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The Thirteenth Madras State Medical Conference - 1958

Madras

Souvenir

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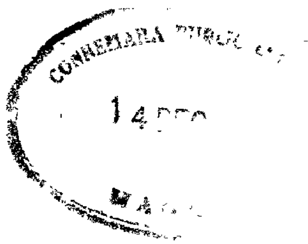

FOREWORD

It is with a sense of great elation that this Souvenir to mark the inauguration of the 13th Madras State Medical Conference is placed before you. We have tried to cater to the needs of all sections of reading Doctors and we are not fully justified in assuming that we have given optimum satisfaction. Due to limitations of time we have been handicapped to a large extent on the size aspect of this volume, but we hope we have provided a varied fare.

'Medical Economics' from Dr. K. S. Sanjivi is really thought provoking. You have the history of the State Medical Association from the President of I.M.A. with fleeting glimpses of years past. The article on 'Ashok Vihar' by its Director gives the various activities of this institution wishing us to provide one in every town. Medical, surgical, E.N.T. and skin diseases are discussed but the obstetrician is prominently absent and we hope in fullness of time, may deliver.

On the pictorial side we have not spared ourselves to make this a cherished book for a long time to come. We have had the fullest co-operation from all the leading Pharmaceutical manufacturers but for which this voluminous venture would have been unthinkable and unattainable. We express our indebtedness to them all.

Our printers "The Central Art Press" deserve our special thanks for having executed this volume with the ease of a leaflet and we have nothing better than our gratitude to offer them.

Convener—Souvenir

CONTENTS

PART I

PAGE

1. Welcome Address —Dr. P. Alagasingari Naidu ... 7
2. Presidential Address —Dr. T. V. Sivanandam ... 13
3. Messages ... 21

PART II

1. Problems and Experiences in the Surgical Treatment of Tetrology of Fallot —Dr. Reeve H. Betts & Dr. N. Gopinath ... 1
2. Diabetes Mellitus —Dr. M. Viswanathan ... 11
3. Respiratory Diseases in Infancy & Childhood—Dr. M.V. Chari ... 21
4. Medical Economics —Dr. K. S. Sanjivi ... 27
5. The Problem of Surgical Pain —Dr. K. A. Kalyanam ... 30
6. Diagnosis of Coronary Thrombosis —Dr. P. K. Krishnan Kutty ... 36
7. Some Syndromes of the Cervical Spinal Cord —Dr. K. P. Ganesan ... 39
8. Some Aspects of Hypertension —Dr. R. Subramaniam ... 50
9. Ashok Vihar—Health and Recreation Centre —Dr. P. S. Sambandam ... 57
10. Management of the Obese Uncomplicated Diabetic —Dr. G. C. Anbunathan ... 59
11. Modern Treatment of Leprosy —Dr. V. Ekambaram ... 63
12. Recent Advances in Medicine —Dr. K. Narayanamurthy ... 79
13. Upper Respiratory Tract *versus* Lower Respiratory Tract in diseases —Dr. G. Lakshmipathy ... 82
14. Common Skin Diseases —Dr. T. V. Venkatesan ... 86

PAGE

15. A Brief Sketch of the Origin and Activities of the Madras City Branch —Dr. D. V. Venkappa ... 99

16. Collections —Dr. V. Vijayaraghavan ... 102

17. Wise & Other Wise —Dr. V. Vijayaraghavan ... 106

PART III

Association Supplement

Members List ... 1

EXHIBITION

List of Stall Holders

1. MacMargaret Pharma Lab.
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3. Parry & Co.
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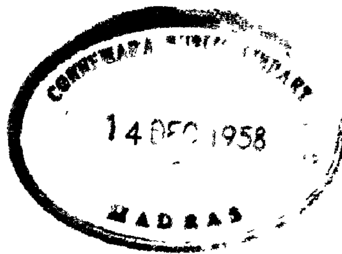

PART I

14 DEC 1958
MADRAS

13th MADRAS STATE MEDICAL CONFERENCE - 1958
MADRAS

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17. DR. V. R THAYUMANASWAMY
18. DR. D. V. VENKAPPA
19. DR. V. VIJAYARAGHAVAN

13th MADRAS STATE MEDICAL CONFERENCE - 1958
MADRAS

November 29th, 30th and December 1st 1958

(*PROGRAMME subject to alteration*)

