoid confusion, it may be pointed out here (Chopra and De 1926) that tinctures that deteriorate and become toxic intravenously,

do not produce any poisonous effect when administered by the mouth, though of course they produce little therapeutic action.

A large number of preparations of digitalis are on the market, purporting to possess all the advantages of digitalis without its drawbacks. Thus it is claimed for some of them, that, being pure principles, the dose is uniform and the treatment is more 'scientific' than when cruder preparations are employed. "But" says Cushny "The active principles of digitalis are so little known that no two chemists working on them are agreed as to how many are present in the plant, and it may be questioned whether any one of them has been isolated. Much less are the preparations available for therapeutic purposes to be regarded as pure substances or as incorporating the whole virtues of digitalis. This would be of less moment if these so called digitalines, digitoxins, gitalins and digalens were reliable in strength; unfortunately it has not yet been proved possible even for the same manufacturer working by the same method to put a uniform product on the market. And the more elaborate the method, the more do different samples of the final product seem to vary in strength." We made an attempt to assay the potency of a sample of Digalen, put on the market by Hoffmann-La-Roche & Co. 15 minims of this preparation is said to have a potency equal to 2 grs. or 0·13 gm. of the dried leaf. As 0·1 gm of the well dried leaf corresponds to a cat unit, the potency of Digalen must be the same as the ordinary 10% tincture of the Pharmacopœia. But assayed in the usual way, Digalen appears to be very weak as compared with the standard B.P. tincture; and the dose required to produce digitalisation or a good therapeutic slowing of the heart is about double or even more, of that required by an ordinary

good tincture. We must therefore agree with Cushny that "'The pure principles' of digitalis on the market are to be regarded with distrust and to be prescribed with care."

Preparations of Strophanthus detiorate even more rapidly than those of digitalis. Of 8 tinctures assayed, (Table II) 4 showed no physiological action and only one could be passed as fair. This latter was prepared by a local firm of chemists, who did not have it biologically standardised. When this firm was asked why they were putting on the market preparations without having them properly assayed, the plea was that if they resorted to such standardisation, the price would have to be increased and owing to competition, they could not afford to do so. Three samples were weak and the cardiac slowing was not well marked.

It is clear from the above data that preparations of digitalis and strophanthus usually sold in the Indian market are unsatisfactory. This may be due to deterioration caused by the action of tropical heat and moisture on the active glucosides in the preparations. This reason holds good for such tinctures as were found to be fully potent on issue, but deteriorated later. The unsatisfactory condition may also be due to an inferior quality of leaf having been used in the preparation of the tinctures. It has been shown by Chopra and De (1926) that digitalis leaf grown in the highly humid climate obtaining on the Nilgiris and near Darjeeling often produce weak or toxic tincture. Even when the leaf is above fault the method of drying and storing may be at fault. It is therefore unwise on the part of manufacturers in India to hold large stocks of these preparations that undergo changes in a few months. It is, we

think, even more unwise for hospitals to indent for more than a six months, supply at a time. It has been pointed out by Chopra and De (1926) that preparations of tincture of digitalis lose about 20 % potency soon after import into India. There should be no difficulty in obtaining preparations certified as potent by biological assay in smaller quantities without delay, as more than one firm of chemists in India prepare these tinctures and have proper arrangements made for their standardisation by a competent pharmacologist. Mention may be here made of Messrs. Smith Stanistreet & Co. and the Bengal Chemical and Pharmaceutical Works, both of Calcutta.

Hospitals and dispensaries including dispensing chemists must send samples of their tinctures for biological assay every six months and must reject such preparations as are condemned by the Pharmacologist. This certainly entails more expense, but it *must* be done in the interest of the vast multitude of patients who have such implicit faith 'in the pills and potions' of the pharmacist.

A word may be said here of the preparation called *Digifortis*, manufactured by Parke Davis & Co., which is said to be 50 % above standard strength. This preparation is free from fat, is sold in convenient one ounce bottles and is further protected from oxidation by the use of carbon di oxide. The date of manufacture is put on each bottle and the manufacturers advise that it should not be used after 12 months from the date of manufacture. The dose advised is 8 minims. Chopra and De (1926) have given as much as 30 minims, diluted with a little water, on an empty stomach without any discomfort to the patient or untoward effects. It would appear from both experimental and clinical data that are

available that, making allowance to the effects of climate, etc., there still remains adequate potency in this preparation. The importance of using only samples that are within the stated period of 12 months is obvious from the assay of tincture No. 8 (Table I.)

