

CONTENTS

1. CLINICAL MEDICINE & THERAPEUTICS by Dr. T. V. Venkatesan	79
2. CORONARY INSUFFICIENCY by Dr. K. Venkata Rao	86
3. SURGICAL SHOCK—MODERN TRENDS IN TREATMENT by Dr. S. Kalyanaraman	93
4. ASSOCIATION NEWS	95
5. ANNOUNCEMENT	97
6. OBITUARY	

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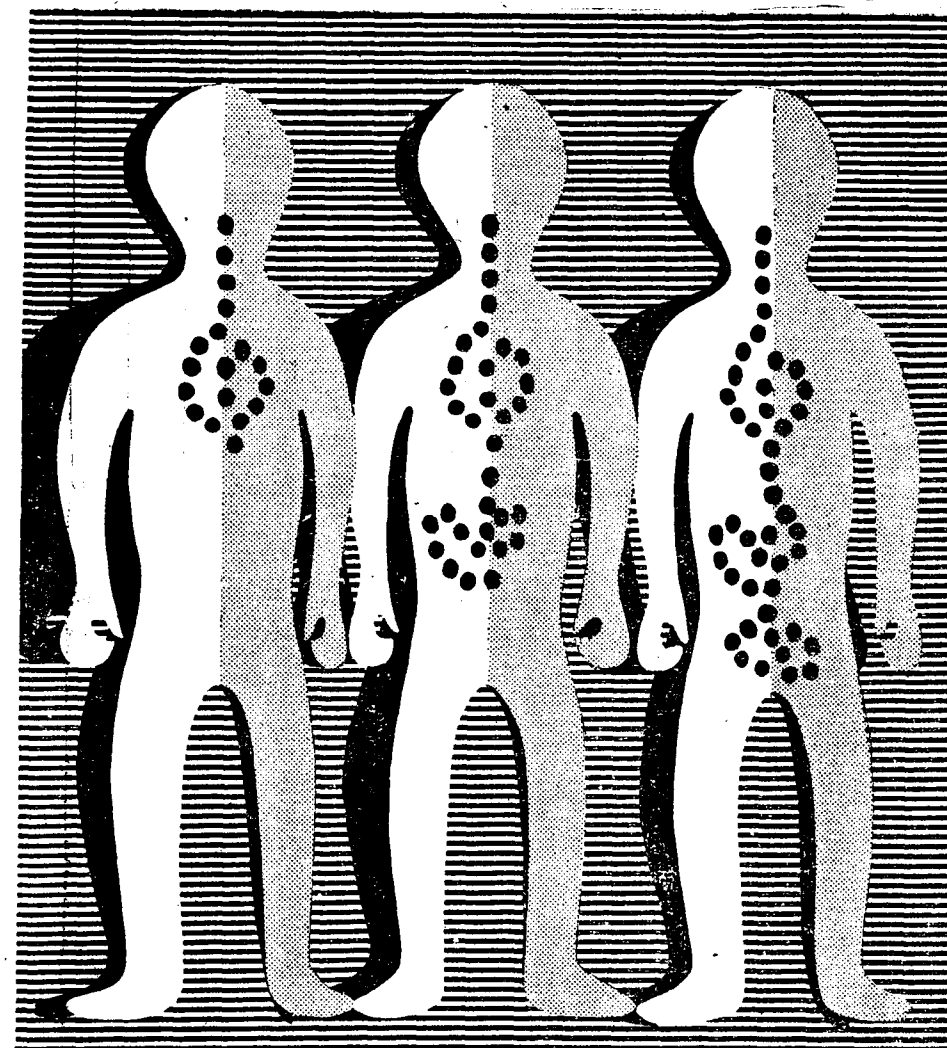
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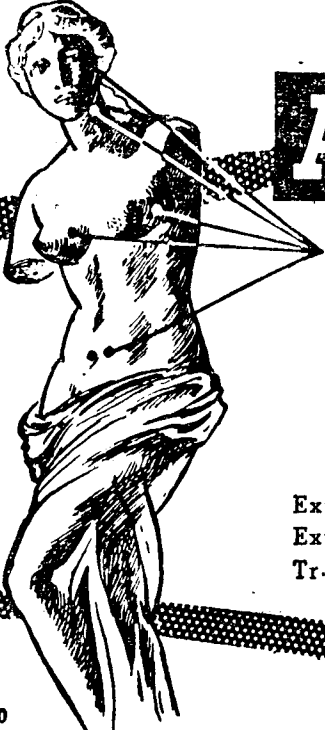
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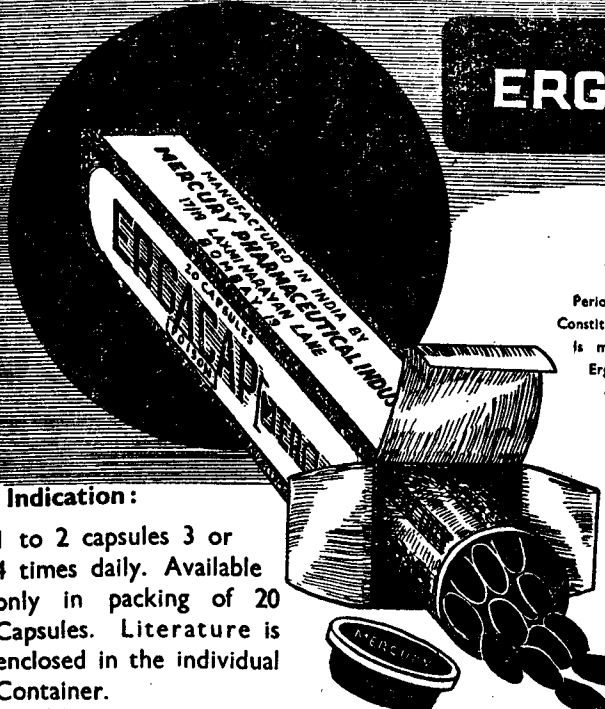
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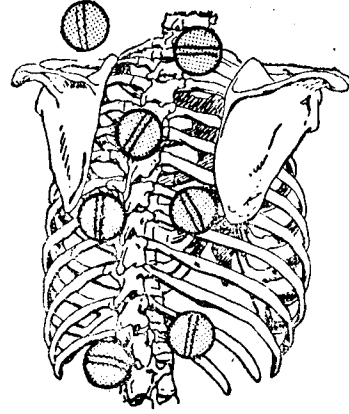
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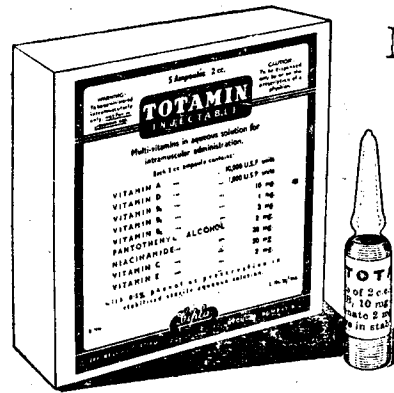
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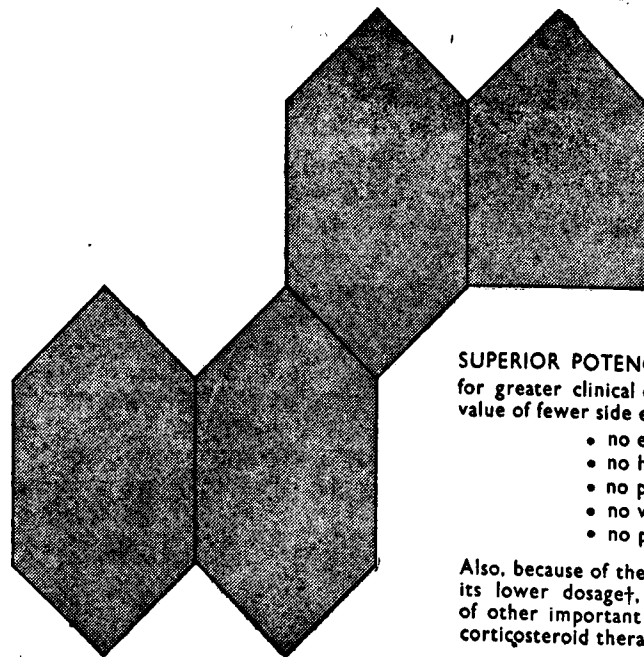
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THE MADRAS CLINICAL JOURNAL