29th November 1958

7-00 A.M. to 9-00 A.M.	Reception and Registration of delegates.
8-30 A.M. to 10-00 A.M.	Breakfast.
10-00 A.M. to 10-15 A.M.	Flag Hoisting. National Flag by Dr. Madhuram Santhosham, out-going President, Madras State Branch. Indian Medical Association Flag by Dr. D. V. Venkappa, President, Indian Medical Association.
10-15 A.M.	His Excellency the Governor of Madras arrives.
10-20 A.M.	Group Photo.
10-30 A.M. to 12-30 P.M.	Inauguration Ceremony. 1. Prayer. 2. Dr. P. Alagasingari Naidu, Chairman, Reception Committee—Welcome Address. 3. His Excellency the Governor's speech. 4. Sri M. A. Manickavelu, Minister for Health—Inauguration Address. 5. Installation of President Dr. T. V. Sivanandam by the out-going President Dr. M. Santhosham. 6. Dr. D. V. Venkappa, President, Indian Medical Association—Speech. 7. Presidential Address by Dr. T. V. Sivanandam, President, Indian Medical Association (Madras State Branch). 8. Reading of Messages—Dr. G. Ramachandra Murthy, Honorary Secretary. 9. Release of Souvenir—Dr. G. Sriramulu, Convener, Souvenir. 10. Opening of Exhibition by Dr. P. V. Cherian, Chairman, Madras Legislative Council. 11. Announcements : Dr. G. Ramachandra Murthy, Honorary Secretary. 12. Vote of thanks : Dr. B. Rama Rau, Honorary Secretary. 13. National Anthem.
1-00 P.M. to 2-00 P.M.	Lunch.

2-30 P.M. to 3-00 P.M.	Opening—Scientific Session. Presiding Officer :—Dr. V. R. Thayumanaswamy, Director of Medical Services, Government of Madras. Session declared open by Dr. A. Lakshmanaswami Mudaliar, Vice-Chancellor, University of Madras.
3-00 P.M. to 5-00 P.M.	Scientific Session.
5-30 P.M.	Tea.
6-00 P.M. to 8-00 P.M.	State Council Meeting.
8-30 P.M.	Dinner.
9-30 P.M.	Subjects Committee Meeting.

30th November 1958

8-00 A.M. to 9-00 A.M.	Breakfast.
9-00 A.M. to 12-30 P.M.	Scientific Session.
12-30 P.M. to 1-00 P.M.	Lunch.
2-30 P.M. to 4-30 P.M.	Scientific Session.
4-30 P.M. to 5-00 P.M.	Tea.
6-30 P.M. to 8-30 P.M.	Entertainment.
8-30 P.M.	Reception Committee Dinner.

1st December 1958

8-00 A.M. to 9-00 A.M.	Breakfast.
9-00 A.M. to 11-00 A.M.	Open Session.
11-00 A.M. to 1-00 P.M.	Scientific Session—(QUIZ)
1-00 P.M. to 2-00 P.M.	Lunch.
2-00 P.M. to 5-00 P.M.	Visit to places of interest.

13th MADRAS STATE MEDICAL CONFERENCE - 1958
MADRAS

INDIAN MEDICAL ASSOCIATION

Programme of Scientific Session

Date.	Time.	Name.	Subject.
29th, Nov. 1958.	3 P.M. to 5 P.M.	1. Dr. S. C. Sen, Ex-President of the Indian Medical Association.	Problem of Medical Economics.
		2. Dr. P. N. Rangiah, Additional Professor of Venereology.	Modern trends in the treatment of Venereal diseases.
		3. Dr. T. T. Ramalingam, Professor of Ophthalmology.	Modern trends in Ophthalmology.
		4. Dr. A. R. Govinda Rao, Professor of Pharmacology.	Modern trends in Pharmacology
30th Nov. 1958.	9 A.M. to 12-30 P.M.	1. Dr. C. Satyanarayana, Professor of E.N.T.	Cancer of the Larynx.
		2. Dr. M. Natarajan, Professor of Orthopaedics.	Modern trends in Orthopaedics.
		3. Dr. P. K. Krishnankutty, Honorary Physician, Government General Hospital.	Pulmonary Embolism.
		4. Dr. M. V. Govindaswamy, Director, All-India Institute of Mental Health, Bangalore.	Trends in Psychiatry.
		5. Dr. S. Krishnamurthy, Scientific Director, Cancer Institute.	Early diagnosis of Cancer.
		6. Dr. A. N. K. Menon, Professor of Radiology, Stanley Hospital, Madras.	Plain Radiography in Congenital Heart Diseases.
		7. Dr. T. Raghavan, Officer-in-charge, Blood Bank, Govt. General Hospital, Madras.	Report on some cases of Erythroblastosis Faetalis.
,,	2-30 P.M. to 4-30 P.M.	1. Dr. U. Ananda Rao, Assistant, E.N.T. Surgeon.	Modern Trends in E.N.T diseases.