Administration of Digitalis and Strophanthus.

When administering digitalis, one must always keep in mind the dangers of cumulative action and look out for danger signals in the heart. But the tendency to err too much on the side of caution should also be combated. Poisoning with digitalis is very rare. Nausea and vomiting supervene quickly, even before toxic dosage is reached. As much as 1 minim of the ordinary tincture per pound weight, *i.e.*, 2 drachms for a man weighing 120 lbs. can be given in a single does if therapeutic effects are desired without producing untoward symptoms. It has been calculated that an average healthy adult destroys about 22 minims of the tincture in 24 hours, and this quantity passes out of the body without producing therapeutic effect. The balance accumulates in the heart muscle till sufficient concentration is reached to produce its effect. After the therapeutic effect is produced, the dose can be reduced to 10 minims *t. i. d.* to keep the heart under digitalis effect. This dosage is particularly important as it has been shown that digitalis loses its potency and deteriorates quickly in the tropics. A series of tinctures was assayed biologically aud tested clinically by Chopra, Bose and De (1925) and with doses of 20 to 30 minims thrice a day, patients could not be got under digitalis effect inside of 12 days. What will then be the effect of such cautious doses as 5 to 10 minims that are given by practitioners in India? Eggleston (1915) has calculated that the average amount inducing full therapeutic effects

within 36 to 48 hours in an adult man, who has not received digitalis previously, is about 1·5 gms. of the leaf or 15 cc of the tincture 4¼ drs.) per 100 lb body weight, the tincture being given in divided doses at intervals of 4 to 6 hours. Absorption of each dose is practically complete after some 6 hours, and any effect will be evident about this time ; if none appears, a smaller amount may be given, and again after each 6 hours this may be repeated if necessary. Theoretically, the daily dose is exactly equal to the amount disposed off by the body daily. The method is certainly worthy of trial, especially in urgent cases.

For such intensive treatment, the tincture must be reliable and uniform in strength and it is necessary to have it assayed by animal experiment. The dose must be accurately measured. There is some danger that dilution with water may lead to decomposition of the glucosides and it is preferable to have it dispensed alone and diluted immediately before being swallowed. This is particularly preferable in the case of tincture of surophanthus which seems to be very sensitive to dilution and especially to the addition of alkalies and is best dispensed alone, to be taken diluted with water.

Strophanthin has been used either as amorphous *Kombe Strophanthin* or the crystalline g-*Strophanthine* or *ouabain* indiscriminately. Given by the mouth it is unreliable and when injected hypodermically it causes pain and irritation and does not generally prove efficient by this method. But the chief use of *Strophanthin* is to obtain rapid effects in emergency by intravenous injection of 1/250 to 1/150 grs. Effects are obtained within half an hour and the full action in 4 to 6 hours. The dose must not be repeated within 24 hours. Later it may be supplemented by digitalis preparations by mouth.

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3. *Chopra and De*—1926. Biological assay of digitalis preparations in the Tropics. Indian Journal of Medical Research, Vol. XIII No. I, April.

4. *Cushny, A. R.*—1925. Action and uses of digitalis and its allies.

A CASE OF ANEURYSM.

By Dr. K. N. PISHARATY, M.B, B.S.,

Lecturer in Medicine, Medical School, Tanjore.

Ponnusamy, Christian male aged 30 years, ryot by occupation, was admitted into the Headquarter Hospital, Tanjore, on 14-5-1925 for pain in the chest and back of seven months' duration. The pain was constant. He had slight cough with scanty expectoration. The patient gave a history of mild fever coming on at times.

Previous history of gonorrhœa two years back. Scars present in the groins, said to be due to operation for bubo. No history of syphilis. The patient was addicted to alcohol.

Condition on admission :—Patient was a faily well built and well nourished individual. No anæmia nor enlargement of glands. Lungs: Slight diminution of movement over the left apex, vocal fremitus increased; diminished resonance on percussion in the region; breathsounds bronchial in character; vocal resonance increased; no adventitious sounds. Heart normal. Other systems normal. Sputum scanty, did not contain tubercle bacilli. The patient was observedto be having a slight rise of temperature in the evenings.

From the signs of consolidation over the apex of one lung the case was thought to be one of tuberculosis of the lung, where the symptoms were few, but the signs marked. The patient was discharged 'Relieved' on 29-5-1925.

