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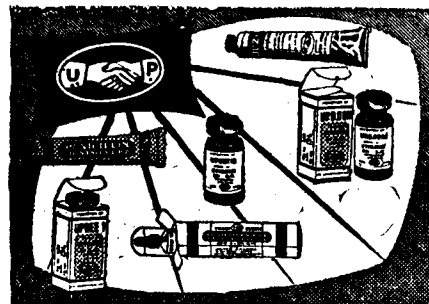
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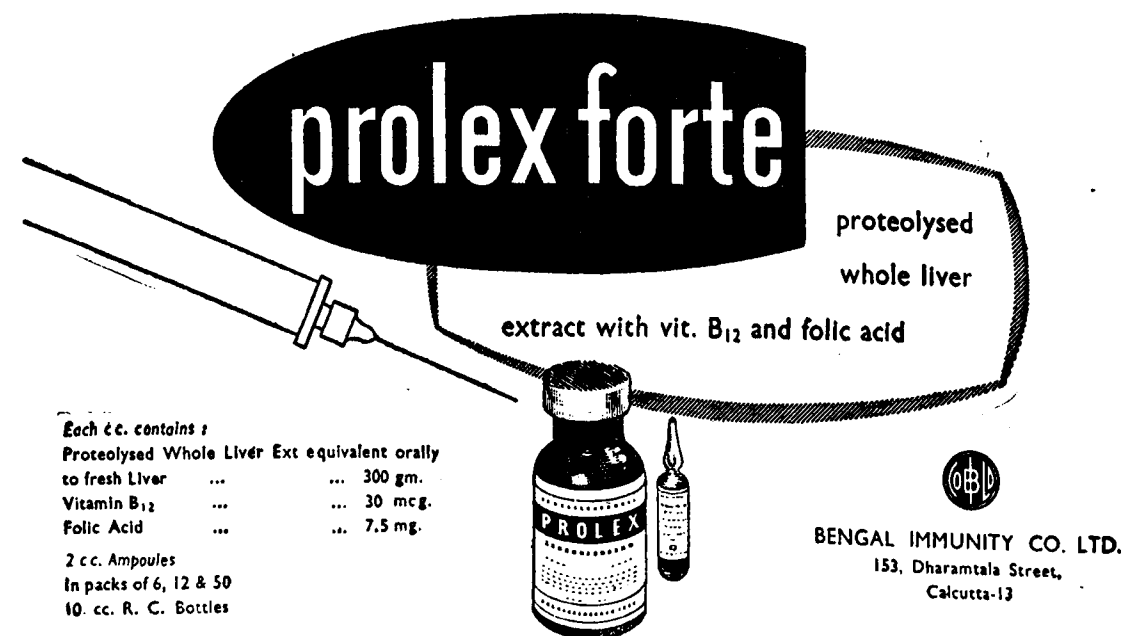
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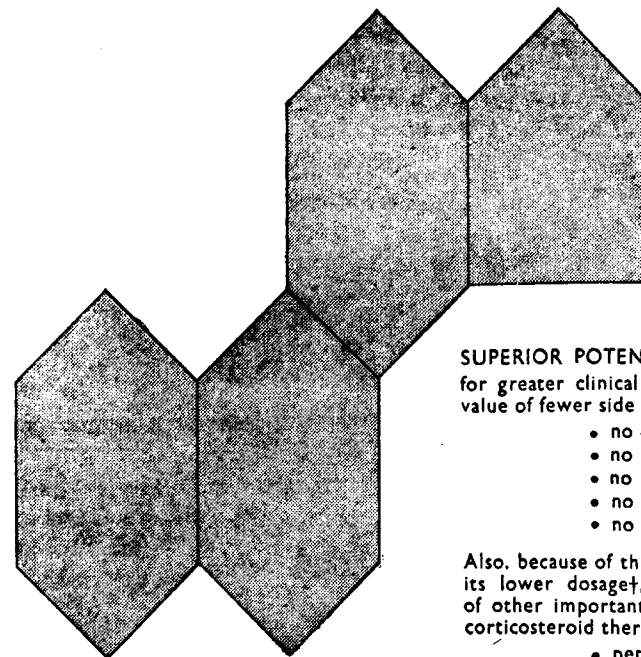
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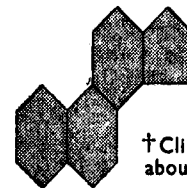
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THE MADRAS CLINICAL JOURNAL

JOURNAL OF THE MADRAS STATE BRANCH OF THE
INDIAN MEDICAL ASSOCIATION

(With which is incorporated the "Miscellany")

Vol. XXVI]

NOVEMBER 1959

[No. 5

CLINICAL MEDICINE AND THERAPEUTICS (Contd.)

by

DR. T. V. VENKATESAN. M.B.,B.S., F.A.I.M., F.C.C.P., (U.S.A.),

F.I.A.A., F.A.C.A., F.D.S. (Lond.), Madurai.

First we shall deal with the Cardio-vascular diseases.

A physician's resources are taxed to the utmost in the management of essential hypertension. In the management of this disease, it must be remembered that the blood pressure readings are of no value. An experienced motorist setting on a long run is less concerned with the tyre pressures which must vary with the bump on the road than the wear and tear of the tyre covers. Similarly, we must be concerned with the condition of the vessel wall. Hence a fundus examination is essential. According to this, the blood pressure cases can be graded into four groups. In grade I there is minimal arterial narrowing with slight or no symptoms and with normal renal function. In grade II, the retinal vessels show sclerosis. There is slight morning headache and vertigo. The urine contains a trace of albumin. In grade III, the retinal vessels show sclerosis and the fundus shows exudates, star figures, cotton wool patches and haemorrhages. The symptoms are dyspnoea, frequent headache, fatigue and nocturia. The renal function is impaired and the urine contains albumin

and casts. In the IV grade, in addition to the changes described in grade III for retina, there is pailloedema. The symptoms are severe headache weakness, loss of weight, confusion and dyspnoea. The renal function shows progressive deterioration. For grade I and Grade II, the total Alkaloids of Rauwolfia as Raudixin is preferred. In Grade III and Grade IV, ganglion blocking drugs as Mecamylamine is given. The blood pressure is estimated in the standing position 4 hours after the dose has been given. Each dose is then increased every third day by 2.5 mgms. until the systolic blood pressure in the upright position 3 to 5 hours after the dose is 120 to 140 mm. mercury. My experience is that the patient does not require more than 10 mgm. twice daily. Twice daily administration is usually sufficient to maintain an effective control of blood pressure for the whole 24 hours period. Because of the added fall and assuming the upright position, it is important that during night, patients could sleep at an angle of 45° by using a back-rest. On this regimen, papilloedema, exudates and haemorrhages disappear rapidly and congestive cardiac failure or left ventricular failure is rapidly relieved. The

side effect is constipation and can be relieved by neostigmine 15 mgm. twice daily and a daily dose of Saline laxative.

Hydralazine (apresoline) produces fall of blood pressure when given by mouth. The drug is recommended when there is renal impairment starting with a dose of 10 mgm. thrice daily and gradually increased to 200 mgm. thrice daily. The toxic effects produced are (1) severe headache (2) tachycardia (3) nasal congestion (4) dependent oedema (5) angina (6) arthralgia (7) lupus erythematosus like rash. In my experience, these toxic manifestations naturally diminish the value of hydralazine in treating hypertension.

In the treatment of chronic heart failure, diuretics play an important part. Two latest drugs deserve mention (1) Amisometradine or Rolicton and (2) Chlorathazide or chlorotride. Rolicton inhibits the tubular reabsorption of sodium ions by mechanism which does not involve succinic dehydrogenase like mercurial diuretics or carbonic anhydrase. It is claimed that it does not interfere significantly with normal potassium metabolism. The dose is 400 mgms. three times a day for 2 days given alternately with mersalyl. Except nausea, no side reactions are observed. Chloro-thiazide is an effective oral diuretic and acts by inhibiting carbonic anhydrase. It is said to be less toxic than diamox. Dose is 0.5 gm. thrice daily for 3 days. It leads to loss of potassium and hence potassium 2 gms. to be given daily. It has been found to potentiate the action of hypotensive drugs in the management of hypotensive failure.

Diseases of the Respiratory System

In the management of pulmonary tuberculosis, two new antitubercular drugs have come into vogue recently.

Viomycin is derived from streptomyces floridiae and is useful in streptomycin resistant tuberculosis in a dose of 2 gm. every third day. The toxic manifestations are eighth nerve damage, skin rashes and electrolyte imbalance. Cycloserine is a broad spectrum antibiotic and in capsules of 250 mgms. is given thrice daily with Isoniazide and is par excellence in pulmonary tuberculosis. The toxic symptoms are insomnia and tremors.

Asthma

Bronchial Asthma is a fascinating subject with an interesting definition. It is defined as an inherent instability of the vasomotor portion of the vagus nucleus caused by peripheral stimulation, psychic stimulation and foreign proteins producing broncho motor spasm and vaso motor turgescence. The main advances in symptomatic treatment of asthma lie in the use of corticoid substances and derivatives. The best measure is to give Prednisolone 5 mgm. 20 tablets in the following dosage :

Day I & II	1 Tablet 4 times daily
„ III & IV	1 „ thrice daily
„ V & VI	1 „ twice daily
„ VII & VIII	1 „ at night.

This is effective in controlling exacerbations in a high proportion of cases. In a case of severe status asthmaticus, an initial administration of hydrocortisone hemisuccinate 100 mgm. I.V. followed by oral prednisolone is effective.

Alimentary System

Heptocellular failure is a real problem to the Medical Practitioner. The treatment should be directed along the following lines — the correction of any (i) precipitating factors (ii) general measures (iii) treatment of neurological complications and (iv) management of patients with ascites.

(i) *Precipitating factors* :—Gastrointestinal haemorrhage or fall of blood pressure necessitate blood transfusion. Acute infection should be dealt with. Alcohol should be eschewed.

(ii) The general measures are bed-rest, a nutritious diet containing 3,500 calories with carbohydrate, moderate proteins and low fats. Vitamins as vitamin C, B-1, B-2 and B-7 are to be given daily. Methionine and Choline have failed to influence the disease. On the other hand, methionine may precipitate prehepatic coma. Intramuscular liver extract is good. Morphine and Pethidine are to be avoided.

(iii) *Treatment of neurological complications* :—Once neurological complications occur, all dietary proteins are to be stopped. Glucose should be given by intragastric drip. Liberal amount of fruit juices supply potassium. Parenteral Vitamin K prevents bleeding. Of the various antibiotics used, oral neomycin is very effective in decreasing gastrointestinal ammonium formation. In acute stage 6 gm. daily is given in divided doses and is maintained for one week. Corticoid hormones have met with little success in hepatic coma. Davidson found that large amounts of glutamic acid 100 gm. daily has some benefit. In control of ascites, repeated tapping leads to depletion of large quantities of proteins and this is particularly dangerous since the liver has a reduced capacity of synthesise albumin. Treatment should be directed to lower portal pressure and raise serum albumin. Salt-poor albumin is given and to reduce portal pressure the measure is only surgical.

Metabolic diseases : In the management of diabetes, mellitus oral hypoglycaemic drugs have come into use. Tolbutamide (D. 860) is a later drug and the limitations of this treatment should be well understood. Patient selected for tolbutamide therapy should have the

following criteria — (i) developed diabetes after the age of 40. (ii) show no previous tendency to develop ketosis easily (iii) their insulin requirements should not have exceeded 40 units a day. (iv) their fasting blood glucose when not taking insulin should not have exceeded 300 mgm. per 100 cc.

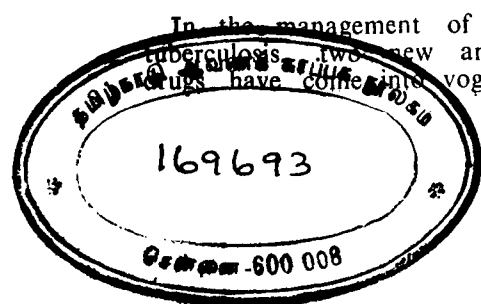
Tranquilisers

Stress and strain of life play a great part in causing and aggravating many diseases. Hence an account of tranquilizers is SINE QUA NON. Tranquilizers are drugs which cause sedation without clouding consciousness. They are classified into two main groups—(1) the first comprises of drugs used mainly in psychosis. They are (i) reserpine (ii) chlorpromazine. (2) Drugs mainly used in neurotic and normal people that lag the profound effects on brain metabolism like the first group. In fact nothing is known about the biochemical side of their mode of action. Four substances used are — (i) oblivon (ii) benactazone (iii) atarax and franquel (iv) meproamate.

Group (1) — (i) Reserpine — This interferes with the retention in brain tissue of 5 hydroxy-tryptamine and catecholamines. The dose is 3 to 6 mgm. a day. The side effects are in bowel habits and jitteriness.

(ii) Chlorpromazine is a potent inhibitor of many enzymatic process. The site most consistently affected appears to be a region of reticular formation responsible for wakefulness and arousing. The threshold to stimuli is raised. Dose 200 to 400 mgm. a day. Toxic symptoms—Jaundice.