JOURNAL OF THE MADRAS STATE BRANCH OF THE
INDIAN MEDICAL ASSOCIATION

(With which is incorporated the "Miscellany")

Vol. XXVI]

OCTOBER 1959

[No. 4

CLINICAL MEDICINE AND THERAPEUTICS

by

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

THIS subject is chosen for the reason that every general medical practitioner must be well off in clinical methods and Therapeutics. The first requisite is a careful history taking. Interrogation of the patient gives an idea of (1) Cardinal symptom (2) Facts concerning with present illness (3) Previous history (4) Personal history and (5) Family history. It is possible for every Doctor to become a sound clinician, if he is able to get into the regular habit of case taking. Many a time it is possible to arrive at a presumptive diagnosis by a careful history taking. The history should reveal the mode of onset, the duration of the symptom, the severity of the symptom and any associated condition. The history regarding previous health of syphilis, rheumatism, etc., is helpful. The family history gives a clue regarding the causal relationship with the patient's malady.

The next important step is the special interrogation. The history taking gives an idea about the probable system involved and with this in view, a special

interrogation is essential. for example, if a patient comes with skin condition, enquiry should be made regarding his diet, his occupation and any history of having taken any drug and if the eruptions are itchy and they appear and disappear. Supposing a patient gives a history of cough, expectoration and dyspnoea, it is better to find out his occupation, viz., does it expose him to the inhalation of irritative fumes or particles? Has he ever had any enlarged glands in the neck? Does he sweat in the night? Is he getting thinner?

Regarding cough, the following details are to be enquired viz., the character and frequency, when is it worst? Does it pain him or not? Does he ever vomit with it? If he has expectoration, its amount and general characters, if blood-tinged or not, if the blood is bright and frothy or dark in colour. If the patient has dyspnoea, when is it felt? Has he to sit up in bed? Can he sleep lying down? Is it paroxysmal? Similarly, for every leading symptom, a careful question is essential. The most

Lecture delivered at the Tiruchi Branch of I. M. A. on 24-1-59.

important duty of a doctor is to make a systematic physical examination.

General condition and appearance.

An observant physician can obtain important clues to diagnosis by the physiognomy. In acute diseases, the face assumes an anxious expression and is characteristic in pericarditis, peritonitis severe pneumonia and also during attacks of angina pectoris. Again in acute Lobar Pneumonia, the appearance is very distinctive. The flushed face, hot dry skin, the widely dilating nostrils, the eruption of herpes near the mouth and the profound disturbances of the pulse respiration ratio (2:1 instead of 4:1 which is the normal) form a picture which greatly gives the recognition of the disease. The Facies Hippocratica - a facies or appearance of which the description has been handed down from Hippocrates is the forerunner of death from exhaustion, such, as for instance, as the final stage of cholera and wasting disorders. The temples are hollow, the eyes sunken, the eyelids slightly parted, the eyes glazed and the lower jaw drooping. The Risus Sardonius is a fixed grin and is met typically in tetanus. The corners of the mouth, which twitch at intervals, are drawn upwards as in laughter and the features assume a fixed sarcastic expression. In chronic diseases, the following features can be noted. A tuberculous patient can be described as a delicate patient. When the disease is advanced he is pale and emaciated.

In chronic bronchitis with dilated right heart, the florid 'healthy' looking cheeks, the distended jugulars in a person over forty are very typical. In Bell's Paralysis, the face is distorted. The smooth expressionless face is seen in Parkinsonism. Swelling of the face occurs in Renal disease. Bilateral swelling on the face is seen in myxoedema, the face is pale, the skin is dry, the supra-clavicular pads and a slow monotonous speech like an old gramophone are characteristic. The complexion in the colour of the face needs careful inspection. There is pallor in anaemia, sallowness in aortic valvular disease, puffy appearance in nephritis, the yellow appearance in jaundice, the pigmentation in Addison's disease and arsenical poisoning are evident. The skin has got a greasy appearance on the face in Acne Vulgaris and seborrhoea.

Decubitus

If a patient is sitting up in bed propped up with pillows, it is the characteristic of cardiac failure. The patient lies on one side in pleural effusion, the position being on the affected side. The dorsal decubitus that is lying on the back, is seen in typhoid fever. Arching of the back or Opisthotonos occurs in tetanus, strychnine poisoning and meningitis. Carphology is characteristic of typhoid state. The nose has sunken bridge in congenital syphilis. The lips are pale in anaemia, blue in congestive heart failure or congenital heart disease. The hands of the patient merit careful observation. Clubbing of the fingers occurs in respiratory and cardiovascular diseases. Koilonychias occurs in hypochromic anaemia. Heberden's knobs occur as nodules in Arthritis deformans. Tremor in the hands occur in Parkinsonism, Grave's disease and alcoholism. Claw-like hands occur in ulnar paralysis and motorneurone disease. "The short stunted stature, the sambestial aspect, the blubber lips, the retrosse nose sunken at the root, the wide open mouth, the lolling tongue, the small eyes half closed with swollen lids, the stupid expressionless face, the squat figure the muddy dry skin" combine to make a picture of a cretin.

Examination of Neck.

Neck should be examined for
(1) enlargement of lymphatic glands.
(2) thyroid glands (3) pulsations in the

vessels both arterial and venous and (4) any bulging of the apices of the lungs during a fit of coughing.

I shall deal with the next subject viz., examination of the various systems.
(a) Cardio-vascular system-pulse. The radial pulse should be felt for rate, rhythm, volume, force (indicates systolic pressure) tension (indicates diastolic pressure), state of the arterial valve and character of pulse.

A small amplitude of aortic stenosis, water hammer pulse of Aortic incompetence, diminution of the pulse on one side in aneurysm of the arch of aorta, irregular pulse of auricular fibrillation are characteristic.

Inspection determines:—

(A) Form—

1. Of praecordial { Bulging
Flattening.
2. Of surrounding parts (especially bulging)

(B) Movements—

1. In praecordial region { Apex-beat.
Diffuse pulsation.
Pulsation at base of heart.
Local in drawing.

The ordinary situation of the nipple in an adult male of average development may be placed in the 4th intercostal space 4 inch from the midsternal line.

2. Outside praecordial region { Pulsations at root of neck.
Pulsations in thorax.
Pulsations in epigastrium.

(C) Dilated veins and venules. The principal features to be observed in apex-beat are, position, character and frequency. A heaving apex-beat is characteristic of left ventricular hypertrophy.

A slapping apex-beat means a poorly contracting left ventricle and occurs in mitral stenosis, coronary arterio stenosis and in diphtheritic myocarditis. Apex-beat should be counted and compared with the pulses and if difference occurs between the two, it is known as the pulse deficit. This occurs in auricular fibrillation and premature beats.

Pulsations outside the precardium—
(1) Supra clavicular pulsation may be arterial or venous. When the patient is recumbent, normally no distinct neck veins can be seen or at the most one third of the way between the Manubrium sterni and the angle of the mandible. In congestive failure, the neck veins are distended and pulsating. If neck veins are distended and *not pulsating*, pericardial effusion or constrictive pericarditis should be suspected. Supra clavicular permanent arterial pulsations are characteristic of aortic regurgitation.

(2) Epigastric pulsations occur due to Hypertrophied right ventricle.