On 7-10-1925 the patient sought readmission into the hospital for pain in the chest, and dyspnœa. He said that since leaving the hospital his condition had been gradually getting worse. The pain had increased. It was now constant, and felt in the chest, back and upper limbs, particularly on the left side. He had irritant cough, with scanty expectoration and continuous dyspnœa increased on exertion with pain while swallowing. His voice was hoarse. He had sleeplessness on account of the pain.

Physical examination :—General health good. The left eyes looked smaller due to drooping of the upper eyelid. The pupil of the left eye is contracted. Circulatory system: Slight localised bulging extending from just below the inner end of the left clavicle to the lower part of the left third intercostal space, and transversely from the midsternal line to the left parasternal line. Visible pulsation over the swelling, but the pulsation is not expansible. The apex beat is seen in the sixth left intercostal space half an inch external to the nipple line. The superficial veins over the upper part of the left side of chest, the leftside of neck and the left arm are distended. Tenderness is present over the swelling and all over the præcordial region. No thrills. The percussion note over the swelling is dull, and it is continuous with that of the heart. Dulness is also present over the manubrium stern and throughout the first and second left intercostal spaces. The left border of the heart is just internal to the left nipple line, the right border over the right lateral sternal line. Heart sounds normal in all the four areas. No murmur is heard over the selling; but a soft systolic murmur is heard in the interscapular region on the left side up to the level of the lower angle

of the scapula. An almost continuous humming bruit is heard in the left axilla.

The radial pulse on the left side is not felt. On the right side it is regular, of fair volume and low tension. Pulsation is felt over the left Carotid. There is absence of pulsation in the abdominal aorta and the femoral arteries. Tracheal tugging in present.

Respiratory system :—Diminished movement in the left supra and infraclavicular fossal ; vocal fremitus same on either side ; dullness in the infraclavicular region and the suprascapular and interscapular regions on the left side ; no adventitious sounds. Sputum blood stained, negative for tubercle bacilli.

The patient is not having fever. The pain and cough are gradually getting worse. A few moist sounds are now heard over the base of the left lung.

The case was diagnosed as one of aneurysm of the arch of the aorta (the transverse and the descending portion). The interest of the case has lies in the fact that it was thought to be a case of tuberculosis of the lung (left apex) when the patient was first admitted into the hospital four months previously. The signs of consolidation then observed must have been due to the aneurysm causing pressure on the bronchus and the lung. There was then no visible swelling or pulsation, no displacement of the apex beat, bruit, or any other signs to suspect aneurysm.

EDITORIAL NOTES.

Magazine :—

Our Magazine has started on another year of its life but it is regretted it does not receive the support it should from the members of service. We are experiencing considerable difficulty in getting articles for publication. There should be no such difficulty with the membership we have. We earnestly request the members of the Madras Provincial Service to co-operate with us to run the Magazine successfully. Articles sent by members need not necessarily be original, but notes on interesting cases, book reviews, abstracts, etc., are all welcome. We expect every member to do his best to make the Magazine worthy of the service it belongs to.

All communications in this connection may please be sent to The Editor, Dr. P. V. Cherian, Egmore, Madras.

Subscriptions:—

Members are requested to send their annual subscription to the Honorary Treasurer, Dr. P. V. Cherian, Egmore, Madras, at a very early date. It has been the custom to send the Magazine by V. P. Post and collect the subscriptions. This involves a large amount of clerical work. The present issue of the Magazine is sent to every member of the Madras Medical Service and we request every one to kindly accept the Magazine and send the annual subscription to the Treasurer. We sincerely trust there will not be any need to send Magazines this year by V. P. Post.

Change of Address:—

Members are kindly requested to intimate to the Editor any change of address.

'*Madras General Hospital*':—

We commend to our readers the book on The Madras General Hospital published as a memento of the laying of the foundation stone of the new Out Patient Department—the first part of the remodeling scheme—by His Excellency the Right Honourable Viscount Goschen of Hawkhurst, G.C.I.E., C.B.E., V.D., Governor of Madras. This is a beautifully got up book with thirty illustrations twenty-eight of which are reprints from photographs. It also contains short notes on the Government General Hospital, the Madras Medical College, and also notes by the Consulting Architect. It is highly desirable that every one of us who have passed through the Madras General Hospital should possess a copy of this book as we are certain that some of the most happy years of our life are associated with the General Hospital and the Medical College. The book is priced at the very moderate figure of Rs. 2-0-0, and any profit accruing from the sale of the books will be devoted to the emergency fund of the General Hospital. Books can be had on application to the Superintendent, General Hospital, Madras.