Group II (i) Methypentynol or oblivon in a dose of 0.5 gm. The side effects have not been noticed.



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(ii) Benactyzine - (Nutinal : Suavitil) - Dose - Tablets 1 to 4 mgm. four times daily. Specific contra indications - none.

(iii) Atarax - 0.1 gm. and franquel 1 gm. Has fewer side effects than Benactyzine but there is tendency to seizures.

(iv) Meproamate (Miltown : Equanil : Pertranquel) - Dose 100 mgm. four times daily. Side effects are skin rashes and dizziness.

Newer Hypnotics - Do we require new hypnotics? Yes. We require better drugs to handle the problem of insomnia. Barbiturates can be abused. Drugs like pethidine produce addiction. The newer hypnotics are (1) glutethemide or doriden (Ciba) is a synthetic compound. Moderately powerful hypnotics in dose of 0.25 gm. The side effects are nausea, skin rashes and occasional excitements.

(2) Methylprylone are Noludar Roche is given in dose of 100 mgm. at bed-time. Its tendency is to produce nausea, vomiting, constipation and pruritis.

Thalidomide or Distaval is a new hypnotic developed in Germany and given in tablet of 100 mgm. with no side effect.

Drugs used in Parkinsonism.

The recent drugs in this disease are (1) Artane 2 mgm. tablets thrice daily and increased to 6 tablets a day. The

side effects are nausea and vomiting, and heavy-headedness.

(2) Cogentin-start with $\frac{1}{2}$ tablet 1 mgm. and increase gradually. The side effects are dryness of mouth and occasional skin rashes.

(3) Kemdrin-5 mgm. tablets. The dose is $\frac{1}{2}$ tablet thrice daily. The side effects are blurring of vision and giddiness.

(4) Lysivane-50 mgm. tablets twice daily. The only disturbing side effect is mental foginess.

(5) Disipal is dispensed in 50 mgm. tablets and the dose is 1 tablet four times daily.

Recent Drugs in Epilepsy.

(i) Primidone or Mysoline is given from half of 0.25 mgm. tablets to one thrice daily. It rectifies angulation of conduct. It is useful in grandmal and psychomotor seizures. The toxic effects are rashes and confusions psychosis.

(2) Phenaurone 0.5 gm. twice daily is given and is effective in all forms of epilepsy but unfortunately it produces anorexia, nausea, liver damage or blood disorder.

(3) Milontin 0.5 gm. thrice daily is effective in Petitmal. The toxic symptoms are nausea, vomiting and occasionally haematuria.

(To be continued)

* UNCLE'S DAUGHTER — WHAT PRICE ?

By

Dr. E. BALAKRISHNAN, M.S., D.O., Madras.

IT is not my intention to preach Social Reforms from the Medical Platform, much less, to complicate the institution of marriage, which already faces multifarious problems of its own. I have selected this article due to the experience of seeing various genetic diseases, especially such diseases as Retinitis Pigmentosa, Macular Degeneration, Myopia Etc., which produce blindness in various age groups in South India. All of us are aware how common custom it is, to marry among the close relatives, such as uncle's daughter, Aunt's daughter, Sister's daughter and in Muslims between the first cousins. These marriages among the closely related individuals who have a common pedigree transmit certain type of general and ocular diseases to their children. Two related individuals are more likely to carry the same pathogenic gene which they would have derived from a common ancestor than would two unrelated individuals.

The discovery of powerful anti-biotics and the control of infections by modern methods of treatment made the infective diseases to be on the decline, adding to the health and longevity of human beings. But hitherto unrecognised diseases which are not due to infections and which are not anemable to antibiotics are on the fore-front, challenging the intelligence of man, trying once again to drown the society in an helpless pathos.

All of you are aware of the ravages produced by diseases like diabetes, haemophilia, sickle cell anaemia and various other general diseases which have genetic transmission. Similarly in the eye genetically transmitted diseases are being diagnosed more and more. A perusal of statistics will prove that, the Smallpox,

Gonorrhoea were the chief causes of blindness in the last century. But during the recent years a sample survey of the blind population carried out in Great Britain prove that 60 percent of blindness were due to congenital and hereditary and developmental defects in the age group of below 13 years. Hence it is necessary that the medical world should wake up to prevent these genetically transmitted diseases. This brings us a careful study of genetics. As all of you are aware each nucleus of a somatic cell carries 48 chromosomes which are arranged in 24 pairs. These undergo a longitudinal division during the division of a cell, so that each of the resultant two daughter cells contain the original number of chromosomes. But the division in a germ cell is not a longitudinal division, but a dissociation of the pairs of chromosomes. In consequence the two daughter cells of a germ cell carry not 48 chromosomes but 24 chromosomes. During fertilisation 24 chromosomes of each germ cell join together and constitute 48 chromosomes, the number belonging to a original somatic cell. These chromosomes carry a hereditary material of the body. Each chromosome carry a particular gene and these gene determine the characters of an individual. Many familial diseases are transmitted through these genes. The methods of transmission may be (1) Dominant inheritance (2) the Recessive Inheritance (3) Sex linked inheritance. Let us consider the ocular diseases that are transmitted by recessive inheritance.

The recessive inheritance means that the parents themselves do not suffer from a particular disease but transmit the disease to their children or grandchildren. Such diseases are Retinitis Pigmentosa.

* Lecture delivered by at the Tanjore branch of Indian Medical Association, on 31st May 1959.

Retinitis Punctate Albescence, Heridofamial Degeneration, Choroidal Sclerosis, Xeroderma Pigmentosa, Albinism, Myopia etc. Such of these diseases mentioned above were commonly found in Europe among children born of such consanguinous marriages. Marrying the first cousin was a common English Custom more prevalent in small confined community such as the Mountain valley small islands, where natural obstructions made communication with other centers of population particularly difficult in the small segregated groups of certain religious sexes, such as, Jews and Quackers, whose members had for many years been discouraged from marrying any but their co-religionists. It is consequently in such genetically insulated communities the abnormalities determine by recessive gene are particularly likely to be observed. The high incidence of colour blindness among the quackers is an important proof. According to the Statistics in England out of 40,000 marriages 39,600 would be at random and 400 would be among the first cousins. If that is so in England we can very well understand the number of such consanguinous marriages should be much more in South India. Probably at the rate of 10 in 100 which work out to be 4,000 marriages out of 40,000 marriages. Probably because of the increased number of such consanguinous marriages in the Moffusil areas, I was able to observe a large number of blind children and adults who were suffering from Retinitis Pigmentosa in Tanjore.

What is Retinitis Pigmentosa? It is degenerative disease of retina which starts usually some years after birth. The chief complaint of a patient will be of nightblindness. After some years it slowly progresses towards day blindness and by the time the patient reaches between 40 and 50 years of age, he may become completely blind. In very severe degrees of the disease the children are blind soon after birth. They may develop a

complicated cataract. The Fundus appearance will be as you see in that model. The optic disc will be atrophic yellowish waxy in colour, the vessels are contracted due to a degenerative disease of its wall. Bone corpuscle like pigments will appear in the periphery of the retina. As years advance the pigments appear in larger areas and the disc will become more and more atrophic leading to complete blindness. The Retinitis Pigmentosa may be associated with other hypopituitary symptoms called Lawrence Moon Biedl Syndrome. It may be also associated with macular degeneration. On many occasions R. P. (Retinitis Pigmentosa) may also be associated with other congenital defects as Polydactyly Syndactyly. Many times the fundus picture will be associated with Drusen crystals. The Disease is frequently found with myopia. There are various forms of this disease:

1. Retinitis pigmentosa associated with deafmutism, with mental deficiency, with ophthalmoplegia, with microphthalmos and with anophthalmos.
2. Acute variety of R. P. This is rare. The patient goes into blindness in a short interval.
3. R. P. with sinepigmento—where the fundus picture will be devoid of pigmentary changes, but all the same with atrophic disc and the patient will become blind.

Differential Diagnosis:

It is very difficult to differentiate it from Syphilitic chorioretinitis, because the Syphilis can cause same fundus picture. Even congenital syphilis will simulate R. P. in a new born child. Hence in all these cases Serum reaction of the blood is very important. (Khan or U.D.R.L.

The other diseases which produce night blindness are; Vitamin A deficiency and Liver Disorders. The chief cause of blindness of children in South India still continues to be Keratomalacia. Hence it is very important to advice the mothers a proper diet for her child. These keratomalacia children have diarrhoeas and night-blindness. I have seen many such children which were treated with all the antibiotics in the hope of curing the diarrhoea. Such treatment upsets Symptoms of the intestinal organisms and the diarrhoea becomes chronic. The mother feeds the child with diluted milk or barely water. The net result is the deficiency increases and the cornea ulcerates. With all this mismanagement if the child lives it becomes blind. All of you will agree that this is the usual story. Hence Vitamin A injections is very important.

Liver disorders as Obstructive Jaundice, Cirrhosis of liver and fatty degeneration of Liver produce night blindness. Hence it is important that these conditions should not be mistaken for R. P.

Aetiology of R. P. not known.

Ocular Investigations: The peripheral field chart show that there is a Ring Scotoma which is diagnostic of the condition. Of late Electroretinography is employed to diagnose the condition.

Pathology: Atrophy of the rods and cones and the migration of the pigment from the pigment epithelial layer. There is occlusion of the vessels due to arteriosclerosis. All that we know about this disease is that it is transmitted in recessive forms in the children born of consanguinous marriages. Even though transmission by Dominant inheritance and sexlinked inheritance is noted, the majority of the patients show recessive inheritance and most of them give history of consanguinous marriages of their parents. Hence it is important to prevent these marriages.

Treatment:—Various drugs were tried and were found to be futile. Placental Extracts by injection and by transplantations of placenta into the sub-conjunctival tissues of the eye were tried without any improvement. Injections of corticosteroids were of a temporary improvement and no cure.

The patients suffering from this disease are painfully aware of their fate on account of their experience after wasteful years of treatment. Hence they tend to choose professions like elementary school teachers, small handicrafts, music teacher etc. In one occasion the acclimatisation of the patient was so perfect that he used to visit picture house to enjoy the music and the conversation. During my stay in Tanjore I was called upon to give evidence in a case of a boy who was charged with illicit distillation. He was found to be suffering from night blindness due to atypical R.P. The crime was said to have been done at 7-30 P.M. Evidently it was a concocted case and the patient was released on medical evidence. In another case a person was charged to have murdered his opponent at night 7-30 P.M. Here again the patient was suffering from R.P. and his dark adaptation was very poor. Fundus examination of the Police Recruits and army personnel are very important to exclude this disease since they may have a normal vision but dark adaptation may be very poor.

There are other diseases as I have mentioned before like Albinism, Myopia etc. which are again transmitted by consanguinous marriages in a recessive inheritance. All of you are aware the ravages of progressive Myopia. It constitutes about 7 percent of total blind population. It produces complications as degeneration of retina, Detachment of retina, complicated cataract.

What then is the price that we pay for these consanguinous marriages? Statistics show that these incurable diseases are in

the increase due to longevity of man in this era of Antibiotics. Hence it is very important to bring to your notice, you the Medical Practitioners of this district in whose hands are the health and welfare of the people, to propagate the idea of random marriages and to discourage the consanguineous marriages. This idea of ours is not new. A deep perusal of Kamathupal in Thirukural will convince any reader that consanguineous marriages are discouraged, and I would like to submit to your notice that in Thirukovaiyar (திருக்கோவையார்) Agananooru (அகநானூறு) Nattirai (நற்றிணை) any number of evidences for propagation of the random marriages can be found. All of us aware how Sagodhara Kothra marriages were prevented in Manu Sastra, with idea of preventing marriages within a group of people.

It has been often mentioned that marriages are made in heaven. You may or you may not agree with the sentiments of this statement. But you have to agree

that the diseases that I have mentioned are transmitted in those marriages. Hence it is no wonder that in Kurunthogai (குறுந்தொகை) marriages among unknown people binded with the only bondage of love has been given a supreme importance as

“யாயும் யாயும் யாராகியரோ
எந்தையும் நுந்தையும் எம்முறை
செம்புலப் பெய் திர்போல
அன்புடை நெஞ்சம் தாங்கந்தனவே”
கேளிர்

Let us follow the advice of our ancients who at the end proved to be more wiser than the wisest in this atomic age.

Reference: Duke-Elder Volumes 1 and 3 Systemic Ophthalmology-Sorsby Genetics in Ophthalmology-Sorsby.

An Introduction to Pathology-Payling Wright.

Parsis Diseases of the Eye-by Duke-Elder.

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CORONARY INSUFFICIENCY (Contd.)

By

Dr. K. VENKATA RAO, M.D.,

Physician, In charge of the Chest Clinic, Govt. Stanley Hospital, Madras.

Diagnosis.