Palpation

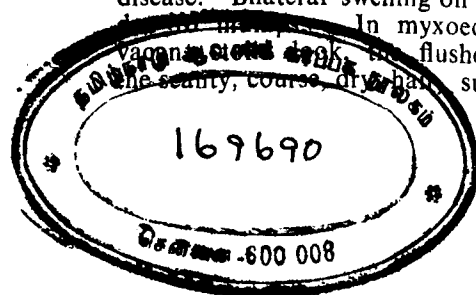
Palpation determines: (a) Form of praecordia etc., (confirms or modifies results of inspection)

(b) Movements:

- (a) Apex-beat { Position
character
- (b) Other praecordial pulsations.
- (c) Pulsations outside praecordia { Heaving
Expansile.

(c) Vibrations:

- (a) Originating within the heart and blood vessels (Thrills)
- (b) Originating exocardially (Friction)



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Thrills are to be termed properly in relation to apex beat or carotid. At the root of the neck, palpation will frequently enable one to identify a pulsating vessel and so clear up a doubtful diagnosis. When pulsation occurs in the episternal notch one should, if possible, try to push the finger below the pulsating vessel.

By so doing one may be saved the mistake of diagnosing an aortic aneurysm when the patient has merely an abnormal origin of his right subclavian.

In the epigastrium the fingers should be pressed gently but firmly upwards under the left costal margin when pulsation of the right ventricle is suspected. By this means it may be readily differentiated from pulsation of the liver.

Expansile pulsation of the liver occurs in tricuspid incompetence.

Percussion of the boundaries of the heart is most helpful in Pericardial effusion. The apex beat is found inside the area of dullness.

Auscultation

The auscultation of the heart is done in all classical area viz., mitral, pulmonary, aortic and tricuspid. The stethoscope should be moved gradually to cover the whole of the pericardium. This process is called inching. The following points are to be determined :—
(1) character of the heart sounds ;
(2) abnormal sounds as murmurs and pericardial friction rubs. The first heart sound is due to closure of the mitral and tricuspid valves splitting of the first heart sound is common in health and is best heard in the tricuspid area. The loud first sound in mitral stenosis is produced in that the high filling pressure keeps the cusps fully open until the last possible moment. The second heart sound is due to aortic and Pulmonary valve closure.

Normally both elements can be heard in the second and third intercostal spaces close to sternum. The first element is aortic and the second is pulmonary. Pathological splitting is due to delay in pulmonary valve closure, as in right ventricle branch block or pulmonary stenosis. In aortic stenosis and left ventricle branch block, the aortic element may lay behind the pulmonary element and the reverse split, so caused tends to close on inspiration. Accentuation of the second heart sound occurs in pulmonary hyper-tension. A soft or absent second heart sound is usual in Emphysema. The third heart sound is soft low-pitched and occurs during the period of rapid ventricular filling. It is best heard and more or less localised to the apex-beat and the best position is when the patient lies on the left side.

Opening snap. This is heard in all cases pure mitral stenosis and is about mid-way between second and third heart sounds. It is produced due to abrupt flapping back of the mitral cusps at the end of the period of Isometric relaxation i.e., when the rapidly falling left ventricular pressure drops below the left atrial pressure. Opening snap disappears when there is associated aortic incompetence. Due to interference between the forward movement of the aortic cusps of the mitral valve, it is best heard in expiration at the lower left sternal edge.

Murmurs : The pan-systolic murmur occurs in ventricular septal defect in the left fourth intercostal space, at the apex in the mitral incompetence, at the tricuspid areas in tricuspid incompetence. The pre-systolic murmur of the apex followed by loud first sound is characteristic of mitral stenosis. The mid systolic ejection aortic or pulmonary murmur is due to aortic or pulmonary stenosis. A systolic murmur is heard at the base or over the back in coarctation of Aorta.

Innocent murmurs : In anaemia systolic murmur is heard over the pulmonary area. Due to the reduced viscosity of the blood, they are also common in Grave's disease. As a routine, the blood pressure should be recorded in both upper and lower limbs and in both upper limbs. Blood pressure is higher in lower limbs in aortic regurgitation. There will be a difference between the blood pressure of the two upper limbs due to aortic aneurysm pressing on the sub-clavia.

Respiratory System : The inspection of the chest is very important and the regions of the chest may be described as suprasternal, superior sternal, inferior sternal, supra clavicular, infra clavicular, Mammary, inframammary, Axillary and infra-axillary, suprascapular, infra-scapular and inter-scapular. Unless this nomenclature is used we will not be able to have an idea of the site of lesion. The first step is to find out the form of the chest. Is the chest symmetrical? If symmetrical, is it normal (Ellipsoid)? If abnormal, is it barrel shaped or rachitic or flat chest or alar chest? The alar and flat chests indicate the proclivity to lung diseases. Next step is to find out any unilateral enlargement or diminution. The movement of chest must be compared on both sides and the rate of respiration has to be recorded. Palpation confirms inspection and in addition look for tenderness and any change in the vocal fremitus on both sides.

The next step is to find out the resonance by percussion. This will help in finding out stony dullness in the presence of fluid dullness in consolidation or thickened pleura. The resonance is increased in emphysema, also it is tympanitic above the level of pleural effusion (Skodaic Resonance). Percussion over cavity produces a cracked pot sound. This can be compared to striking the knee with a closed fist.

Auscultation

Respiratory system :— The normal breath sound is vesicular i.e., expiratory sound follows inspiration with a distinct pause and is audible only for a short time. Over consolidation the sound is tubular and can be imitated by saying "Kai" and over cavity it is cavernous and can be imitated by keeping the mouth as "O" shaped and breathing.

A special variety of bronchial breathing known as amphoric breathing resembles the sound produced by blowing across the mouth of a bottle. It is characteristic of a direct communication between a bronchus and either a considerable cavity with fairly smooth walls or a pneumothorax. The vocal resonance is diminished in effusion, thickened pleura and emphysema. The increased vocal resonance occurs in pneumonia over tubercular or other forms of cavity. If whispered voice could be heard clearly, it is a sign of great value in diagnosis of cavitation and consolidation.

Adventitious sound :— Dry sounds or rhonchi are produced due to obstruction of the lumen of the bronchioles due to swelling of the mucosa or presence of tough secretion. Moist sounds or rales are due to the presence of fluid secretions in the air-cells or tubes. In a cavity, the rales may be resonant. A friction sound is characteristic of dry pleurisy. A splashing sound is produced in Hydro pneumothorax. Examination of the nose and throat forms part of the examination of the respiratory system.

Alimentary system :— The mouth should be examined for the teeth. Carious teeth are an undoubted contributory cause of many forms of dyspepsia. The gums may show deep root infection, congestion and bleeding in scurvy. Pyorrhoea should be looked for. The gums are pale in anaemia and are blue in lead poisoning. Furring of the tongue

occurs in dehydration, fevers and peritonitis. Magenta tongue is characteristic of sprue. Abdomen should be examined for protuberance or retraction. Peristaltic waves are visible in abdominal wall in chronic intestinal obstruction. It is of the ladder pattern if there is a constriction at the ileocaecal valve. If the obstruction is low down, the pattern of tumidity is present. Peristaltic waves in the stomach run from left to right and in a distended transverse colon from right to left.

Attention should be paid to the surface of the abdomen for distended veins. In obstruction of the inferior vena cava, a large lateral vein can be seen running up to the mid-axillary line. In portal obstruction, the distended veins radiate from the umbilicus. The abdomen should be palpated in all regions. Palpation of normal abdomen is painless. If tenderness is elicited, the exact extent and point of maximum intensity should be noted. If a tumour is present, try to move the abdominal wall over the tumour to find if it is intra-abdominal. The mobility of a tumour is very important. Tumours connected with the liver, spleen and stomach move freely with respiration, while tumours of kidney may be slightly movable. The others do not move with respiration at all. In malignant disease of the liver, the umbilicus becomes infiltrated and can be recognised by the "mooring of the umbilicus". The next step is to find out the presence of free fluid.