ABSTRACTS FROM JOURNALS.

A Clinical Study of Human and Bovine Types of Tuberculosis in Man

BY J. T. MORRISON, M.D., F.R.C.S.

The Lancet, August 18, 1928, p. 319.

This paper gives a synopsis of the bacteriological investigation of 100 cases of non-pulmonary tuberculosis from different institutions in and about Liverpool. In 69 of the 100 cases inoculation experiments gave a negative result in the animal though most of them were clinically undoubted cases of tuberculosis. The material was obtained from glands, bones, peritoneum, etc., Laboratory investigation showed that of 56 gland cases, 25 were bovine, 11 human and 20 gave no result. Of 25 bone and joint cases, 5 were bovine, 13 human and 7 gave no result. Both the Abdominal cases investigated were bovine, and of the two genito urinary cases one was bovine and the other human. Of the lymphatic glands infections of children 16 % were human, whilst amongst adults 62 % were human. Of all surgical cases in adults 72 % were human. Family history was not constantly present in these cases and it was not always possible to determine the portal of entry of B. tuberculosis. The bovine infections were more amenable to treatment than the human and there was a much larger percentage of recoveries amongst the bovine class of infections.

M. K. P.

Sources of Infection in Non-Pulmonary Tuberculosis

By C. O. STALLYBRASS, M.D., D.P.H.,

The Lancet 18th August 1928, page 320.

The paper gives an analysis of 4,469 cases. It was found that tuberculosis of bones and joints is more frequent when there is no family history, but tuberculosis of abdomen and of the meninges is more frequent in people giving a family history or history of exposure. Tuberculosis of the peripheral lymphatic glands is in excess in persons giving a family history. 46 % of the cervical glands, 18 % of bone and joint diseases, 50 % of the Lupus cases and 16 % of the meningeal cases were bovine. Arranged according to the age period 30 % between the age 0 to 10, 11 % between 10 to 15, and 20 % between 15 and 25 were bovine in origin, whilst over 25 years no case of bovine infection could be made out.

M. K. P.

Artificial Pneumothroax Treatment in India

By C. FRIMODT-MOLLER.

Indian Medical Gazette No. 5, May 1928.

Dr. Frimodt Moller gives in this article an analysis of 306 cases of pulmonary tuberculosis to whom artificial pnemothorax was applied at the Arogyvaram Sanatorium during the years 1921-1928. Of these 90 cases were failures due to total adhesions. These 90 cases were treated by the ordinary Sanatorium methods and of these 10% became clinically well.

Of the 216 cases in whom there was available pleural space 124 had less than 6 months of inflations and of these only 7% became well. Of the remaining 92 that had more than 6 months of pneumothorax treatment 44·5% became clinically well. Of these 92, 57 had a good pneumothorax and 35 a partial pnuemothorax. 58% of the good pneumothorax cases and 23% of the partial pnuemothorax cases became arrested. 6 of the total number had 2 to 3 years of pneumothorax treatment and the rest less than 2 years and many or these are on full work now, 6 years after treatment was began. Of the 4,000 inflations during 7 years there were no fatal accidents and only two patients showed temporary alarming symptoms. Effusion which occurred in 43·5% cases did not unfavourably influence the progress of the cases. It is difficult in a country like India to follow up the treatment for a long time; yet even with the shorter courses of pneumothorax treatment there was remarkable benefit. 44·5 became clinically well, as contrasted with 7 to 10% of similar advanced cases treated by ordinary methods.

M. K. P.

The Diagnosis of Tuberculosis in General Practice

By Dr. LAWRASON BROWN.

Journal of the American Medical Association, 1928, X-C.-1032.

Dr. Lawrason Brown of Saranac Lake, the great authority on Tuberculosis, has in this article tried to put down certain standards for the diagnosis of tuberculosis. He considers that hæmoptysis to be called so,

should be at least a drachm in amount. Streaks of blood or rusty sputm are more often due to other lung conditions. Pleurisy with effusion is, as a rule, tuberculous in origin, whilst dry pleurisy is very suspicious of tuberculosis. It is erroneous to look for rales always at the apex. Many grave types of phthisis showed rales first in the axilla or in the parasternal region below the clavicles or in the inter scapular regions behind. A careful and repeated sputum examination for bacilli is essential. If five careful repeated weekly examinations are negative it denotes as a rule a fair prognosis, though there are exceptions. A bacillus negative case very rarely becomes bacillus positive unless the first set of examinations was carelessly made. A skiagram is very helpful but should be taken with a good apparatus and in the proper way. In the absence of X-Ray help physical examination alone has to be conducted and relied upon. When all the above criteria are negative a diagnosis of " no tuberculosis " can be made.