The features of coronary thrombosis with infarction are too well known that I need not enumerate them all here. All the text books give in detail the differential clinical features of Angina Pectoris and Cardiac infarction. Apart from the different clinical features such as prolonged excruciating pain with restlessness, signs of peripheral collapse, sometimes nausea with vomiting, there are other evidences which go to prove that cardiac infarction has occurred. The more important of these are the E. C. G. evidences with a typical S.T. displacement in the limb leads (may develop immediately or in a few hours to three or four days) blood leucocytosis evident after 24 to 36 hours, rise of temperature from the 2nd or 3rd day (after the signs of shock disappears) and most important it discovered the appearance of pericardial rub. Along with the fever and leucocytosis the E. S. R. is raised the degree of rise being in commensuration with the area of cardiac necrosis. The E. S. R. is also prognostically very important. It comes to normal only when the infarcted area is completely replaced by fibrous tissue with healing. Its sustained rise for some months after the episode indicates that the necrosed area is being still absorbed. It takes nearly four to five months to reach normal in a severe case with large infarction.

The Blood pressure may be normal at onset or somewhat raised and soon falls and becomes severely low with severe shock and after the shock is over it may rise and remain at a lower level than what the patients previous record was (if any) or may reach the previous level itself.

A careful and meticulous history taking is very important in the diagnosis of cardiovascular diseases. This is all the more true in the diagnosis of coronary insufficiency. Paul White gives the following values to various methods in the diagnosis of heart disease in general. He gives 45% to the careful history and 25% to the clinical examination, 10% to the sphygmomanometer, 10% to X-rays, 5% to E. C. G. and the remaining 5% to the other recent advances in the methods of diagnosis such as angiocardigraph, cardiac catheterisation, manometric pressure taking in the chambers of the heart and gas analysis of the blood in the chambers and great vessels. I agree with him and many serious cardiac diseases will be missed with dire consequence to the patient if a careful history is not taken. Cardiac pain cannot be diagnosed by any other method than a careful history. So also the efficiency of the myocardium, whose efficiency alone counts in prognosis will be missed without a proper history. If a man aged 50 or 60 years says that he has climbed up four stories without stopping for taking extra breaths and on going to the top he felt perfectly alright, then what other evidence or investigation you require to assess his cardiac reserve? Even with gross limitation of cardiac reserve all other investigations may be perfectly normal.

Careful history should note the following things:—

(a) When did he begin to get the first symptom of pain?

(b) Any preceding history of hypertension, diabetes, Syphilis et.

(c) How and when he gets the pain? If on walking after what distance? Does its onsets keep to the same distance?

(d) What does he do when the pain comes on?

(e) How soon the pain stops and what does he do after the pain stops?

(f) Situation of pain.

(g) Nature of pain - constrictive as if held in a vice or squeezing pain or a dull heaviness or burning or glowing, or pricking or stabbing.

(h) Where is it propagated.

Answer to these questions will in the majority settle the diagnosis of anginal or cardiac ischaemic pain. If seen during the pain there may be a rise in B. P. and the heart rate may be slow.

When faced with a case of coronary thrombosis the diagnosis is fairly easy. Agonising retrosternal pain, of squeezing character, especially behind the lower sternum and radiating to the usual situations or downwards to the abdomen with a tendency to defaecate or nausea and vomiting, marked restlessness in contrast to Angina (where the patient remains immobile like a statue) cold drenching perspiration, thready imperceptible pulse, muffled heart sounds or arrhythmias especially triple rhythm will at once reveal the true nature of the lesion. Sphygmomanometer will record a falling blood pressure and lungs may reveal basal moist sounds.

The following conditions may occasionally be confused with coronary thrombosis. They are, perforated peptic ulcer, gall stone colic, tabetic gastric crisis, acute haemorrhagic pancreatitis, acute appendicitis and acute intestinal obstruction. Pulmonary embolism (acute

cor pulmonale) may almost exactly simulate coronary thrombosis. Both have circulatory collapse, thready pulse, dyspnoea and cyanosis. Pain in chest though less common in the former may be also absent in the latter. If a source embolism such as phlebitis is detected then it could be suspected in pulmonary embolism P_2 is loud and there may be a basal systolic murmur and the neck veins may be prominent. E.C.G. may be almost similar but a very high take off of the S. T. wave or a short rounded inversion of the T. wave occurs only in cardiac infarction. Another condition that may come differential diagnosis is acute pneumothorax. It may come with sudden collapse and suffocation with a rapid thready pulse. There may not be the usual characteristic pain in this condition but in some cases of cardiac infarction there may not be any pain but only signs of collapse, dyspnoea, cyanosis etc..

Diabetic acidosis with coma may be simulated by coronary thrombosis. Spontaneous interstitial emphysema of the lungs may closely simulate coronary thrombosis. The most important and characteristic findings in the presence of unusual clicking, grunting, or crunching sounds synchronises with the heart beat.

Dissecting aortic aneurysm may come for differential diagnosis. Hypertension is present and tends to persist after the attack. No cardiac arrhythmias and no pericardial rub. There may be some fever but E. S. R. is not raised.

In a case of coronary thrombosis the diagnosis can be made in the majority at the bed side itself and it has to be made from clinical examination alone as there is no time to be lost in calling to the aid E. C. G. or other technical methods as the treatment has to be prompt and immediate. Once the acute condition is properly treated and immediate crisis is over we have to resort to other technical

methods for confirming our diagnosis. In this the E. C. G. is of the greatest help. The standard ideas alone give decisive evidence in the majority of cases. Further E. C. G. is also of help to know whether cardiac infarction has occurred or whether we were faced with a case of acute coronary insufficiency. Briefly in anterior infarction high take off S. T. segment in No. 1, limb lead with depressing of S. T. segment in No. 3, lead occurs immediate following cardiac infarction or within two or three days. In posterior infarction the opposite to this occurs. After a few days S. T. reaches the isoelectric line and even goes below this with the inversion of T. wave. In anterior infarction a 'Q₁' wave develops and 'Q₃' in posterior infarction. With healing the S. T. and even T. wave may become normal, but 'Q' wave generally remains permanently as a pointer to the previous occurrence, of cardiac infarction. Unipolar leads from V₁ to V₆ are also helpful in exactly localizing the area of infarction. It has been said that S. T. waves are not as specific as to 'Q' waves as the latter occurs only in cardiac infarction. Persistent elevation of S.T. segment may be due to the development cardiac aneurysm.

An addition to changes in the E. C. G. in the standard limb leads changes in the augmented limb leads and the unipolar chest leads should be noted. They are important especially in localizing and also in confirming the findings in the limb leads. The E. C. G. records taken at frequent intervals shows progressive changes because of the progressing pathology and this progressing of characteristic is of considerable importance in diagnosis; the changes due to anoxia etc. are likely to be transient or irregular rather than progressive. 'Q' waves are due to actual death of tissue or may be permanent if the area is large or may decrease in size or even disappear if the necrotic area is small and heals with a small scar. The

R. S-T. changes being due to injured but viable tissue, soon disappear (usually in several days) as the tissue become non-viable or recovers (with development of collateral circulation). The R.S-T. elevation is due to the current of injury transmitted through the partially damaged tissue at the periphery of the infarct. In leads facing the epicardial aspect of the infarct, R.S-T appears as an elevation and in leads facing the endocardial aspect R.S-T appears as a depression.

Later changes

Within a few days after the onset usually as the R.S.T. changes begin to recede the T. wave begin to change. In leads which faces the epicardial surface (leads where R.S.T. elevation occurred) T. decreases in amplitude and eventually becomes inverted while leads facing the endocardial aspect T. remains upright and even increases in amplitude. At a still later date weeks or months after the occurrence these T. wave changes may show regression and may even return to normal.

Anterior Myocardial infarction

Leads facing the epicardial aspect of the infarct (usually V 4, 5, 6 and a VL and I) show prominent and vide 'Q' waves, delayed or absent R waves, elevated R.S-T. segment, and later inverted T. waves,

Leads facing the endocardial aspect of the infarct (usually aVF and 3) show no 'Q' waves depressed R.S-T segment and upright T waves.

Posterior Myocardial infarction

The leads with face the epicardial aspect of the infarct (usually aVF and 3 of an exploring electrode in the oesophagus at the level of the left ventricle) show prominent 'Q' waves, delayed or absent R waves, elevated R.S-T segments and later inverted T waves.

The leads which may face the endocardial aspect of the infarct (aVL, I and often V 3, 4 and 5) show no 'Q' waves, moderate depression of R.S-T waves and upright T wave (often high in amplitude).

Complications

Shock is a feature of a severe attack and therefore is not a complication. Sudden death : The patient may die of increasing shock. But most of the patients who die in the attack complain of some discomfort in the chest, or pain and die before the medical man arrives i.e., in a few minutes. Cardiac arrest or ventricular fibrillation may be cause of sudden death. Others fall dead while on their legs, lecturing or doing some work.

The Arrhythmias : Every known form of arrhythmias has been witnessed during the attacks of acute myocardial infarction. Extra systoles both auricular or ventricular are the most common irregularities met with. Paroxysmal ventricular tachycardia a possible precursor of ventricular fibrillation is the most serious.

Rupture of the ventricle : The infarcted area giving way is another fatal complication, though rare excepting in mental Hospitals. This is likely to occur during the first two weeks following infarction (myelomalacia). Rupture of the ventricle septum can occur if the infarction is septal. A loud systolic murmur develops in the mid praecordium in the 4th or 5th space just to the left of the sternum. There may be engorgement of the cervical vein or perhaps a thrill over the mid praecordium.

Aneurysm of the Ventricle : This develops later when the necrosed area is replaced by fibrous tissue. X-ray shows a bulge on the ventricular wall and screening shows paradoxical movement. Mural thrombi from this may give rise to embolic phenomenon at any time.

Thromboembolic episodes: Most dangerous and frequent complications. Thrombosis may extend proximally or peripherally in the thrombosed coronary artery causing death of a larger area of myocardium, killing the patient. A second infarction can occur by the breaking of the original thrombus and lodging in a peripheral branch. It is to prevent these fatal complications that the anti-coagulant therapy is advocated.

Mural thrombi may get dislodged and lodge in the brain, extremities, spleen, kidneys or other organs.

Pulmonary embolism is supposed to be frequent. Source may be from the right ventricular surface of the septum or from the peripheral veins, from the lower limbs or pelvis. Factors forming thrombosis in the peripheral vessels are prolonged rest with inactivity of the limbs, lowered blood pressure and increased coagulability of the blood after the onset of myocardial infarction. These embolisms are frequent within the first four weeks.

Shoulder Syndrome or "Shoulder Hand" Syndrome.

Pain in the left shoulder joint or sometime right shoulder develops a few days to a few months after the infarction and at times in the hand and arm of either side. In some cases the shoulder becomes stiff painful and ultimately almost immobile (frozen shoulder). Less commonly the hand becomes stiff swollen and may show tropic changes in the skin.

Spontaneous recovery after a variable period may occur. I have not seen this complication in any of my cases excepting in one recent patient, a complaint of pain in the shoulders especially the right. But X-ray did not show any changes. Later the patient complained of mild joint pains or stiffness in the inter-phalangeal joints of both the hands.

Anginal pain may recur in some cases. In others who suffered from Angina prior to the attack the pain may not recur at all (the area, the seat of pain having become dead due to infarction).

Prognosis

The disease has a most unpredictable prognosis. Death may occur even in what looks like a mild case, whereas another with marked symptoms and signs of cardiac infarction may surprisingly survive. In general, cases with severe degree of collapse (cold clammy perspiration, dysnoea, cyanosis and a marked fall in B.P.) carry a greater mortality than a milder attack with few or no signs of collapse.

Pain does not indicate the gravity of the case. There are cases of cardiac infarction with little or no pain and yet the patients die in the attack.

Cardiac arrhythmias excepting paroxysmal ventricular tachycardia (heralds the advent of ventricular fibrillation) and ventricular fibrillation do not carry grave prognosis. They disappear in a few days.

Cardiac rupture is a complication that may occur during the first week or two. It cannot be predicted. This is common in mental patients.

Extension of the thrombus in the coronaries or dislodgement of a fragment of the thrombus in the coronary which may block another branch causing either extension or fresh infarction is a complication to be feared in the first two or three weeks (hence the rationale of anti-coagulant therapy).

If a case of coronary thrombosis has passed the first three weeks, the chances of recovery is great. It is said that prognosis is good in the younger age

group than in the elderly. But what I feel from personal experience of a limited number of cases, the chances of recovery are greater in the later age-group for by then a greater amount of collateral circulation would have been established in the heart of these people from milder or mild non-clinical infarctions.

It is said there is an all round mortality of 25% in cases of coronary thrombosis.

Deaths from cardiac infarction can occur either instantaneously or in five minutes or in five days or within the first five weeks or within the first five months or during the five years following the first attack. Following this five years period it is unlikely that he will die of heart disease.

Treatment Preventive :—

Curative :—a. Angina pectoris,

b. Acute coronary failure without cardiac infarction,

c. Coronary thrombosis.

Preventive

Cardiac infarction does not occur in cases where the coronary arteries are normal. Atheroma of the coronaries is the prerequisite pathology for the occurrence of thrombosis in the majority of cases. A block may occur also from the breaking down of an thrombotic ulcer, the detached piece blocking the artery further down.