An examination of the individual viscera may be made now. Rectal examination should never be forgotten. It should be made for the presence of any haemorrhoids or any new growth.

Central nervous system examination : It is possible to arrive at the correct diagnosis of the site of lesion by a methodical examination of the central nervous system.

Psychical functions : Intelligence, Attentiveness, Memory, Orientation, Emotional-Phobias, Hallucinations, Delusions, Sleep, Delirium, Coma.

Speech and Articulation : Is the patient right or left handed? Aphasia, Apraxia, Articulation.

Cranial Nerves : Smell—Visual acuity; Fields of Vision; Optic Discs and Fundi—Pupils; External ocular movements; Nystagmus; Oculo-puillary sympathetic phenomena—Corneal reflexes; Sensation over face; Masseters, Temporals—Facial movements and symmetry of face—Hearing—Palate; Tongue; Sterno-mastoids and Trapezii.

Motor functions : (1) Power, (2) Cooperation; (3) Tonus; (4) Wasting and Fibrillation. Hypertrophy of muscles; (5) Involuntary movements and Fits.

Examination of Gait. Sensory functions: Cutaneous sensibility (1) Touch (cotton-wood) (2) Pain (pin-prick); (3) Temperature. Deep Sensibility (4) Joint sense; (5) Vibration; (6) Sensibility of Muscles and Tendons to Deep pressure. Stereognosis, Tactile Localisation, Compass Tests.

Reflex Functions : (1) Tendon Reflexes—Biceps, Triceps and Supinator Jerks. Knee-jerks and Ankle-jerks. Presence of Clonus.

(2) Cutaneous Reflexes.

(3) Micturition and Defaecation.

(4) Tonic Reflexes—Kering's Sign.

Spine and Cranium :—

Deformities or Tenderness.

Trophic Changes :—

Skin-bed sores, perforating ulcers. Bones and Joints—Arthropathies, pes cavus.

A fairly detailed account is given of the clinical methods since it is possible for every Doctor to acquire a skill to make the correct diagnosis which is essential before giving the treatment. The habit of jumping to examination of the patient with a stethoscope before adopting the conventional procedures of inspection, palpation, percussion and

auscultation will lead to a wrong diagnosis and naturally to a wrong treatment.

The next duty of a Doctor is to be familiar with the latest modes of treatment. It is with this object in view that recent trends in therapeutics will be discussed now.

—(To be Continued.)

THE INDIAN COUNCIL OF CHILD WELFARE

TIRUCHY DIST. BRANCH

President :

Sri G. M. BADSHA, I.A.S.,

Dist. Collector.

Hony. Secretary :

Dr. P. A. S. RAGHAVAN,

Z.M.R.E.

ANNOUNCEMENT

The above Council is willing to help indigent handicapped children to rehabilitate them either by treatment or with aids such as Crutches, Correction boots, Hearing aids, Spectacles etc.

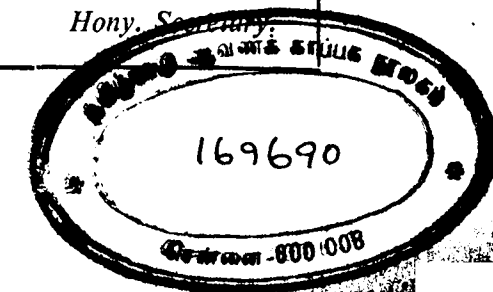
Doctors in Tiruchy District can write to the undersigned, the age, name and address of the handicapped children that require help, the nature of assistance they require and the probable cost of the same. A certificate testifying that the child is too poor to be helped by the parent or relations may also be sent.

8, WILLIAMS ROAD,
CANTT., TIRUCHY-1.

P. A. S. RAGHAVAN,

Hony. Secretary.

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CORONARY INSUFFICIENCY

By

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

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

THE clinical manifestations of coronary insufficiency comprises the following three conditions:— (1) Angina pectoris (2) Status Angina or Acute coronary insufficiency without thrombosis and infarction; and (3) coronary thrombosis, better termed cardiac infarction.

Definition: Coronary insufficiency is a condition where there is a relative deficiency or inadequacy of supply of blood to the cardiac muscle, for its needs in performing its work at any time; in other words its physiological needs are not adequately met with. Cardiac ischaemia is the result. Normally cardiac reserve is such that when the heart works during exertion, it receives augmented supply of sufficiently oxygenated blood, that its requirements for the extra work is squarely and perfectly met with. When this physiological adjustment is disturbed there is a relative ischaemia of the cardiac muscle, the condition being called Coronary insufficiency.

Frequency: In recent years there is a definite increase in coronary disease. The increase is not due to an efficiency in diagnosis or due to an increase in the span of life as once thought. But the incidence is definitely on the increase probably due to the modern strenuous materialistic life. In the pre chemotherapeutic days (1939) Pneumonia was the captain of death. Now coronary disease and coronary thrombosis has taken its place as the Captain of death. The majority of cardiac deaths are due to coronary thrombosis. This fact, that in these modern days there is a definite increase in the incidence of coronary disease, support the suggestion that the disease is due to environmental conditions

rather than hereditary. The greater incidence in certain families is probably due to the environmental condition rather than due to hereditary dispositions or influences.

Aetiology.

Race: In primitive races the disease is rare. The incidence of hypertension and atherosclerosis, the two primary causes of coronary heart disease is very much less in these people. Contentment and simple living which we find in these people, are probably the two major factors which prevent the occurrence of hypertension and atherosclerosis.

Sex: The disease incidence is high in men, being very rare in women at present. The word 'at present' is important. For when women change their mode of life, and from one of home life to one of public life and when the environmental factors are the same for them as for men then the gap of difference in incidence may become less and less. Another reason why women rarely get this disease is perhaps because they do much physical labour at home. One has estimated that the average Indian woman walks nearly 8 miles per day in the house in doing all the household works.

Age: The disease is very common in the fifties and sixties. The majority of deaths from cardiac infarction occur round about the fiftieth, or 55th year when the man is preparing to retire from service. Deaths from coronary disease has also occurred in younger people of thirty or less.

The third and perhaps important factor in the rarity of the disease in women is perhaps that the entire responsibility for the maintenance of family is left to the male partner, her life being one of resignation and contentment.

My youngest patient who died of cardiac infarction was aged 28 years. Death from coronary heart disease is also rare after 60 or 70 years.

Physiognomy: Stout, stocky people are supposed to get the disease more frequently than the lean people. This is not invariable. I should think it is the mental status rather than the physical status that predisposes to hypertension and coronary disease.

Social status: The disease is extremely rare in the labouring class of people who do physical labour. Physical work leads to better supply of blood to the heart muscle. Recently the British Heart Association sent a circular to nearly 250 Hospitals to supply them with the post-mortem findings of the heart in their post-mortem records. From the examination of records of 5,000 postmortems, they have found that the heart of a working man is 15 years younger than that of a professional man, e.g. a Manager in an Office, Clerk or Secretary in an office whose work entails no physical work. They have also found that coronary heart disease is more common in bus drivers, but not in double deck bus conductors. This is because the latter goes up and down, i.e. moves about in issuing tickets. I have recently seen (March 1959) a washerman dying of coronary thrombosis whom I had examined on two or three occasions with Anginal pain about 2 weeks prior to his sudden death.

The disease affects the professional class much more frequently than the others employed in jobs involving physical exertion. Even here it affects the highly

ambitious, conscientious, and rigid type of person who rarely relaxes. It also affects one who indulges in rich greasy food with much animal fat. Atheroma of the coronary arteries is more common in these class of people with or without diabetes mellitus.

It is said that musicians rarely get the disease. Singing devotional songs perhaps acts as a great soothing both to the soul and mind. Therefore it is thought hypertension is rare in this class of people. I have no statistics to prove or disprove this surmise. But perhaps there must be some truth in this since devotional songs elevate the mind and soul not only of the singer, but also those of the hearer. All worldly worries are forgotten at least during the time of singing and thus the stage for hypertension is removed.