M. K. P.

ASSOCIATION NOTES.

Minutes of a meeting of the Executive Committee of the Association held on Tuesday the 6th November 1928 at 5-30 p. m. at the residence of the Honorary Secretary.

PRESENT:—Dr. M. Kesava Pai, *President* ; Drs. G. Srinivasamoorthy ; P. V. Cherian ; P. Venkatagiri and K. Koman Nayar.

1. The Annual Report and the Auditor's Report for 1927-28 were read and adopted.

2. The date of the Annual General Meeting was fixed for 1st December 1928.

3. The letter drafted by the President to the Vice-Chancellor, University of Madras, regarding the terms of award of the Raja of Panagal Gold Medal was approved.

Minutes of the Annual Meeting held on Monday the 10th December 1928 at 5-30 p. m. at the Medical College, Madras, with Rao Bahadur Dr. M. Kesava Pai the President in the Chair.

There were about 25 Members Present.

1. The Annual audited statement of accounts was taken as read and adopted.

2. Read letter from Rao Bahadur Dr. T. M. K. Nedungady regarding the evidence of our Association would like to give before the Committee on the reorganisation of Medical Education. It was decided that the questionnaire to be prepared by the Committee be awaited before the Association discusses the question.

3. Read the reply received from the Registrar, University of Madras, regarding the Raja of Panagal Gold Medal, stating

that the Syndicate considers the suggested alteration impracticable and inconsistent with the terms of the endowment'.

Resolved that the matter may be left over for further consideration.

4. Read the letter from the Secretary, All India Medical Conference, Calcutta, regarding the formation of a Central Association. Their appeal was circulated at the meeting and the letter was recorded.

5. Resolved that the subscription for the Madras Medical Journal for Non-members be reduced to Rs. 2-8-0 per annum as the Journal has been made a quarterly.

6. There was general discussion at the meeting, in which several members took part, regarding certain grievances of the Officers who held temporary commissions in the I. M. S. during the great war. The following resolution was finally proposed.

Proposed by Capt. A. Krishnamoorthi, B.A., M.B. & C.M. :—

That with a view to suggest remedial measures, a Committee (consisting of the undermentioned) be appointed to enquire into certain grievances of—

(*a*) Those permanent members of the Madras Provincial Medical Service who volunteered their services as Temporary I.M.S. Officers on general service (in or out of India) during the last Great War ;

(*b*) Those permanent members of the Madras Provincial Medical Service who volunteered their services as Temporary I. M.S. Officers for service in India only during the last Great War.

(*c*) Those temporary members of the Madras Provincial Medical Service and others who volunteered their services as Temporary I.M.S. Officers during the last Great War and who were subsequently taken as permanent incumbents in the Madras Provincial Medical Service,

Members of the Committee.

For Group A.—Capt. N. Mangesha Rao, M.B. & C.M., F.R.C.S.
„ T. V. Rajaratnam, M.B. & C.M.
„ M. Narasingha Rao, M.B.B.S.
For Group B.—Capt. U. Ananthayya, M.B. & C.M.
„ B. V. Varadachari, B.A., M.B. & C.M.
„ M. Sanjeeva Rao, M.B., B.S.
For Group C.—Capt. M. Subba Rao, M.B. B.S.
„ Benjamin, L.M.S.
„ S. Thambiah, B.A., L.M.S.

Captain T. V. Rajaratnam, M.B. & C.M. (Chairman).

The proposal was seconded by Capt. N. Mangesha Rao, M.B. & C.M., F.R.C.S., and was unanimously carried by the members present at the meeting.

7. The voting papers for the election of Office-bearers for 1929-30 were scrutinised by Drs. Benjamin, Bhavanishanker and Prabhu at the request of the President. The result was then announced which was as follows :—

President :—Rao Bahadur M. Kesava Pai ; *Vice-Presidents* :—Rao Bahadur P. Krishnaswamy and Khan Bahadur Dr. M. Azizulla Sahib.