So the prevention of coronary insufficiency is one of preventing the predisposing condition that favour atherosclerosis. Hypertension and diabetes are the two diseases in which atherosclerosis of the coronaries are frequently found. If possible these two diseases should be

prevented from developing. These two diseases are frequent in certain families and the members of such families should take particular care in avoiding factors that favours the development of diabetes and hypertension. Since there is no definite aetiology for these two diseases the preventive methods can only be in the direction of avoiding certain situation in life which favours the developments of hyper tensions and diabetes.

Briefly they are

Relaxation: One should set apart a period daily for relaxation after the days work and also he should take weekly holiday for relaxation.

Hobby: To cultivate a hobby which will divert the mind from the routine drudgery and which will be a welcome source of diversion especially to men after retirement from service or from profession.

Retirement: People must retire from active service after 55 years or atleast after 60 years and then lead a simple contended life with restricted activities.

4. Every individual should take particular care about his activities eating habits, exercise and relaxation from the age of 45 years to about 55 years. This is the period when most of the coronary death occur. A thorough medical checkup to detect any disease at its onset

is essential. A review of his diet habit, activities and rest must be done and proper advice taken.

5. Since after the age of 25 or 30 years no growth takes place and food is necessary for the supply of energy for the day's work and for the replacement of wear and tear (Katabolism) of the tissues, a drastic cut in diet is essential. Fat should be considerably reduced in the diet and also protein to the bare minimum. Protein should not exceed half a gram to the pound of weight. Even with regard to the fat, it is now presumed animal fat in excess are harmful leading to lipoemia and athrosclerosis. Vegetable fats are not harmful. If glycosuria is found then a proper diet should be prescribed taking into consideration the caloric requirements for the nature of the work of the individual.

6. A periodic check on weight is necessary. In this the weighing machine and the abdominal girth are very helpful. If the individual is of more weight then a proper reducing diet should be prescribed.

7. A simple course of breathing exercises will go a long way in preventing the advent of coronary disease. There are some simple physical exercises in which breathing exercise is also combined. This can be practised easily even by the aged person. These I shall describe in more detail when the treatment is discussed later.

—(To be continued)

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DISCHARGE FROM THE EAR — DIAGNOSIS AND TREATMENT

By

Dr. N. S. PALANIAPPAN, M.B., B.S., D.L.O., Coimbatore.

I FEEL greatly honoured to have been asked to address you on the subject, 'Discharge from the Ear, Diagnosis and brief treatment'. The title should not be misleading. I am going to limit myself to discharges only as against diseases and defects. I will be only mentioning certain complications and not the treatment which requires special technique.

cases is a *preventable disease*, and its presence can be taken to indicate negligence on the part of some one; be it parent, doctor or specialist.

Before proceeding further, I would like to recollect the broad anatomical feature of the external auditory meatus, Tympanic membrane and the middle ear.

Discharge from the ear generally originates in the middle ear. But it may also come from the external acoustic meatus in cases of Dermatitis, boils, etc., The running ear has been a problem to many of us. In my clinic about 60 per cent of the cases are ear cases; and out of which, 30 per cent are chronically discharging ear.

The external auditory meatus is a narrow passage connecting the outside with the middle ear cavity proper, but effectively intercepted by Tympanic membrane at its medial end. The canal is never straight except in young infants. There is slight curve in its length with the convexity directed upwards and forwards so that in an adult for a proper view of the tympanic membrane the Pinna should be held slightly pulled upwards, outwards and backwards. In infants owing to the non development of the bony external meatus the auricle has to be pulled downwards and backwards.

The discharging ear is demanding an increasing amount of attention lately because of the widespread use of operating microscope and the development of plastic operation in the ear (e.g.) Myringo-plasty and tympano-plasty. The running ear is a serious physical debility. The fact that in ordinary life it may pass almost unnoticed is one of its greatest dangers. For its symptoms may be so light that some sufferers are unaware of its presence. It is also necessary to realise that there is still a great deal of indifference amongst the public to these common forms of disease in the Ear and that further education among the public is necessary before the doctor will have the opportunity of treating suffering children in the stages at which cure can be effected. The most important point to be remembered is that discharging ear in the majority of

Tympanic Membrane. In health, the Tympanic membrane presents a highly polished grey surface of which posterior and upper part is distinctly nearer the eye than the anterior and inferior part. The colour alone is not sufficient for the recognition of the membrane. [Fig. 1.] The handle of the malleous also must be seen. At the upper end of the handle the short process is noted as a small projection and running downwards and backwards from this the handle of the malleous appears as a whitish yellow streak ending at a point the umbo below the centre of the membrane. Extending downwards and forwards from the umbo the cone shaped light reflex is seen. This

Lecture Delivered at the Erode Branch of Indian Medical Association on 29-8-1959,

is fairly constant in position. Immediately above the short process, the membrana flaccida or sharpnells membrane fills the gap in the Tympanic Ring. The normal membrane is easily recognized after a little practice. There may be a considerable difficulty in distinguishing many abnormal conditions. The short process, handle of the malleus and cone of the light reflex are the chief landmarks to be looked for. When, however, they are not detected, the observer must attempt to estimate the depth of the structure, at which he is looking. If it is obviously nearer the eye than the drum, the appearance may be due to Polypus, granulations, Wax, Foreign body etc.. When the appearance is not compatible with any condition with which the observer is familiar, the ear should be syringed or mopped out as the presence of even a very little puss or flack of wax may be quite misleading and give rise to incorrect diagnosis.

The middle ear is a narrow cleft like space in the temporal bone. Anteriorly it communicates with the Eustachian tube. Posteriorly the middle ear communicates by means of an opening with the tympanic antrum, and through that with the mastoid air cells. The middle ear cleft with the Eustachian tube, tympanic cavity and the air cells is developed from first visceral pouch of the embryo. The tympanic antrum is present from birth onwards. The mastoid process is absent at birth and is small in early childhood. The mastoid temporal bone may consist almost entirely of air cells with thin bony walls. This is called cellular or Pneumatic mastoid and forms about 80 per cent or it may be composed of mainly sclerotic bone. This is called acellular or dense mastoid and forms 20 per cent. Normally the two mastoid processes are symmetrical and their cellular development is identical. The cells are arranged in a definite pattern, and they are grouped according to their anatomical position. These groups are ; zygomatic

cells, Peri Antral cells, Attic cells, Tip cells, Tubal cells etc., Apart from fenestration operation for Otorclerosis Endaural approach for mastoidectomy is not quite suitable in a well developed cellular mastoid. Even in Post Auricular approach there is a likelihood of not opening some of the cells and hence some cases results in otorrhoea even after mastoid operation, on the other hand endaural approach is ideal for sclerotic mastoid.

Reaction to Infection. With a non-pneumatized or acellular mastoid the re-action to infection is often proliferative. This may give rise to acute Polypus through a perforation in sharpnells membrane. It is an undoubted fact that the very great majority of cases of chronic otitis media and its complication occur in subjects with poorly pneumatized mastoid, though of course acute mastoiditis does not occur in them, and they may not respond for conventional treatment. Chronic otitis media in a Pneumatized mastoid often respond favourably to conservative treatment.

The primary function of the external and the middle ear is to convey sound vibrations to the cochlear mechanism and for this purpose maintenance of equilibrium of air pressure on either side of the tympanic membrane is a compulsory factor. Similarly, the presence of intact mucous lining with columnar epithelium in the whole of the middle ear is necessary for efficient functioning of ossicles.

There are certain basic Accessory anatomical features which inadvertently aid and abet diseases of ear. Foremost among them are Adenoids and Tonsils. No doubt they have their own function. But in a great majority of Discharging Ear cases, they are inflamed, infected and septic. When by sheer continuity of tissues alone, the ears are affected,

Indirectly the presence of infection in the teeth and paranasal sinuses also start diseases in the ear.

Examination of the Ear : For a proper examination, a good source of illumination is necessary. An otoscope is quite convenient. There is one essential prerequisite for the successful examination of the ear which must be strongly emphasised and this is proper cleansing of the external auditory meatus before examination. Lack of this precaution is probably responsible for more errors than any other single cause. Until the drum membrane is perfectly seen and clearly seen, especially the attic region, it is almost useless to form conclusions. Cleansing should be done with the ear syringe or by gentle mopping.

Of all the other observations that of hearing is the most important and this should always be examined with care. For, especially in cases of acute middle ear disease, it should always be borne in mind that the preservation of hearing is the chief aim. Conversation voice, whispered voice and tuning forks are the usual media for a consulting room testing. After a complete examination of the affected ear, the opposite ear should also be examined followed by an examination of the nose, throat and if necessary, the whole body.

History of the case is very important. The following facts must be ascertained :

1. **Onset :** When taking a history of otitis media, the fact must be established that it is a recent discharge, because acute symptoms in a chronically discharging ear must be taken more seriously.

2. The nature of the discharge whether constant or intermittent, whether odourless or offensive, mucoid or purulent.

3. The degree of deafness.

4. Are there any noises in the ear or giddiness ?

5. Is there any deep throbbing pain or head-ache ?

How the nature of the discharge helps in the proper diagnosis of the extent of the disease ?

If Mucoid : The infection is from the Nasopharynx and if it is influenced by attacks of cold, the cause of the condition mostly lies in the adenoids and sinuses.

If the discharge is frankly purulent it reveals the necrosis of the underlying bone.

If blood stained, it should suggest granulations in the middle ear or if blood stained and offensive, a Vincent's infection from outside must be thought of.

If the discharge is greenish or bluish in colour, it is often caused by *Pseudomonas pyocyaneus*. If the discharge is offensive or foetid it is associated with bony necrosis or cholesteatomas.

The discharge in the ear is highly variable in quality and quantity. Thus it may be thick or thin, scanty or profuse, Mucoid, Muco-purulent or frankly purulent serous or sero sanguineous, with or without foul smell. It is also a noticeable fact that most of the patients remark that their ear discharge is maximal at night and early mornings. This is mainly due to the recumbent position assumed at nights. Only in chronic disease of the ear discharge though scanty is found alone and as a matter of fact, pain in a chronically discharging ear should put us on guard against occurrence of complications. Any incidental upset in the general health often adversely influence the discharge in the ear.

The position of the perforation is also considerable importance.

1. Perforation of the *anterior part* of drum head are seldom of serious significance as regards danger to life. They indicate a chronic Tubo-tympanic infection and call for treatment of nose, Pharynx and Eustachian tube.—Fig. 2.

2. *Central perforation* in the post inferior part is associated with suppuration in the lower part of tympanic cavity.—Fig. 3.

3. A combination of the above two results in a central *Kidney shaped* perforation on the lower half of the drum head. These three kinds of the perforation may be regarded as favourable from the point of view of prognosis, because intracranial complications are seldom met with such cases.—Fig. 4.

4. *Marginal perforation* in the post-superior part is associated with earies of the underlying bone.—Fig. 5.

5. *Attic perforation* of shrapnells membrane indicates cholesteatoma. In order to observe this attic perforation the patient's head should be tilted towards the shoulder of the side opposite the ear under examination. The last two types of perforation will not respond for conservative treatment.—Fig. 6.