It can be said that coronary heart disease is very rare in the truly religious people. That is a life of contentment and mental calmness, derived from a firm trust in God. There is no frustration to them. The causes of hypertension may be classified as follows:—

- (a) Primary factor.
- (b) Secondary predisposing influences favouring the development of the primary etiologic factors.
- (c) Precipitating factors.

The primary factor in the causation of coronary heart disease is atherosclerosis of the coronary arteries. In nearly ninety percent of cases coronary atherosclerosis with narrowing and occlusion is observed. In the absence of atherosclerosis in about 10% of cases other causes such as hypertension, valvular disease of the heart syphilitic aortitis with or without regurgitation, peri-arteritis nodosa thrombo angitis obliterans etc., operate in inducing cardiac pain or infarction.

Coronary atherosclerosis is perhaps a sign of aging process. In a mild form it may exist in the majority of elderly people. A. D. Morgan thinks that atherosclerosis and arteriosclerosis by themselves do not cause narrowing of the blood vessels. From the very nature of it, viz., fibrosis with destruction of elastic fibres of the vessel wall, the lumen should become wider with the lateral pressure of the blood flow, which is either normal or more as in hypertension. He is surprised how this view hitherto remained unassailed by all thinking medical men and pathologists. The narrowing in atherosclerosis is due to a haemorrhage into the intima or a small thrombus on the wall which becomes the seat of infiltration with Cholesterol and other lipid material as a part of organisation of the thrombus. He agrees with Duguid's observation that atherosclerosis of the coronaries may arise in two ways (a) by lipid infiltration and (b) by thrombosis. The thrombus undergoes atherosclerotic changes exactly similar to the primary atherosclerosis due to lipid infiltration.

That the narrowing of the lumen of the arteries in arteriosclerosis is only due to a previous intimal haemorrhage or due to an organising thrombus on the wall, we do not agree. For in cases of essential hypertension or hypertension secondary to nephritis, we can feel the arteries of the limbs uniformly thickened narrow and hard like whip cord. Fundoscopy shows also the narrowed retinal arteries with a thin stream of blood traversing this lumen, the lumen being uniformly narrowed throughout.

But in diabetes with lipoamia on the otherhand, there may occur irregular narrowing of the lumen of the blood vessels due to atherosclerotic patches protruding into the lumen. These areas may become the seat of calcium deposits and give rise to Monkeyberg type of arteriosclerosis. Further, in any case of

long standing atherosclerosis there may occur irregular narrowing or occlusion on account of the deposition of cholesterol on the diseased wall or a small thrombus on the wall in undergoing organisation become deposited with cholesterol. Final total occlusion occurs either with the breaking of an atherosclerotic ulcer or from the formation of a fresh thrombus in the previously narrowed area.

Therefore it can be said that in the majority of cases of coronary insufficiency, the underlying lesion is atherosclerosis of the coronary arteries with narrowing and complete obstruction.

In 10% of the cases coronary insufficiency leading to cardiac pain may be due to other factors than coronary atherosclerosis. Arterial hypertension and valvular disease increase the work of the heart even under basal conditions and limit the cardiac reserve and leads to cardiac ischaemia with cardiac pain or distress on exertion. In others the following conditions may be responsible for the cardiac pain. Syphilitic aortitis with or without aortic regurgitation, peri arteritis nodosa, thromboangitis obliterans, rheumatic arteritis, scleroderma, amyloid, degeneration corpulmonale with chronic pulmonary infection and anoxaemia and the arteritis or myocarditis associated with systemic infections may be encountered.

Secondary predisposing factors; These are discussed already in the earlier paragraphs dealing with aetiology, sex, age, race and type of person in whom it is frequently found.

Coronary insufficiency leading to cardiac pain or death from infarction is frequently encountered in patients with arterial hypertension, diabetes, polycythaemia vera, thromboangitis obliterans and peri arteritis nodosa. Spasm and narrowing of the lumen of the coronaries

occurs in hypertension leading to thrombosis and occlusion. Diabetes favours atherosclerosis of the coronaries. Polycythaemia vera favours intra vascular thrombosis. Thromboangitis obliterans and peri arteritis nodosa also favour intra vascular thrombosis. In cases of aortic regurgitation of syphilitic or rheumatic origin, the diastolic pressure in the aorta is low and this interferes with the coronary flow. Hence cardiac pain is a frequent symptom in this condition. Aortic stenosis may distort the coronary mouths and lead to cardiac pain due to ischaemia.

Myxoedema is frequently associated with cardiac pain. Atherosclerosis of the coronary arteries is favoured by high cholestero-loemia in this condition and the cardiac ischaemia is due to this.

Gall bladder disease as such does not lead to angina pectoris, but the two diseases are frequently associated in the same individual.

It is suggested that when coronary insufficiency is associated with other diseases in upper part of abdomen as e.g. gall bladder disease, peptic ulcer, etc., the anginal pain may be referred to these diseased organs.

Anaemia with anoxaemia may also lead to cardiac ischaemia and pain. Correction of anaemia if the coronary arteries are not diseased will result in complete cessation of cardiac pain. This has been observed in a case of mine where a man aged sixty years without hypertension or diabetes was suffering from cardiac pain. His motion showed anky ova and his blood showed a secondary or normocytic hypochromic anaemia. Correction of the anaemia resulted complete cure. It is since four years that he did not get any cardiac pain.

Mitral stenosis is another condition where cardiac pain may be a dominant

symptom. The small out put of the left ventricle with greatly reduced head pressure of the blood flow reduces the coronary flow and leads to cardiac ischaemia. Rheumatic arteritis may be yet another factor.

Precipitating factors

A sudden spasm may be responsible for the acute coronary insufficiency characterised by prolonged cardiac pain. Anginal pain of short durations occurs on exertion such as brisk walk or a walk up the slope or climbing the steps or a walk after a heavy meal. In states of anxiety and worries especially that of any litigation or income-tax the diseased and narrowed coronary arteries may get spasmodically contracted resulting in pain even while resting. If prolonged, thrombosis occurs resulting in cardiac infarction.

Acute Dehydration may be another factor favouring intra arterial clotting on account of concentration of blood. Cardiac infarction sometimes occurs after severe diarrhoea or burn.

Exposure to chill may induce cardiac pain in one whose coronaries are already the seat of atherosclerosis. A brisk walk in the morning chill weather may give rise to cardiac pain the person not getting similar pain when a similar walk is taken during the rest of the day.

A sudden death from coronary thrombosis is not unknown when a sudden exciting or even happy news is heard. Deaths have occurred on hearing news of a sudden realisation of a heavy fortune from a raffle or horse race. Spasm with thrombosis must have been the cause of death.

Lodging of emboli in the coronary arteries, the emboli coming from the periphery or cardiac chamber may be responsible for cardiac infarction and sudden death.

In a case of sudden death in one patient suffering from mitral stenosis who was moving about at the time previous to death, I presume death must have resulted from coronary block by an emboli from the heart itself (Intra mural thrombus). This is only a surmise and I think such a thing can occur. She complained of uneasiness in the chest, and soon was dead.

Cardiac pain due to ischaemia therefore can occur under the following clinical conditions.

(a) In the elderly or middle aged person with primary atherosclerosis of the coronary arteries.

(b) In diastolic hypertension essential and nephritic.

(c) In diabetes mellitus, sometimes with insulin treatment.

(d) In thyroid deficiency i.e. myxoedema.

(e) In polycythaemia, primary (Polycythaemia vera) or secondary to pulmonary sclerosis and hypertension.

(f) Concentration of the blood as after gastro intestinal upset with vomiting and or diarrhoea. After a cathartic purgative.

(g) For similar reasons after severe burns or after haemorrhage.