Members of the Executive Committee :—Drs. C. Ramamurty ; C. Abbu ; K. Koman Nayar ; S. Swaminathan ; G. Srinivasa Murty ; P. Venkitagiri ; P. V. Cherian ; E. Achuta Menon and A. S. Mannady Nayar.

Minutes of a meeting of the Executive Committee held at the President's house on Thursday the 10th January 1929 at 5 p.m.

In the unavoidable absence of the President Khan Bahadur Dr. Azizullah Sahib presided.

PRESENT :—Drs. Azizullah Sahib ; G. Srinivasamurthy ; A. S. Mannady Nayar ; E. Achuta Menon ; P. Venkitagiri ; P. V. Cherian ; K. Koman Nayar.

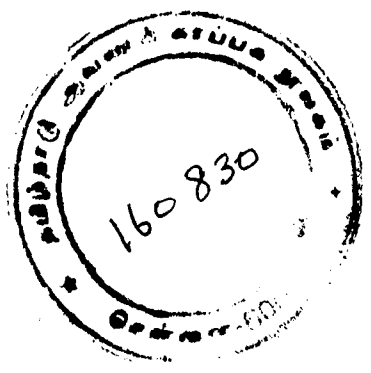
1. Resolved that Drs. Koman Nayar and Mannady Nayar be elected Honorary Secretaries and P. V. Cherian as Honorary Treasurer for the year.

2. Resolved that Dr. P. V. Cherian be elected Editor of the Journal.

3. Read letter from the Commissioner of Police, Madras, to the Superintendent, Royapuram Hospital, Madras, dated 9th November 1926, in which it states that the rate of carriage hire allowed for attending the Courts for purposes of giving evidence were fixed at Rs. 3-0-0 per diem for European Officers, and at Rs. 2-0-0 per diem for Indian Officers.

This letter was brought to the notice of the Committee by one of the members of the Association and was discussed at the meeting.

It was resolved that representations may be made in the proper quarters by the President so as to get this grievance redressed.



OBITUARY.

THE LATE DR. V. G. MUTHAYYA.

With deep regret we record the rather sudden death, after a few days illness of Dr. V. G. Muthayya, Civil Assistant Surgeon, Government Ophthalmic Hospital, Madras, at his residence in Egmore at about 6 p.m. on the 31st January 1929. Born in February 1880 at Palamcottah in the Tinnevelly district, he took to the study of medicine at a very early age and qualifying as a Sub-Assistant Surgeon entered the service of Government on 1st April 1902. After spending the first five years of his service in the Muffassil he was posted to the Royapettah Hospital, Madras. A few years of his good work there drew the attention of the authorities and his services were transferred to the Government Ophthalmic Hospital, Madras, nearly 20 years ago when Lieut.-Col. R. H. Elliot, I.M.S., was the Superintendent of the Hospital. His good work at this institution thereafter for such a long and continuous period has won the esteem and praise of successive Superintendents of the Hospital. He was in charge of the Refraction Department of the Hospital for a large number of years and his services there have been largely appreciated by all classes of patients visiting the department, no less by his superiors and colleagues at the Hospital. In recognition of his good work the Government were pleased, on the recommendation of his superior officers, to promote him to the grade of a Civil Assistant Surgeon in April 1927. The staff of the Government Ophthalmic Hospital have suffered an almost irreparable loss in his untimely demise, the Government service has lost one of its valued specialists and the public one of its ablest Refractionists. We convey our sympathy and condolences to his family in thei· sad bereavement.

THE LATE DR. B. V. NARASIMHAYYA.

We regret to announce the untimely death of Dr. B. V. Narasimhayya on 13th January 1929 at the General Hospital, Madras, after a short period of illness. After a successful College career he entered the service by competition standing first in the list of successful candidates of his batch. He joined the Service in September 1913. After working in the General Hospital, Madras and in the Districts he joined the staff of the Medical College, Madras, in 1918 as Assistant to the Professor of Surgery. In 1921, after qualifying himself in Dental Surgery, he was posted to the Northern Circars and was a lecturer attached to the Medical School at Vizagapatam from 1923. When the Vizagapatam Medical College was started he was appointed Lecturer in Dental Surgery, which place he filled with distinction till the time of his death. A person of quiet and amiable manners, keen and enthusiastic in his work, interesting in debates, well informed in various of medical and surgical activities, the death of Dr. B. V. Narasimhayya has created a void which it will not be easy to fill. We offer our sincere condolences to the members of the bereaved family.

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