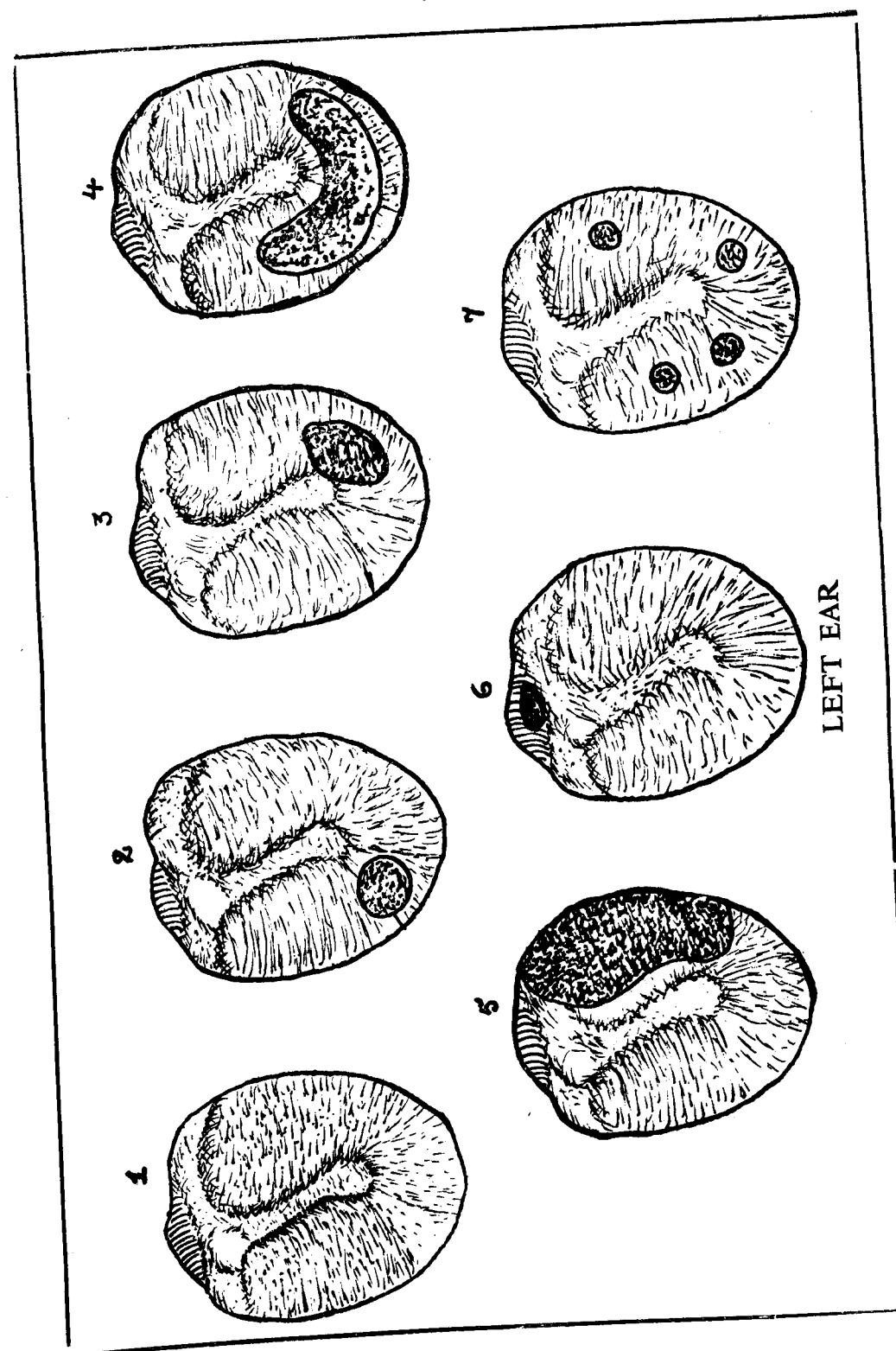
6. *Multiple Perforations* with thickening of Membrane arouses suspicion of Tuberculous origin. Primary focus is elsewhere Facial palsy, painless Otorrhoea, Enlarged cervical lymph nodes are symptoms of T. B. otitis—Fig. 7.

Etiology: Socially the chronic Ear discharge is far more common among the poorer section of the population than among the more wealthy; but this may be explained by the fact that among the latter acute inflammation of the

ear is rarely allowed to become chronic. The tendency of poorer people to acquire frequently acute upper respiratory infections while living among not so satisfactory sanitary surrounding is also a factor. Hence, we find that a much larger percentage of patients require mastoid operations in the hospital than in private practice.

Acute inflammation in the middle ear occurs frequently during acute coryza and in influenza. It is a very common sequela of exanthema, especially measles and scarlet fever. In all these cases, infection reaches the ear from the upper respiratory tract, via, the Eustachian tube, especially with faulty drainage in the tympanic cavity. It may be caused by too vigorous syringing of the Nose and by operations on the Nasal cavities and the Naso-pharynx. Packing the nose or plugging the Naso-pharynx in severe cases of Epistaxis may give rise to acute middle ear suppuration, especially if the plugs are left in for more than 24 hours. Otitis media may also result from bathing in the sea or public baths during which water may enter the nose and pharynx and so infect the Eustachian tube and tympanic cavity. Bathers who get water into the nose and naso-pharynx should not blow the nose violently, but should allow the water to run out. Violent blowing of the Nose drives the fluid in the Eustachian tube and if the Naso-pharynx happens to be infected than acute ear discharge results. For the same reason it is unwise to fly during the course of a head cold. The patency of the Eustachian Tube is affected and negative pressure results during descent and causes acute Barotrauma.

The second route of infection is via the external meatus following injuries of drum head. Fracture of skull involving the temporal bone are sometimes followed by otitis media - the infection coming either by way of ruptured membrane or Eustachian tube.



Rarely otitis media may follow suppuration of the labyrinth resulting from meningitis, the infection invading through the oval or round window.

The chief pre-disposing causes are the presence of adenoid vegetations in the Naso-pharynx and a hereditary disposition to catarrhal affections of upper respiratory tract.

In Infectious diseases cases those cases which have nasal purulent complications go in for acute otitis media. In order to prevent such condition in children suffering from infectious diseases, it is recommended that a few drops of a 10 per cent solution of Argyrol should be instilled into each nostril three times a day.

Causes of persistent of discharge : If all cases of acute otitis media were treated efficiently, then there will be great demerit in the number of chronic cases. One or more of the following may be the factors of persistent otorrhea.

1. *Disease of the underlying bone :* The original infection may have been of such severity that there has been bone destructions either in the mastoid cell system or in the middle ear itself or the ossicles (incus - due to its poor blood supply).

2. *Granulations and polypi.* Granulations may be formed by the destruction of lining membrane of the middle ear or a portion of the lining membrane or the granulations may be drawn through a perforation in the drum becoming a polypus which grows steadily until it may on occasions project from the external meatus.

3. *Cholestatoma :* Cholestatoma consists mass of epithelial debris in which cholesterol crystals are deposited. It generally appears as a smooth glistening and pearly body. But in other and more septic

cases it looks like a mass of putty and smells abominably. This mass though inert in itself may by the steady pressure of growth erode the vital structures surrounding the middle ear and may eventually invade the brain also.

4. *Persistent Nasopharyngeal Disease :* *Middle ear suppuration in children :* There is very definite relationship between otitis media and gastro enteritis in Infancy. The Eustachian tube in infants is short and wide and nearly horizontal than in adults, and discharge from an infected middle ear can drain quite easily into the pharynx and then be swallowed with the possible productions of gastro enteritis. On the other hand, the infection of the middle ear may take place by the reverse method and vomit from a case of gastro enteritis may be forced into the Eustachian tube and a vicious circle so established. During the first two years aural suppuration is more frequent than at any other subsequent age. In the vast majority of cases, infection is the result of a common cold.

This condition is more common in bottle fed babies than in the breast fed babies. (1) Milk in the bottle is more likely to be infected than breast milk. (2) Breast fed baby is often healthier and its resistance is greater. (3) Feeding position of lying flat facilitates regurgitation.

Diagnosis : Once there is discharge in the ear even in acute otitis media, there is lessening of pain and improvement of hearing.

If there is persistence or increase of pain and if there be increase of deafness then it is a sure sign that the disease is not clearing up or that some complications are to be expected. The presence of tenderness over the mastoid or post auricular swelling marks the presence of extension of disease. If there is acute pain in a case where there is painless discharge then the case is to be examined

thoroughly to ascertain the origin of the pain. Swollen and tender mastoid suggests mastoiditis with or without lateral sinus Thrombosis or brain abscess. Facial pulsus denotes extension of inflammation into the facial canal. But fortunately these dreaded complications are rarer now-a-days.

Treatment : Essential treatment of ear discharge resolves itself into two parts. The first part deals with measures where by the recovery of the function of the ear is given priority whereas the second part deals with preserving the life of the patient even at the expense of the function.

General Lines of Treatment as bed rest, warm surroundings are to be followed depending upon the severity of the case - acute or chronic. Treatment of the causative condition should not be forgotten.

Treatment is Directed Locally : (1) to the relief of pain (2) The maintenance of patency of Eustachian Tube (3) Neutralisation of Toxins.

Relief of Pain is with local warmth and prescribing sedative and analgesics.

Maintenance of Patency of Eustachian Tube : Instillation of nasal decongestants into the Nostrils usually a solution containing ephedrine is useful. The frequency depending upon the severity of the condition. It helps to free the air way. The nasal instillations are to be discontinued as soon as possible. It will be wise to order steam inhalations for which Tr. Benzoin is a very good vehicle. In these inhalations, it has been found that the vehicles do not play any active part, but it is the hot atmosphere that acts on the vasomotor condition of the nasal mucosa and brings about necessary changes to effective removal of all infected matter from the nasal passage. (3) Large and adequate dose of antibiotics is

necessary or else the symptoms will be "watered down", but not entirely eliminated. Later on a chronic condition or complications will result. Local treatment consists either conservative or surgical.

Conservative Treatment. Conservative treatment consists in ear toilet (i. e.) cleansing the ear and keeping it clean by wet or dry method. Cleanliness is the chief point. It is no use putting in more and more of drops everyday if the same were not to come in contact with the diseased areas.

Wet Method. The ear should be gently syringed with a warm and mild antiseptic solution such as a warm boric lotion. After syringing the ear clean, it should be mopped as dry as possible. The ear should be inflated and again dried with mops. The ear should be treated with drying drops such as boric acid in rectified spirit. The treatment may be repeated one to three times a day according to the amount of discharge.

Dry Method : Lately the tendency among specialist is to clean the meatus and then in placing a strip of ribbon gauze against the drumhead. This acts as a wick to the discharge and drains off the secretions by capillary actions. This line of treatment is very good, but the great drawback is that the gauze wick has to be inserted by direct vision and is quite unsuitable for home treatment. Instillation of Hydrogen peroxide for cleaning purposes of the discharge of the ear is condemned since there is a chance of infection being carried into nooks and corners. Insufflation of powders are also to be condemned for if it is allowed to accumulate in any quantity it gets mixed up with secretions and forms a hard cement like plug which acts as a cork to the drainage from the middle ear and thus causes retention of pus and also tends to promote by its irritation otitis externa.

Otic Drops: Several proprietary preparations are available. Chloromphenecol Ear drops and other broad spectrum Antibiotic drops are good to control secondary infections.

All coloured solutions (e. g.) Tr. Methiolate.

Acriflamine in spirit and other dye's should be avoided since they will mask the appearance in subsequent examinations.

Some common drops instilled into the ear are :

1. Ghee - Particularly cows ghee - older the preparation more effective that is the belief.

2. Urine - (eg) animals like - Ass - Probably for the antiseptic actions of Urea.

3. Gingelly oil - Chilles or garlic and warmed. Here it is the warmth that gives relief in acute otitis media and before the rupture of T. M

4. Glycerine carbolic drops - This causes moeration of the epithelium.

5. 10% Icthyol in glycerine - This is good in cases of External otitis and furuncles.

It may be stated generally that conservative treatment of chronic middle ear supuration by means of drops and syringing if efficiently carried out results in cure in many cases success being proportional to the thoroughness with which it is done. A further number may be cured by special methods such as syringing through Eustachian tubes, Intra Tympanic syringing or the use of zinc Ionization. Polypus must be removed as the first stage of treatment granulations well tend to disappear with aural toilet.

The process can be speeded up by cauterising the granulations with silver nitrate beed or chromic acid. In all cases complicated by the presence of adenoids in the Nasopharynx, the removal of these growths is called for as soon as the acute symptoms have passed off. Purulent sinusitis should be treated. There are occasionally cases of diffuse Lymphoid hyperplasia in the Nasopharynx particularly on the Eustachian orifices and there may not be circumscribed mass of adenoids suitable for surgical removal. These cases are best treated by radio therapy. Local application of radium or deep x-ray.

Classification of cases treated conservatively: It is found that most cases which finally become dry in two weeks time. At the end of two weeks, all cases given conservative treatment should be reviewed and they can be divided into three groups. The first is a large group with dry perforations. These patients must be instructed in the management of their perforation and to report immediately for treatment if the discharge reccurs. The few minutes spent in getting these patients to understand the nature of their disability are minutes well spent.

Water should not be allowed to enter the meatus. Sea bathing, underwater swimming should therefore be prohibited and a pledget of wool worn when the patient has a bath. Another group will have persistant smelly discharge from a marginal or attic perforation and will show no real improvement as a result of their two weeks treatment. These patients will require a mastoid operation.

There will be a third small group in which considerable improvement has been obtained by the treatment. But in which the ear remains moist. This group needs careful consideration some ears with large reniform perforations will appear healthy though moist and these

cases can be instructed in home treatment. Aural toilet and spirit drops and kept under observation perhaps at intervals of three months.

Since the advent of penicillin especially acute mastoiditis has been greatly reduced in its instances and by far the greatest number of cases of acute suppurative otitis media now resolve by early and adequate treatment by parenteral penicillin.

Indications for mastoid operation

(1) Pain and mastoid tenderness continuing for two or three days in spite of antibiotic therapy.

(2) Fever over 100° with rapid pulse.

(3) Copious pulsating Purulent discharge filling the meatus in a few minutes after mopping out and therefore too plentiful to be secreted by the tympanum and antrum alone - "Reservoir sign".

(4) Symptoms of intra-cranial complications such as headache, vomiting, rigois etc.,

(5) Granulations which recur rapidly after removal.

(6) Facial paralysis.

(7) Cholestatoma.

Surgical Treatment: A defect in the tympanic membrane has always been a provocation to otologists. The defect may be approached in one of the three ways. It may be plugged, it may be patched or it may be stimulated to close itself. **Plugs:** The simplest plug is the piece of cotton wool soaked in sterile paraffin which is used to occlude the perforation and contact the medial wall of the middle ear. However such an

artificial plug needs constant attention as it is apt to cause recurrence of otitis media and otitis externa.