(h) Anaemias especially the secondary anaemia due to iron deficiency.

(i) Valvular diseases of the heart.

Aortic regurgitation mitral or rheumatic.

Aortic stenosis with distortion of the mouths of the coronaries.

Mitral stenosis.

(j) Syphilitic aortitis and rheumatic arteritis.

(k) Congenital heart disease viz cardiomegaly due to one of the coronary or

both coronaries arising from the pulmonary artery.

(l) Constrictive pericarditis.

(m) Cardiac arrhythmias especially in paroxysmal auricular tachycardia. The diastolic interval is so shortened the coronary flow is considerably decreased resulting in cardiac ischaemia.

(n) Emboli peripheral or from the heart chambers.

Septic as in ulcerative endocarditis or subacute bacterial endocarditis.

(o) Disease of the arteries as in periarteritis nodosa, thrombo-angitis obliterans etc.

Clinical manifestations

Cardiac ischaemia is manifested by chest pain. The pain characteristically comes during exertion and not after the exertion is over. A typical history will be as follows. A middle aged individual who did not have any pain in the chest on walking any distance gets retro-sternal pain after walking some distance. The pain increases in intensity as he proceeds and then he is unable to proceed any further but has to stop walking for a few minutes when the pain disappears completely and then he can proceed in his journey without getting any pain. During the attack of pain he has to sit or stand still, there is pallor of the face, and sensation of impending dissolution. For each individual the pain comes exactly after the same amount of exertion unless the underlying pathology is progressive, when the pain may come with lessening amount of exertion or later even while at rest.

Nature of pain

The pain is that of a sensation of constriction as if held in a vice, or dull pressure or a squeezing pain or a sensation of burning or glow in the centre of the chest. It is never pricking or stabbing in character.

Situation :

It is characteristically situated behind the sternum (retrosternal) or in some cases over the praecordium. It is never inframammary. In some it may be so low behind the sternum as to simulate epigastric pain.

Duration :

The pain is of short duration and is over in a few minutes after resting or after Trinitrin.

Radiation :

It may remain localised or may radiate to either shoulder but more characteristically to the left shoulder and then down the inner border of the arm to the forearm and hand where it characteristically radiates to the little or ring finger.

Sometimes the pain may radiate to the tooth, jaw, neck, below to the epigastrium and to the back. If the patient is a subject of peptic ulcer it may radiate to the epigastrium or if suffering from gall bladder disease the pain may be referred to the gall bladder region to simulate gall bladder disease. If he is the subject of carious teeth with root abscesses, the pain may be referred to diseased tooth. In all these conditions if the retrosternal pain is mild or absent the danger of missing the right diagnosis is possible with fatal results.

Therefore it can be summarised that cardiac pain is retrosternal, comes during exertion, disappears on rest or with trinitrin with a characteristic of its own in that it is a dull, heavy or squeezing or constricting pain, and radiates to the various places mentioned above.

Rarely pain may start and remain in the areas where the pain usually radiates, though characteristically occurring after exertion. In these cases the right diagnosis

will be missed especially if the patient has had no previous attack of chest pain and may be treated for a local condition and his death from coronary thrombosis will reveal that the original pain was due to coronary thrombosis. I recall the instance of a medical practitioner who died of coronary thrombosis whose complaint was of severe pain in his carious tooth.

Another senior medical man died of coronary thrombosis who complained of epigastric pain which was attributed to dyspepsia and the cardiac condition was not thought of as he till then did not suffer from cardiac pain. Yet there may be present some unusual features which help to show that it is due to coronary insufficiency. These, are, the immobility or restlessness, perspiration pallor or some difficulty in breathing.

In acute coronary insufficiency the pain is prolonged, though its other features are the same as in Angina Pectoris. There is no coronary block and so no cardiac infarction. The attack is probably due to a prolonged spasm of the coronary vessels. The sudden diminution of blood supply or greatly increased cardiac work is responsible for this condition in the majority of cases as can be judged from the situation in which acute coronary deficiency can occur viz (1) Haemorrhage and other causes of shock and hypotension (2) Acute heart failure (3) Asphyxia and especially carbon monoxide poisoning (4) Acute severe pulmonary disease (increased demand) (5) Pulmonary embolism (6) Severe effort or great emotional stress (prolonged spasm) (7) Paroxysmal tachycardia (8) Acute hypertension and (9) change to a high altitude in the first few days (anoxaemia) of occupation. The features of Angina pectoris, coronary insufficiency without infarction and cardiac infarction are given below in a table form.

Angina pectoris (1)	Acute Coronary insufficiency (2)	Acute myocardial infarction (3)
Clinical : Pain (Retrosternal) on exertion	Pain retrosternal may be on exertion and is usually associated with the conditions which predispose to its occurrence.	Pain retrosternal. No necessary relation to exertion. Chill and emotion may precipitate the attack.
Duration of pain : Short - 15 minutes	Prolonged into several minutes	Prolonged
Response to Nitroglycerine : Striking	No response or temporary	None
Signs of shock and fall of Blood pressure etc.: Nil. Blood pressure may arise during the attack	Unusual excepting when occurring with shock	Frequently present
Death : Uncommon	Uncommon	Frequent
Evidence of myocardial necrosis Absent	Absent	Frequent (Leucocytosis) fever raised E.S.R.,
Pericardial rub : Absent	Absent	May be present (10%)
E.C.G. Changes : No change or transient changes in S. T. Segment or T-waves	Same as in Angina	Progressive typical pattern

—(To be continued)

SURGICAL SHOCK—MODERN TRENDS IN TREATMENT

By

Dr. S. KALYANARAMAN, M.S.,

Asst. Professor of Surgery, Stanley Medical College, Madras.

It is well known that there are a set of reflexes in the human body constituting a natural defence whenever there is a diminution of the volume of circulating blood. Of these reflexes the carotid sinus mechanism plays a very important part. If due to any reason, these mechanisms fail, what is at present termed as irreversible shock, sets in. But it should be realised that there is essentially no such entity scientifically as irreversible shock. What is irreversible today with our present methods of therapy may not be so tomorrow with newer methods of treatment.

Some of the newer trends in treatment of surgical shock stem from better understanding of its pathology. The most important recent advances relate to the understanding of the roles played by the adrenal cortex, the adrenal medulla and the autonomic nervous system in the pathology of shock and consequently the treatment of shock by hydrocortisone, by nor-adrenaline and by chlorpromazine.

The role of the adrenal cortex in the body's defences was first emphasised by Hans Selye in his concept of the General Adaptation Syndrome—consisting of the Alarm reaction, the Stage of resistance and the Stage of exhaustion. In cases of subclinical adrenal cortical deficiency minor injuries and infections are combated successfully but in case of major infections, haemorrhage or surgical operations unaccountable sudden severe shock sets in.

In such cases replacement of the deficient cortical hormones is reasonable.

Cortisone or ACTH however is not recommended. In those cases in which it helped to raise the blood pressure it was found that, in not a few, after the drug was withdrawn shock reappeared suddenly after a variable period and sometimes proved fatal. Hydrocortisone however appears to be free from these dangerous effects of withdrawal. Water-soluble hydrocortisone hemisuccinate sodium can be given either intravenously or intramuscularly.

It has been shown that if the blood pressure remains below a critical level for more than a critical period, irreversible changes set in due to anoxia of tissues as yet unknown. Nor adrenaline which is free from the dangerous effects of adrenaline is often used as a drip to maintain the blood pressure at a sufficiently high level until the body's natural defences are able to take over.