Patches: The patches recommended include rubber, sized paper, colloidion, egg membrane, cigarette paper, gold silver foils and most recently human amniotic membrane.

Stimulations: Tricolour Acetic Acid applied to the margins of perforation repeatedly are sometimes attended by success. This method is only suitable for smaller perforation. Previous treatments have failed to prove entirely satisfactory. Recently attention has been turned to skin grafting and this is called Myringo Plasty and forms part of reconstructive surgery of the middle ear.

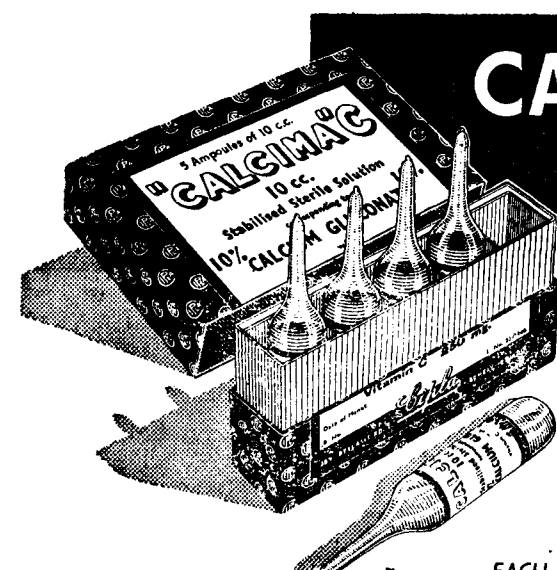
Functional Surgery is the catch word of our time and otology has not been insensible to its call. Labyrinth operation for meniere's disease plastic repair of the facial nerve, conservative surgery of the middle ear, fenestration have been in the last decades notable examples of the constant contribution of otologist to the functional revolution of the modern surgery. However, the preservation or restoration of hearing when opening a chronically infected middle ear and mastoid had proved so far one of the most difficult and deceiving tasks. We may observe that only a little more than 20 percent of cases have any hearing improvement after the classical radical mastoid operation. Practically none reaching a socially useful hearing level. The conservative or modified radical operation offers much better results with an average percentage of hearing improvements of 30 per cent and only a minor percentage of post operative hearing deteriorations.

What we really needed was an operative procedure enabling us not only to master the infection in every case, but also to pave the way to proper healing, restoring

its functional properties when they had been lost through disease process. Such a procedure has been developed and it has been named Tympano plastic by its authors. Thanks to it, we are now in a position to restore a socially serviceable hearing in about 60 per cent of all chronically discharging ears.

The principles of tympano plastic operations are essentially (1) good bone-conduction (2) Integrity of the labyrinthine windows (3) *Patent Eustachian tube*. By tympano plastic technique many and many cases of so called chronic tubarian otorhoea with central perforation and fairly good hearing in which operations are generally avoided on

account of bad functional results, for outweighing the advantages of a healed cavity are now gained back to surgery which makes possible the healing of the infection. The improvement of hearing and even more important the prevention of the progressive deafness which is unavoidable in these cases in the long run. The general opinion is that the old routine mastoid surgery should be abandoned in view of the recent progress or should be limited to the cases of impending or actual complications. The technique of the operation is now very well defined and standardised. Tympano plasty is a difficult surgical procedure, but well within reach of otologists trained in the micro surgery of the ear.



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* ENCEPHALITIS IN SOUTH INDIA

By

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During the last five years, a number of reports have drawn attention to the importance of encephalitis as a cause of morbidity and mortality in Indian Children. Table I shows figures for paediatric admissions to the Christian Medical College Hospital, Vellore for the past 3 years.

TABLE I
Paediatric Admissions—C.M.C.H.
Sept. 1955 — Nov. 1958

Disease	No.	Per cent of total
Encephalitis	53	1.6
Pyogenic Meningitis	31	0.9
Tuberculous Meningitis	75	2.3

Interest here has been focussed on this problem since 1955, when a Japanese B type virus was proved by Dr. Telford H. Work, Director at that time of the Virus Research Centre, (V.R.C.) Poona, to be responsible for an encephalitis in 14 children admitted in a 12-week period to the pediatric ward. From then until June 30, 1958, Dr. Sheila Pereira followed these first cases both in hospital and in their homes and studied all new cases of encephalitis admitted to hospital. A Field Laboratory was established at C. M. C. H. by the Virus Research Centre and a joint clinical, virological and epidemiological investigation of this problem has been maintained. The material which forms the basis of this article is largely the product of this collaborative effort.

"Encephalitis" as a clinical diagnosis is bound to embrace an assortment of diseases of very varied aetiology. Recognising this, and being anxious to study carefully all cases of probable viral encephalitis, we adopted the following criteria for this clinical diagnosis.

(1) There must be a definite onset of illness with fever.

(2) After a variable prodromal period, involvement of the central nervous system (C. N. S.) must be marked by disturbance of sensorium, drowsiness or frank coma. Convulsions, focal pareses, extrapyramidal or cerebellar signs may, but need not be present. If convulsions occur, the period of unconsciousness which follows should be more than can be accounted for by the fit, i.e. usually more than 24 hours after the fit.

(3) The cerebro-spinal fluid (C.S.F.) findings must be consistent with the diagnosis, i.e. bacteriologically negative and usually with a normal or raised cell count and normal or slightly increased protein and sugar concentrations.

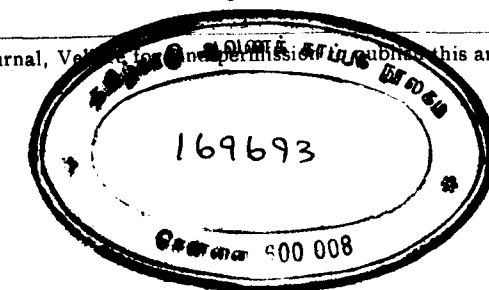
(4) Negative history and findings for measles, vaccination etc. must exclude one of the recognised forms of encephalomyelitis, associated with perivascular myelino-clasis.

Cases satisfying these criteria have included examples of Japanese B encephalitis proved serologically, of fulminating bacterial infection, and of

* We thank the Editor, C. M. C. Journal, Vellore for permission to publish this article.

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encephalitis which we presume to be due to viruses other than those at present known in the arthropod-borne group (arboviruses.)

The forms of encephalitis commonly encountered in Indian children are shown in Table II. Polio-encephalitis can usually be recognised clinically by the presence of lower motor neurone lesions, and encephalomyelitis complicating the exanthemata by the signs or history of the exanthem. In the present state of our knowledge (or ignorance,) the so-called North Indian encephalitis, or Jamshedpur Fever, can only be diagnosed at all confidently in the presence of an

epidemic sporadic cases of presumed viral encephalitis may well be due to the same organism as the epidemic cases, but in the absence of any specific diagnostic test this is conjectural. In S. India no epidemic of this N. Indian type of encephalitis has been recognised, and at Vellore we have found ourselves concerned with Japanese B encephalitis, sporadic cases of other "viral" encephalitis and toxic encephalopathies of bacterial origin. This article deals with Japanese B encephalitis as we have seen it, in all its aspects, at Vellore, these other conditions being discussed briefly and essentially from the point of view of differential diagnosis.

TABLE II
Types of Encephalitis in India.

Diagnosis	Aetiology
1. Japanese B encephalitis.	J. B. E. virus; infection identified by isolation of agent or serology.
2. North Indian Encephalitis (Jamshedpur Fever)	Enterovirus presumed responsible; known arboviruses excluded at Jamshedpur.
3. Polio-encephalitis	3 Types of Polio myelitis virus.
4. "Virus Encephalitis"	Probable but speculative.
5. Encephalo-myelitis	Measles, Vaccinia, Variola etc.
6. Toxic Haemorrhagic Encephalopathy	Typhoid and other bacterial infections; cerebral malaria.

Japanese B Encephalitis

In 1955 the diagnosis of Japanese B encephalitis was proved in 14 children, and in 1956 in a further 4. No cases were seen in 1957, but this year again, in the last three months typical cases have been seen though the diagnosis has

not yet been confirmed serologically. This year a number of typical adult cases have been seen for the first time though in these too, serological tests have not been completed. The following description is based on the 18 proved cases, with reference to a few points of special interest in the current cases.

Clinical Features:

The course of the disease can be conveniently divided into three stages, a prodromal stage before signs of C.N.S. involvement, an acute encephalitic stage marked by C.N.S. signs and continuing fever, and a late stage marked by recovery or the persistence of signs of irreversible neurone injury.

Prodromal Stage:

There is nothing specific about this stage which is marked by fever, headache, and occasionally vomiting, but one interesting feature has been its variable duration; the mean duration was 1½ days, but extremes have ranged from a few hours to 14 days.

Acute Encephalitic Stage:

Continuing fever, usually high, and coma have been the constant features of this stage. In over half the children fits ushered in the coma; in the remainder coma developed more gradually. Fits occurred at some time in the great majority of children. Together with these features of high fever, coma and convulsions, nuchal rigidity, a moderate polymorph leucocytosis in the peripheral blood, and an almost constant pleocytosis without other changes in the C.S.F. made up the clinical picture. Focal neurological signs were found in half the cases. In the first few days of the encephalitic stage a not uncommon finding was a monoparesis, or hemiparesis, with spontaneous movements and movements in response to painful stimulation diminished, and hypotonia, but tendon reflexes either preserved, or returning within a few days. Later in the course of the encephalitis the affected limbs tended to become spastic. Apart from a facial palsy as part of a hemiparesis, cranial nerve lesions were not seen. In several of the cases which ran a long course, extra-pyramidal signs were conspicuous; decerebrate rigidity developed

in one fatal case, and very striking choreo-athetosis persisted in others for weeks on end. These children unless heavily sedated, would continue to thrash and writhe in a most distressing way.

The course of this acute encephalitic stage was extremely variable and quite unpredictable. Two children who appeared desperately ill on admission made complete recoveries within 10 days; others died within a short period. In the most distinctive cases, high fever, coma, and varying C.N.S. signs persisted for periods of 1, 2 or even 3 months, death or the beginning of a gradual and incomplete recovery marking the end of the active phase.

Recovery Stage:

Experience has taught that until the temperature settles completely, and the C.S.F. becomes normal, there is the possibility of a recrudescence of the active encephalitic process. Once the active phase has passed recovery begins. The only 2 children to make a complete recovery, regained consciousness within a week, and were completely normal within a fortnight. 7 of the 18 cases died. Of the 9 survivors of the first 14 cases, 5 left hospital with quite gross motor defects, and 7 with gross intellectual impairment. During the 3 years of follow-up, the degree of improvement has been astonishing in these children. Really gross spastic paraparesis has resolved to the point of a nearly normal gait. Spastic mono-paresis of upper limbs have resolved completely. The extent of intellectual recovery has also been impressive, but some defect looks like being permanent in all these children. 2 children had recurrent epilepsy following their illness. Most of the children have constituted behaviour problems of more or less severity.

Treatment:

7 deaths, 9 permanent defectives, and only 2 complete recoveries.

marks Japanese B encephalitis as a pretty sinister disease. Unfortunately no known antibiotic significantly alters its course. Symptomatic treatment is important and includes sedation for control of convulsions, nursing in a semi-prone head-down position with frequent changes from one side to the other, attention to pressure-points, and maintenance of nutrition by tube-feeding. For reasons discussed under differential diagnosis, we believe it is important to give all cases a broad-spectrum antibiotic of the start, after first taking blood for culture.

Differential Diagnosis

The normal or high C. S. F. sugar marks these cases at once as examples of aseptic meningitis, and excludes bacterial meningitis. It does not, however, exclude a fulminating bacterial infection with septicaemia or a meningeal reaction, without frank meningitis. We have seen typhoid, pneumonia, and infection with Salmonella enteritidis, all

proved by blood culture, present clinical profiles quite indistinguishable from viral encephalitis. A prodromal stage of fever and headache, followed by convulsions and loss of consciousness, polymorph leucocytosis and pleocytosis of more than 100 cells per cu. mm. in the C. S. F. have all been present. A correct diagnosis will be made in these cases only by means of blood culture and it is while waiting for this result that it has seemed to us imperative to start treatment with a broad spectrum antibiotic.

The differentiation on clinical grounds of Japanese B encephalitis from other forms of presumed viral encephalitis is virtually impossible. Tables III, IV, V and VI show that in our limited experience there were certain points of possible distinction. Japanese B encephalitis tended to have a slightly longer prodromal stage, a considerably longer active encephalitic stage, and a more intense and prolonged meningeal reaction. The mortality was also higher.

TABLE III
ENCEPHALITIS

	Japanese B Type	Miscellaneous
Total	18	11
Acute	17	9
Prodromal Phase in days Median	1.5	0.5
Range	0—14 days	0—1.5 days
F E V E R		
Range of Maxima	101—108	99.8—106.8
Duration > 8 days	15/17	2/9

TABLE IV

	JBE Type	Miscellaneous
Convulsions	17/17	8/9
Coma duration in days Median	15	9.5
Neck rigidity	15/17	7/9
Focal C. N. S. Signs.	11/17	6/9
(a) Paralysis	10	4
(b) Involuntary movements	5	2

TABLE V

	JBE Type	Miscellaneous
WBC Maximum (Range)	10,900—34,300 (1 case, 2,100)	12,200—25,700 (1 case, 2,200)
C Cells > 10	15/17	6/9
S > 50	14/17	3/9
F Sugar (Min.)	60	50 (1 case 22 mg.%)

TABLE VI

	JBE Type	Miscellaneous
Died	7/18 (39 percent)	2/11 (18 percent)
R E S I D U A		
Nil	3/18	3/11
Mental Defect	7/18 (one with paralysis)	4/11 (one with paralysis)
Pareses	3/18	3/11

Descriptions of the North Indian encephalitis suggest this is more like our presumptive cases of viral encephalitis than the Japanese B group. However, the fairly constant occurrence of diarrhoea and the occasional finding of low blood and C. S. F. sugars in the North Indian cases have not been observed at Vellore. I do not think this rules out the possibility that sporadic infections with the N. Indian "virus" are seen in the South, but this must remain pure speculation until a virus has been isolated and specific serological tests become available. In any case it is probable that these presumed viral encephalitides represent a miscellaneous group of infections.

Pathology :

Our pathological material includes 5 brains from autopsies, and two specimens of cerebral tissue obtained by biopsy-needle through burr-holes made post-mortem. These have shown the features characteristic of the arbor meningo-encephalitides, moderate inflammatory cell infiltration of the lepto-meninges, foci of glial infiltration in the cerebral parenchyma, and small round cell infiltration of peri-vascular spaces.

Serological Diagnosis.

Infection with an arbor virus stimulates formation of at least three distinct types of antibody, haemagglutination inhibiting (HI) neutralising (N) and complement fixing (CF). The chief interest and importance of the HI antibodies lies in the fact that the relevant antigens are common to many different arbor viruses; it has proved possible to classify all the known arbor viruses into 3 groups, A, B

and C on the basis of a common group HI antigen. Group B includes most of the arbor viruses of interest to us in India, namely Japanese B, West Nile, Dengue, and Kyasanur Forest, as well as many viruses important in other parts of the world e.g. Yellow Fever, St. Louis, Murray Valley, and Russian Spring Summer. N antibodies are demonstrated by the mouse-protection test. Lethal doses of virus are mixed with serial dilutions of the serum under test and injected intra-cerebrally into mice. Survival of the mice, checked against controls, indicates the presence in the serum of N antibody and the dilution up to which protection is conferred gives the antibody titre. The titre of N antibody rises rapidly during an infection and tends to remain high for years afterwards. CF antibodies, on the other hand, rise quickly but fall away quickly sometimes disappearing within a year of the provoking infection. Although N and CF antigens are more type-specific than HI antigens there is still a good deal of overlap to form as it were, family subdivisions within the B group. For instance there is a very close immunological relationship between Japanese B, Murray Valley, St. Louis and West Nile viruses, and between Russian Spring Summer, Omsk Haemorrhagic Fever, and Kyasanur Forest Disease viruses. Infection with one of these viruses will provoke significant antibody formation against the closely related viruses. In the serological diagnosis of an arbor virus infection it is therefore essential to titrate each antibody through to an end-point against each of the closely related viruses. The titre of antibody against the agent actually responsible for the infection will be higher, but only slightly higher than the titre against the related viruses. Tables VII and VIII illustrate some of these points in two on the 1955 cases.

TABLE VII
SERIAL ANTIBODY STUDIES
C. M. C. H. I. P. No. 25289

Days PI	Compt. Fixation			Haemggn. Inhibn.			NT
	JBE	MVE	E101	JBE	MVE	E101	JBE
31	64	4	8	>7	2	1	>4.2
51	64	4	8	>7	1	1	3.2
57	128	ac	8	5	1	1	
65	32	4	<4	5	1	1	
81	16	4	<4	5	0	0	
129	12	<4	<4	0	0	0	
207	12	<4	<4	1	0	0	
357	4	<4	<4	0	0	0	

TABLE VIII
SERIAL ANTIBODY STUDIES
C. M. C. H. I. P. No. 25954

Days PI	Compt. Fixation			Haemggn. Inhibn.			NT
	JBE	MVE	E101	JBE	MVE	E101	JBE
9				>7	>7	>7	3.6
10	256	32	8	>7	>7	>7	3.9
30	256	128	64	>7	>7	>7	4.4
36	256	128	64	>7	>7	>7	4.2
44	32	16	<4	>7	>7	>7	3.4
50				NS			3.1
64	32	12	<4	7	7	7	3.9
71	12	12	<4	6	6	5	4.0
81	12	12	<4	6	6	5	3.6
88	12	4	<4	0	0		3.1
95	12	4	<4	6	6	5	3.4
102	12	4	<4	6	6	5	3.4
119	12	12	<4	5	5	5	3.9
298	4	4	<4	4	4	4	
424	<4	<4	<4				

In these particular cases, the high C.F. antibody titres indicated a recent J.B.E. infection and in conjunction with an illness clinically typical of an arthropod-borne meningo-encephalitis furnished good presumptive evidence for a diagnosis of Japanese B encephalitis. Proof of the diagnosis depends on the demonstration of a significant rise in antibody titre during the course of the disease. This was achieved in only a few cases, as in most instances by the time the first specimen of blood was taken, the antibody titre was already high. The rise of antibodies is very rapid, and the first blood really needs to be collected within 4 days of the onset of illness for there to be a good chance of demonstrating a diagnostic rise.

Isolation of Virus

Complete proof of the virus aetiology of an illness is obtained when the virus itself is isolated, as well as the rise of specific antibodies demonstrated. For a long time, our efforts to isolate a virus from cases were unsuccessful. Samples of serum and C.S.F. from all acute cases were inoculated intracerebrally in infant mice, but the mice remained obstinately healthy and no agent was recovered. In encephalitis, the viraemic phase is probably quite short and, as in polio-myelitis, precedes the stage of C.N.S. invasion. As our cases were all frankly encephalitic, it is not surprising we failed to recover virus from the blood. 4 of the 1955 cases came to autopsy and extracts of brain tissue were inoculated but without success. These cases had all been ill a month or longer and it is only in early deaths that there is much chance of isolating a virus. This year we have had three children who died on the fifth, fifth and Ninth day of illness. Although permission for autopsy was not obtained, we were allowed to make burr-holes in the skull and to obtain pieces of brain by means of biopsy-needles. In a further case, a boy aged 13 years under the care

of Dr. Jacob Chandy, cerebral tissue was obtained through a burr-hole on the third day of illness, and from this a virus was isolated. This child went on to make an apparently complete recovery. Some of this material was inoculated intracerebrally in infant mice and this time the mice came down. Dr. Charles Anderson informs me that final identification of the agents obtained is not yet complete, but the first is almost certainly a Japanese B encephalitis virus.

Epidemiology :

In those parts of the world where the natural history of arbor viruses has been worked out in greater detail than has so far been possible in India, there is a certain time of the year when the virus is widespread and numerous among birds or animals and the vector arthropods. For instance, in Japan in certain very large heronries during the breeding season, at one time it may be very difficult to find virus, but within a period of weeks, almost all the fledglings are found to be circulating JBE virus in their blood, and the virus can be recovered with relative ease from the vector mosquito, *C. tritaeniorhynchus*. The question of where the virus lurks during the off-season has nowhere been completely answered but it is clear that in each region where these arbor viruses cause disease, one or more species of animal or bird act as a reservoir of infection which is disseminated by the arthropods feeding on them. Human cases occur only when the virus is teeming in the animal world around them. These cases are like the visible part of an ice-berg, the great viral activity in animals corresponding to the submerged nine tenths.

The epidemiological problems posed by the recognition of Japanese B encephalitis in S. India have been tackled by the V. R. C. in a number of ways. All the homes of cases were visited. These

appeared to be ecologically homogeneous. They were all isolated farms, or houses in small villages, with paddy growing nearby. Mosquitoes in the houses or adjoining cattle byres were numerous. Blood samples were taken from other members of the family or household, and from neighbours. Most of them were positive for JBE antibodies. (See table IX). This was in line with the general observation that for every case of overt meningo-encephalitis, there is a very large number of inapparent infections. The predominant mosquito proved to be "*C. Vishnui*". In that this "species" is closely related to *C. tritaeniorhynchus*, the proved J. B. E. vector in Japan, it seemed likely this was the vector in India. A system of mosquito-collections was instituted, and has been maintained, partly by hand, partly by means of baited traps. The mosquitoes were pooled according to species and location where caught, and the pool ground up, extracted and the extract injected into mice. A considerable number of viruses have been obtained from these mosquitoes; both J.B.E. and West Nile virus have been isolated several times. These viruses were recovered most often from "*C. vishnui*," thus establishing this "species" as a vector. However, careful study by the V.R.C. entomologist, Dr. Harold Trapido, revealed that what had originally been thought to be a single species of mosquito was in fact made up of 4 species, closely related but possibly with distinct habits. More investigation is still needed to establish which species of this "*C. vishnui*" complex is the true vector.

Limited studies of bird and animal sera by the V.R.C. zoologist have so far failed to indicate the probable reservoir from which the mosquitoes become infected.

In an extensive survey of S. India in 1956, some 5000 sera were collected. These have been tested for C.F. antibody

against J.B.E. virus. Positive results indicated recent infection, probably within the previous year. By this means, some idea of the extent of Japanese B virus activity in 1955 was obtained. A considerable area in the northern part of Madras State extending from the coast as far inland as Bangalore, and involving adjacent parts of Andhra, and a smaller area further south in the vicinity of the Cauvery River were defined. However, it is probable that the total area involved is very much larger than this, as will be revealed when the sera obtained on this first survey are run for neutralising antibodies, and when more extensive surveys can be made.

TABLE IX

C.M.C.H. I.P. No. 25289

Onset of illness 30—9—'55

Antibody studies in Contacts 1—11—'55

Contact	Age / Sex	JBE.NT.	Sero-logy CF.
Patient	5 / M	†	64
Father	56 / M	†	32
Mother	35 / F	†	4
Brother	23 / M	†	16
Brother	14 / M	—	4
Sister	18 / F	—	4
Sister	8 / F	†	12
Grandmother	65 / F	†	4
Uncle	65 / M	†	32
Aunt	55 / F	†	12
Aunt	35 / F	†	12
Cousin	15 / F	†	4
Cousin	10 / M	†	12
Cousin	12 / F	†	4

N.T : Neutralization Test.

C.F : Complement fixation : results given as reciprocal of dilution giving 75% fixation of 2 units of antigen.

Conclusion

Japanese B encephalitis is an interesting disease and its epidemiology poses fascinating problems, whose solution will take many years of sustained, imaginative and expensive work. And yet Japanese B encephalitis is clearly no more than quite a small fraction of the total encephalitis problem of the region. The tools for the investigation of these non-arbovirus encephalitides are becoming more widely available. More clinicians are being trained. More laboratory virologists are being trained. The one member of the team required for the investigation of these and similar problems who is not being trained in S. E. Asia in numbers equal to the task, is the field epidemiologist. We can certainly hope to see progress in the next decade in the understanding of these distressing diseases. That progress would be very much more rapid if there were more young medical graduates prepared to undergo training in field epidemiology. They need to be able, imaginative and prepared for hard work, hot and uncomfortable work, but men of such quality have a great contribution to make in the solution of some of their country's medical problems.

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ASSOCIATION NEWS

ANAMALLAI BRANCH

The meeting of the Association was held on 27th June 59 at Dr. K. John's residence, when 3 films "Defeat Tuberculosis" "His fighting chance" and "The Undefeated", lent by the British Information Service, were shown.

* * *

A monthly meeting of the Association was held on 25th July, at Dr. Perumal's residence. Election for one President & 3 Vice presidents for the Centre was held. After that, two cases of interest, one "Rheumatic Carditis", and another, "Heavy infection of Round Worms" were demonstrated by Dr. M. P. Gundappa.

* * *

The Clinical meeting of the Association was held on 22-8-59, at Dr. Gundappa's residence, when Dr. Jayachandran gave a talk on Cholera. Election for one President and 2 Vice Presidents for the State Branch of the I. M. A. was held.

* * *

A monthly meeting of the Association was held on 26-9-59 at Dr. Narayanan's residence, when Dr. Narayanan demonstrated a few X-ray films of interest.

* * *

The clinical meeting of Association was held on 24-10-59 at Dr. Janardhanan's place, when Dr. John gave a talk on "Convulsions in children."

CHINGLEPUT BRANCH

Proceedings of the general body meeting held on Saturday 24th October 1959 at the Venugopal Club, Chingleput. Dr. K. R. Ganesa Mudaliar, president of the association presiding.