It is advised especially in desperately shocked patients who fail to respond to the usual measures. It is important to diagnose the cause of the shock before noradrenaline is given, lest concealed haemorrhage be aggravated by the drug. Noradrenaline is a powerful peripheral vasoconstricting agent that produces a rise in both the systolic and diastolic pressures provided of course that there is sufficient circulating blood to effect this all important change. It should never be given until after the more usual methods have proved ineffectual. Noradrenaline must always be given intravenously commencing with a very dilute solution. 2 cc of Noradrenaline is added to a bottle of 500 cc of glucose saline. To commence

(Summary of a lecture delivered at The Tiruchy Medical Association on 16—5—1959.)

with, the rate of administration is 30 drops a minute but a faster rate upto 40 drops a minute of a more concentrated solution, upto 10 times this strength has been used with success. As the blood pressure rises the rate of the drip and the concentration of noradrenaline are reduced gradually to 7 drops a minute. Blood pressure readings should be taken at ten minutes intervals throughout day and night. If as a result of slowing the rate of the drip, the blood pressure again falls, the rate of the drip is accelerated. If a satisfactory blood pressure is maintained over several hours, the drip is slowed gradually until it is considered safe to withdraw the cannula from the vein. A considerable number of cases of extensive necrosis around the site of infusion have followed the use of noradrenaline. As a preventive measure, polythene tubing should be passed some distance into the vein allowing more rapid dilution of the drug by the circulating blood.

Noradrenaline is wonderful drug in cases of emergencies. There are reports of cases which were given noradrenaline for 20 days, 22 days at a stretch. There is no doubt that in such instances Noradrenaline was the sole lifesaving factor.

The most recent advance on which I want to lay stress is what is referred to as 'Artificial Hibernation' or 'Intravenous lytic cocktail'. Simple to administer, it is extremely efficient in its results.

Artificial hibernation is so called because the state produced by the pharmacological suppression of the sympathetic nervous system resembles closely the oblivion, the low metabolism and the respiratory and cardiac rhythm of a hibernating animal. Of several combinations of drugs employed for this purpose, the most favoured by majority consists of

Chlorpromazine Hydrochloride or Largactil 50mg.

Promethazine Hydrochloride or Phenergan 50mg.

Pethidine Hydrochloride 100mg.

All these 3 drugs are dissolved in 14ml of normal saline solution and given I. V. in divided doses.

These drugs create a partial blockage in pathways to the vital centres including the vasomotor centre. Thus the sum total of noxious stimuli causing and maintaining shock that reach the hypothalamus is by reason of action of these drugs, greatly reduced.

Provided artificial heating of the patient is disallowed, some degree of hypothermia results, metabolism is decreased and the oxygen demands of the tissues are reduced correspondingly. Another most important effect is that the peripheral vasoconstriction of shock passes off, permitting blood or blood substitutes to be gravitated into a vein or veins without hindrance.

Artificial hibernation is indicated particularly in those cases of profound shock that, without it would be classified as irreversible. It has proved priceless when urgent operation is imperative but the patient is in a profound shock and has enabled operative treatment to be undertaken without adding to the shock further.

Artificial hibernation greatly decreases the complexity of skilled nursing care, otherwise inseparable from desperately ill patients. The only accidents to be avoided are firstly overhydration in a semiconscious patient. It is always better to use subcutaneous saline with hyaluronidase in place of intravenous saline, once blood pressure is restored to normal. Secondly no post operative sedation should be used, for an additional dose of morphine at night, may well produce fatal respiratory depression.

ASSOCIATION NEWS

CHETTINAD BRANCH

The monthly meeting of the above association was held on 29th August '59 at the S. M. S. High School, Karaikudi. Dr. A. Vaidyanathan, MBBS presided.

Election to the office of the President and two Vice-presidents of the Madras State Branch of I.M.A. was gone through and the votes recorded. Major G. A. Naidu, MBBS FDS (London) Hony. Lecturer in Dermatologist, The Erskine Hospital, Mathurai gave a lecture on 'Common Skin diseases and their treatment'.

COIMBATORE BRANCH

A monthly meeting of the Association, was held on Saturday, the 26th September 1959 in the Association premises. Dr. D. Sundareswaran, the President, presided.

The President congratulated the following:—

1. Dr. T. V. Sivanandam for having been elected as a member of the Indian Medical Council, and on his re-election as President of the Madras State Branch of I. M. A.
2. Dr. A. G. Leelakrishnan, for having been elected as a senior Vice-President of the Madras State Branch of the I.M.A.
3. Dr. C. V. Ramraj, for having returned after the continental tour.
4. Dr. Sarojini, for having returned after the studies in U. K.
5. Dr. S. G. Rajaratnam for having been elected as a Zonal Chairman of the Lions Club for the districts from Colombo to Madras.

Dr. M. V. Krisnamurthy, B.A., M.B., B.S., M.R.C.P., D. T. M. & H., District Medical Officer, Coimbatore, delivered an interesting and elaborate lecture with X-Ray pictures on "The Common Congenital Defects of the Heart" which was followed

by a Film Show on "Atrial Septal Defects" by May and Baker Limited which was enjoyed by all the members.

MADURA BRANCH

A monthly meeting of the Association was held on 26th September 1959, under the Presidentship of Dr. K. Gopal, MBBS, TDD, at Thekkady. The following condolence resolution was moved. "It is with profound and deep sense of sorrow we the members of the Madura Medical Association express our heart felt sympathies on the demise of Dr. A. Narayana Menon, MBBS, Ex-president, Ex-secretary, and Life member of our Association." The Association decided to invite the XIVth Madras State Medical Conference to be held at Madurai in January 1960. Dr. S. Perumal Rajan, B.D.Sc. Dental Surgeon, Erskine Hospital, Madurai, gave an interesting lecture on "Dental Surgery in General Practice."

NILGIRI BRANCH

The monthly meeting of the Association was held at the Lawley Institute, Ootacamund on 26th September 1959 with Dr. (Major) T. D. Jeevarathnam, the President in the chair.

Dr. P. V. Kurian, M.B., B.S., D.T.M., M.S. (Mich.), Assistant Director, Pasteur Institute, Coonoor read a paper on "The Hospital Staphylococcus."

SALEM BRANCH

A meeting of the above Association was held on Saturday, the 26th September, 1959 at Foulkes Hall, District Board, Salem.

Dr. C. S. Sadasivam, M.S., Professor of Surgery and Thoracic Surgeon, Government General Hospital, Madras and Dr. A. L. ANNAMALAI, M.R.C.P., Physician, Government General Hospital, Madras

addressed the members on "Modern trends in the treatment of diseases of chest - Surgical and Medical aspects".

TIRUNELVELI BRANCH

The Annual meeting of the Association was held on Saturday, the 26th September 1959 at St. John's College premises, Palayamkottai. Dr. G. Victor, M.D., Professor of Medicine, Madurai Medical College & Superintendent, Govt. Erskine Hospital, Madurai addressed on "Precardial Pain". Dr. Sam G. P. Moses B. Sc., M.D., Hony Physician, Govt. General Hospital, Madras, addressed on "A few trends in Diabetes Mellitus".

The following office bearers have been elected for 1959-60.

President :

Dr. P. Ragavendra Acharya,

Vice-President :

Dr. S. Maragathavel.

Honorary Secretary & Treasurer :

Dr. P. Vadivale.

Members for the Managing Committee :

1. Dr. K. Rama Ayyar,
2. Dr. R. Subramaniam,
3. Dr. S. Chandrasekaran,
4. Dr. D. I. Abraham,
5. Dr. V. Sridhara Rao,

Member-Auditor :

Dr. R. V. Chockalingam,

Member for the Central Council :

Dr. K. Ramachandra Naidu.

Members for the Madras State Branch :

1. Dr. K. Rama Ayyar,
2. Dr. S. Chandrasekaran,
3. Dr. R. Subramaniam,
4. Dr. (Capt.) S. Padmanaban.

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ANNOUNCEMENT PRIZE COMPETITION ESSAY

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CONTEST RULES

1. The competition is open to all the medical practitioners in India (registered under the Indian Medical Council Act 1933).
2. The Essay should be written in the English language and should not exceed 3,000 words.
3. Six type-written copies, with double spacing and typed on one side of the page should be submitted.
4. Six type-written copies of the summary not exceeding 300 words should also be submitted.
5. The essay must be sent by registered post/hand delivery and addressed to "The Editor, The Bombay Hospital Journal, 19, Marine Lines, Bombay 1."
6. The result will be decided by a panel of judges and their decision will be final.
7. The prizes will be awarded only if the essays attain a high degree of excellence.
8. The contestants are requested **not to write their name and address** on any copy of the essay or summary.
9. The name and address of the contestant should, however, be written on the envelope as also on the covering letter.
10. The prize winners will be required to read, if necessary, their essays at a special function in **April 1960** (exact date to be announced later). Their air travel/air conditioned rail transport and stay for three days in Bombay will be provided for.
11. The result will be announced on **March 31, 1960** in the leading newspapers.
12. The prize distribution will be held at the Birla Matushri Sabhagar, 19, Marine Lines, Bombay 1, in **April 1960**. (exact date to be announced later).
13. Those wishing to enter the competition may kindly intimate the Editor.



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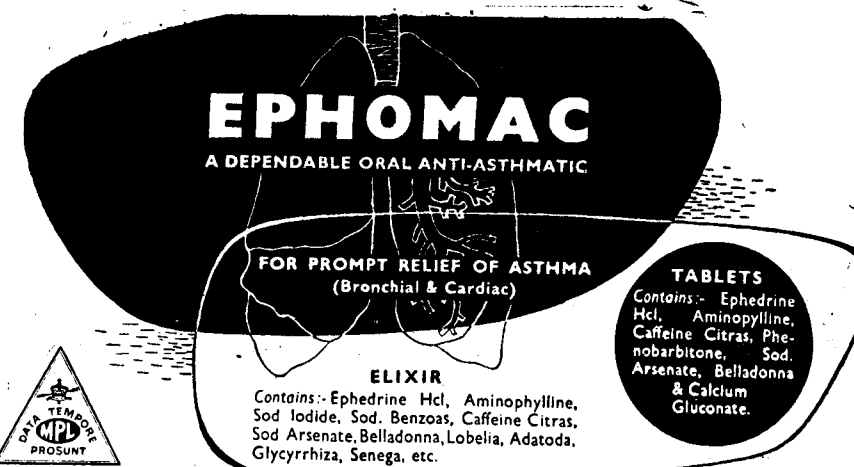
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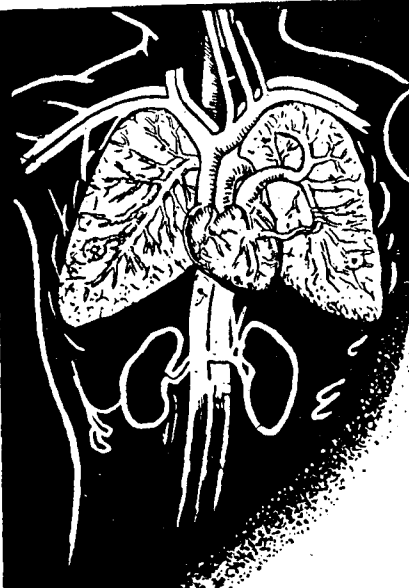
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Dr. B. A. Rajaratnam, Salem

Dr. B. A. Rajaratnam was born on 31st August 1899. He started general practice at Salem in 1923 and was in active practice till his sudden death on 17th July 1959. During his 35 years of very successful practice, he won the admiration, respect, affection and confidence of all his patients by intelligent and painstaking methods of diagnosis and treatment.

Besides his profession, he was taking part in political activities. He was a member of the Congress Party.

He was :—Chairman of the Salem Municipal Council from 1935 to 1938.

Member of the Senate, University of Madras.

Member of the District Board, Salem

President, Y. M. C. A., Salem.

President, Railway Passengers' Association, Salem.

Founder-President, Fairlands Building Society, Salem.

Founder-Member, District Club, Salem.

Mason of 26 years' standing till his death..

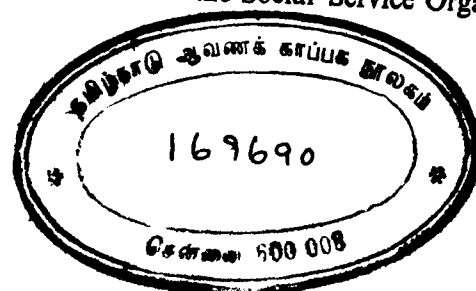
A very active member of the Indian Medical Association, he was President of the Salem Branch of I. M. A. in 1948-49 and was President of the Building Committee of Salem Branch of I.M.A. till his death.

In his death the country has lost a worker of outstanding ability, the medical profession a genial, courteous and sincere colleague and the Indian Medical Association an ardent member.

His genial and pleasant temperament endeared him to all who came in contact with him.



Dr. AMBAT NARAYANA MENON, M.B., B.S., well-known Medical Practitioner and prominent citizen of Madurai, passed away on the 21st of September '59. He was the first Secretary (for 7 years continuously) and one of the Founder members of the Madura Branch of the I. M. A. He edited for 2 years a journal of the Madura Medical Association. Besides making a liberal contribution to Rs. 1000/- to the Building Fund of the Madura Medical Association, and serving on the Governing Body of the Local Branch, he served as a Vice-Chairman of the Reception Committee of the All India Medical Conference at Madurai in 1946. Born in 1894 at Chittur, Kerala State, he was educated at Chittur, Kozhikode and Madras, graduating in Medicine in 1920 from the Madras Medical College; he won the "Doctor Nair Gold Medal" for proficiency in Surgery. After serving as "House Surgeon and temporary Assistant Surgeon in the Presy. Hospitals, Govt. Maternity & Govt. General Hospitals from 1920-22, he set up practice in Madurai, which grew rapidly and attracted a wide clientele. He served as Hony. Venereologist Govt. H. Q. Hospital, Madurai from 1930-39. He was connected with almost all of the Social Service Organizations of the city of Madurai.



British Medical Journal - at concession rates for I.M.A. members.

The members of the Indian Medical Association, Madras State Branch, are reminded that facilities exist for subscription to the British Medical Journal at concession rates if subscribed through the I. M. A. Central Office. The concession rate works out roughly to one half of the actual subscription rate. Such of those members who may be desirous of availing this facility may remit a sum of Rs. 28-50 if by money order or a sum of Rs. 29-00 if by cheque to the undersigned who will take necessary further action and arrange to enrol them as subscribers to the Journal for one year. Such of those who are already subscribers in this scheme are advised to renew their subscription in time so as to have continuous supply of the journal. Subscribers are being individually reminded also; but this may be kindly taken as general reminder and necessary action taken. All subscriptions and renewal subscriptions at the rate aforementioned may kindly be sent to:

Dr. C. ARUMUGAM, M. B., B. S.,
Hony. State Secretary, Madras State Branch, I.M.A.,
328, Raja Street, Coimbatore.

ANNOUNCEMENT

At the recent election of one President and two Vice-Presidents of the Madras State Branch of I. M. A. held in September 1959 the results of which were announced by the State Council of the Madras State Branch of I. M. A. at its meeting held on 19-9-1959 at Cape Comorin, the following were elected to the respective offices shown against their names for the year 1959-60:

1. Dr. T. V. SIVANANDAM, M.B.B.S., M.L.C., — President
Hony. Surgeon, R. S. Puram, Coimbatore.
2. Dr. A. G. LEELAKRISHNAN, M.B.B.S., — Vice-President
Methil Clinic, Raja Street, Coimbatore. (Senior)
3. Dr. C. NATHAMUNI NAIDU, L.M. (Dub.), — Vice-President
Z. T. D. (vienna)
The Poly Clinic, Ambur, N. Arcot Dist.

The 14th Madras State Medical Conference will be held at Madurai, during January 1960, the exact dates of which will be announced later.

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

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.

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