2. The following office bearers were elected unanimously for the year 1959-60.

- (a) President : Dr. A. Rajagopalan.
- (b) Vice-president : Dr. P. K. Varghese
- (c) Secretary & Treasurer : Dr. A. Sundararaja Rao

(d) Members of the Executive Committee :—

1. Dr. B. K. Sheno
2. Dr. A. N. Jagadesan
3. Dr. K. Vettal Doss Pai
4. Dr. T. K. Thayumanava Mudaliar

(e) Election of representative to the central council :
Dr. P. K. Varghese

(f) Election of representative to the provincial council, Madras :
1. Secretary
2. Dr. P. S. Ananthanarayanan
3. Dr. P. K. Varghese

Dr. Ratnavelu Subramaniam, M.R.C.P., (Lond.) dealt exhaustively on "Cirrhosis Liver".

* * *

The Ordinary Meeting of the Association was held on Saturday the 28th November 1959 in the Chingleput Venugopal Club, Chingleput. A paper on "Tetanus" was read.

MADURAI BRANCH

The Monthly meeting of the Association was held on 24th October 1959, under the Presidentship of Dr. K. Gopal, M.B., B.S. T. D. D., References were made by a few members about the late Dr. A. Narayana Menon, M.B., B. S., who was one of the founder members of this Association.

The Secretary conducted the election of Office-bearers for the Reception Committee and other sub-committees to be formed for the coming XIV Madras State Medical Conference to be held at Madurai in January 1960.

Dr. S.A. Kabir, M.B., B.S., D. A. (Lond.) Chief Anaesthetist, Government Erskine Hospital, and Lecturer in Anaesthesia, Madurai Medical College, Madurai, gave an interesting lecture on "MODERN TRENDS IN ANAESTHESIA".

* * * *

The monthly meeting of the Association was held on 21st November 1959, under the presidentship of Dr. K. Gopal MBBS, TDD, Madurai. Dr. W. J. C. Rebello, MBBS, DGO, MRCOG, Lecturer in Obstetrics and Gynaecology, Madurai Medical College, and Obstetrician and Gynaecologist, Erskine Hospital, Madurai gave an interesting lecture on "DIRECT INTERNAL PELVIMETRY".

MADRAS CITY BRANCH

The above association had its monthly clinical meeting on 26—9—'59, with the president Dr. G. Sriramulu in the chair.

At the outset a condolence resolution was passed touching the death of Dr. R. K. K. Thampan.

This was followed by a lecture on "The management of Essential Hypertension" by Dr. R. Subramaniam, MD., MRCP.

After the dinner, the new members, in whose honour the dinner was arranged, were welcomed by the president, Dr. Sriramulu. He said that as a result of an intensive membership campaign the association was able to enrol about 150 members. He earnestly requested those present to help in enrolling more members, and thereby strengthen the association. Dr. D. V. Venkappa later addressed the new members.

Later Dr. K. S. Sanjivi, MD., spoke on "Voluntary Health Services".

SALEM BRANCH

A meeting of the Association was held at the Centenary Hall of the Govt. Head Quarters Hospital, Salem, on 24th October 1959. Dr. S. Sundaram MBBS., FICS., presided. Dr. S. R. Rajaram paid tribute to Dr. Sundaram on his securing the Fellowship. He welcomed Dr. Azceezulla, the new District Medical Officer.

Dr. S. Srikantan, M.S. gave an interesting talk on 'Chronic Ulcers of the Leg'.

SOUTH ARCOT BRANCH

The General Body of the above association was held on the 24th October 1959 at the Pondicherry Medical College premises. Dr. V. Krishnamoorthy, the President, was in the Chair. The Principal Dr. Vengsarkar gave a concise talk on the future of the college. Dr. Lucien Brumpt, M.D., Aggregate Professor of the Faculty of Medicine at Paris and Visiting Professor of Parasitology, Medical College, Pondicherry, addressed the members on (1) Bedside Diagnosis of Typhoid Fever and (2) Eosinophile Parasitaire.

TANJORE BRANCH

The monthly meeting of the Association was held on the 30th August 1959 at the Medical School Buildings. Dr. T. M. Pillay, the Vice President of the Association was in the Chair. Elections of two Vice Presidents to the Madras State Branch of the I. M. A. were held out of the list of nominations sent by the State Branch. Dr. N. R. Subramanyam; M.B., B.S. Hony. Physician, H. Q. Hospital, Tanjore and President of the Association spoke on 'The Management of Heart Failure'.

* * *

The Twenty Third Annual Day of the Association was celebrated on the 18th October '59. Dr. N. R. Subramanyam, the President of the Association was in

TIRUCHY BRANCH

The 30th Annual meeting of the Association was held on Sunday the 25th October 1959. Dr. R. Sambasivan, the President presided. The following office-bearers were elected for the year 1959-1960.

President :

Dr. G. Viswanathan

Vice-President :

Dr. P. V. Sundaram

Secretary :

Dr. V. K. Ranganathan

Treasurer :

Dr. M. V. A. Iswaran

Executive Committee :

Dr. V. Ananthagiri
Dr. A. Dharmarajan
Dr. M. Balammal
Dr. P. Palaniyandi
Dr. Abraham Paul
Dr. S. Venkatesan
Dr. T. V. Sundaram

State Council :

Dr. V. Ananthagiri
Dr. R. Sambasivan
Dr. N. C. Subramanyam
Dr. S. Venkatesan
Dr. R. Kalamegham
Dr. T. S. Balasubramanyam

Central Council :

Dr. T. V. Srinivasan
Dr. T. S. Balasubramanyam

The following lecturers addressed the gathering on the following subjects respectively.

the Chair. Dr. P. M. Palani, M. D. Hony. Physician, Government General Hospital, Madras addressed the gathering on 'Medical Emergencies and other treatment'. After this Dr. H. T. Vira Reddy M.B., F.R.C.S., Hony. Surgeon, Govt. Stanley Hospital, Madras spoke on 'Abdominal Emergencies in Surgical Practice' and exhibited some slides.

The business meeting was held when the following office bearers were elected :

President :

Dr. N. R. Subramanyam.

Vice President :

Dr. T. M. Pillay

Hony. Secretary & Treasurer :

Dr. S. Narayanan.

Members of the Committee :

Dr. (Miss) D. Andal.
Dr. R. Krishnasamy.
Dr. P. Narayana Rao.
Dr. K. Rama Rao.
Dr. T. R. Ramachandran.
Dr. L. T. Appadurai.

Members of the State Council :

Dr. T. M. Pillay.
Dr. V. C. Gupta.
Dr. T. R. Ramachandran.
Dr. P. Narayana Rao.

Representative to the Central Council :

Dr. T. M. Pillay.

A symposium on 'Some Aspects of Chest diseases - Medical and Surgical' was held at which Dr. AL. Annamalai M.B.C.P., D.T.M. & H. Chief of the Chest Clinics, Govt. General Hospital, Madras explained the medical aspects and Dr. C. S. Sadasivan, M.S., Thoracic Surgeon, Madras, General Hospital explained the Surgical Aspects of Chest diseases with the aid of about 150 slides projected on the screen.

Cost of Treatment in the Dermato Venereologic Practice: Dr. P. N. Rangiah, M.D., Director, Institute of Venereology, Govt. General Hospital and Madras Medical College, Madras.

Surgical Emergencies in Paediatric Practice: Dr. C. K. Padmanabha Menon, M.S., F.R.C.S., Principal and Professor of Surgery, Madurai Medical College and Surgeon, Govt. Erskine Hospital, Madurai.

The Role of a General Surgeon in the Field of Neuro Surgery: Dr. K. C. Nambiar, F.R.C.S., (Eng. and Edin.),

Professor, Principles and Practice of Surgery, Govt. Stanley Medical College, Madras.

Ano Rectal Conditions: Dr. Frank I. Tovey, F.R.C.S., Holdsworth Memorial Hospital, Mysore.

The Medical Treatment of Cancer: Dr. S. Kalyanaraman, M.S., Asst. Professor of Surgery, Govt. Stanley Medical College, Madras.

Beware of Glaucoma: Dr. T. V. Ranganathan, M.B., B.S., L.O., Z.O., D.O., (Lond.) Hony. Asst. Medical Officer, Govt. Hd. Qrs. Hospital, Tiruchi.

ANNOUNCEMENT

The 14th Madras State Medical Conference will be held at MADURAI on the 23rd and 24th of January 1960. The Madurai Branch of I. M. A. has formed itself into a Reception Committee with Dr. P. Vadamalayan, MBBS., as the Chairman, Drs. C. K. P. Menon, MS., FRCS., and K. Gopal, MBBS., TDD., as Vice-Chairmen, Dr. T. Thirugnanam, MBBS., as the Hony. General Secretary and Dr. A. Arunachalam, MBBS., as the Hony. Treasurer. Necessary Sub-Committees have also been formed with influential personnel.

There will be the Scientific Sessions with interesting programme. A Souvenir will also be brought out on the occasion.

All the local branches of I. M. A. and all the members of I. M. A. are requested to kindly extend their hearty co-operation with the organising branch and make the Conference a grand success.

INTERNSHIP/RESIDENCY TRAINING IN UNITED STATES HOSPITALS

Members of the I. M. A. Madras State Branch are informed that according to the decision of the Educational Council for Foreign Medical Graduates (ECFMG), 1710, Orrington Avenue, Evanston, Illinois, USA, the foreign medical graduates who are desirous of taking up appointments as Interns, Assistant Residents and Residents in American Hospitals after January 1960 must have passed the "American Medical Qualification Examination." The next examination is scheduled to be held in India (at four centres, namely, Delhi, Bombay, Calcutta and Madras) on March 16, 1960; and the last date for submitting applications to the ECFMG is December 16, 1959. All applicants have to be approved by the Educational Council for Foreign Medical Graduates before they are allowed to sit for the examination.

Members of I. M. A. Madras State Branch who are interested in taking this examination on March 16, 1960, should obtain the application forms etc., immediately from the Hony. General Secretary, I. M. A. Hanging Bridge, Darya Ganj, Delhi-7, and return the same duly filled in along with the necessary photographs and photostat copies of certificates, DIRECT BY AIR MAIL to Dr. Ruth E. Burrows, Assistant Director, Educational Council for Foreign Medical Graduates, Orrington Hotel, 1710, Orrington Avenue, Evanston, Illinois, USA, to reach her before DECEMBER 16, 1959.

Sub: British Medical Journal - Subscription at concession rates for members of the I. M. A. increase in the rate of subscription - intimated.

The members of the I. M. A., Madras State Branch, are hereby informed that since the announcement in The Madras Clinical Journal of October 1959 by which the availability of the B. M. J. at concession rate of Rs. 28/50 nP. has been published for the information of members, a communication (No. 4/59-60 dated 21-10-1959) has been received from the Central I. M. A. Delhi informing all its branches that the British Medical Association by their letter of October 14, 1959, had intimated that the concession rate of subscription has been raised by 10sh. 6d., i.e. from £ 2-2-0 to £ 2-12-6 and that the revised rates will come into force with immediate effect. Therefore, the concession rate, in terms of Rupees, comes to Rs. 36-50 nP., as against Rs. 28-50 hitherto prevailing. Intending subscribers will, therefore, have to send Rs. 36-50 if by money order, or Rs. 37-00 if by cheque (inclusive of bank charges).

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C. ARUMUGAM, M.B., B.S.,
Hony. State Secretary,
Madras State Branch, I. M. A.

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EDITORIAL NOTICE

All contributions for publication and correspondence should be sent to the Editor, Madras Clinical Journal, Journal of the Madras State Branch of the Indian Medical Association, The Nursing Home, Cantt., Tiruchirapalli-1.

Manuscripts for articles, case notes, etc., must be type-written, double-spaced, on one side only of foolscap or quarto paper. Only original type-scripts (not carbon copies) will be considered for publication. Graphs, charts, diagrams or pen drawing must be drawn in Indian ink on white drawing paper. Blue or coloured inks cannot be reproduced.

In the selection of paper and in regard to priority of publication, the opinion of the Editor will be final. The Editor shall have the right to edit, condense, alter, re-arrange or re-write approved articles, case notes and other communications before publication, without reference to the authors concerned. The editor will not be responsible for the views and statements of authors of articles and other communications. Manuscripts not accepted for publication will not be returned to the authors unless the necessary postage is supplied by the author.

References—Reference to authors in the body of the papers should contain the name of the author, with the year of publication mentioned within brackets. For the list of references at the end of the communications, the system followed by the Cumulative Index Medicus, should be adopted. This requires the following in the order mentioned : surname and initials of author name of periodical with its volume number, the page number and the year of publication. The volume number should be underlined as follows :—21 : 279, 1952. The references should be arranged according to alphabetical order of the surname of the authors. In case of reference from books, name of author, title of book, edition, name of the publisher, and place and year of publication should be furnished.

Reprints—Ordinarily, 25 copies of articles and case notes (not correspondence, etc. published shall be supplied, free of charge, to the authors. Additional reprints may be obtained at cost, if a request for the same be made when sending the article.

Reviews—As the space in this journal allotted for reviewing books and periodicals is limited, the publishers, authors and editors are informed that these can be reviewed according to their merits and space at disposal. Books and periodicals, for review, should be sent in duplicate, to the Editor.