Date	Time	Name	Subject
		2. Dr. M. G. Varadarajan, Assistant Radiologist.	Soft tissue radiography in Placenta Praevia.
		3. Dr. Harivaishnava, Vellore.	Pyelonephritis— A mimic of various diseases.
		4. Dr. M. S. Narayanan, Tiruchirapalli.	Changes in Serum Iron levels of Patients with Nutritional Macrocytic anaemia following single injections of vitamin B_{12} and Folic acid.

Quiz Programme

Date	Time		
1st Dec. 1958.	11 A.M. to 1 P.M.	*Moderator* :	Dr. P. Arunachalam.
		Members :	Dr. K. G. Krishnaswami—Professor of Surgery.
			Dr. R. Subramaniam—Professor of Medicine.
			Dr. P. Vedachalam—Professor of Pathology.
			Dr. K. Anandan—Professor of Physiology.
			Dr. S. T. Achar—Professor of Paediatrics.

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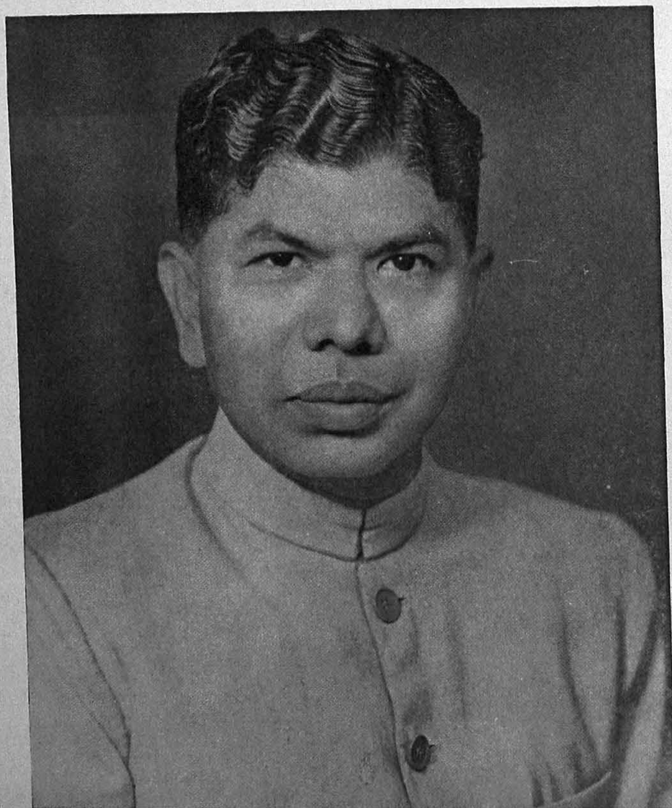
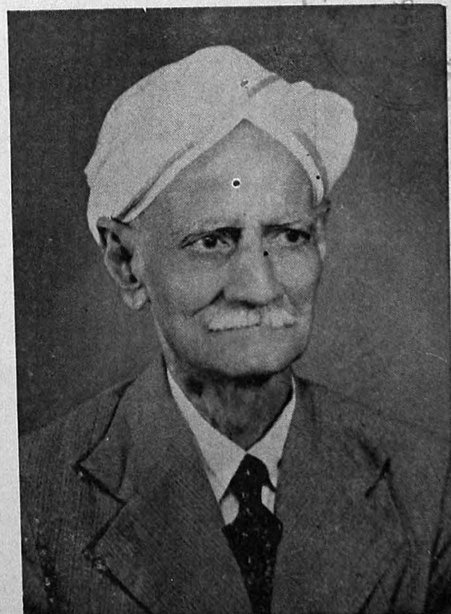


Dr. M. Santhosham, President, Madras State Branch I.M.A. (1957-58) unfurls the National Flag.



Dr. D. V. Venkappa, President, Indian Medical Association (Central) unfurls the I.M.A. Flag.

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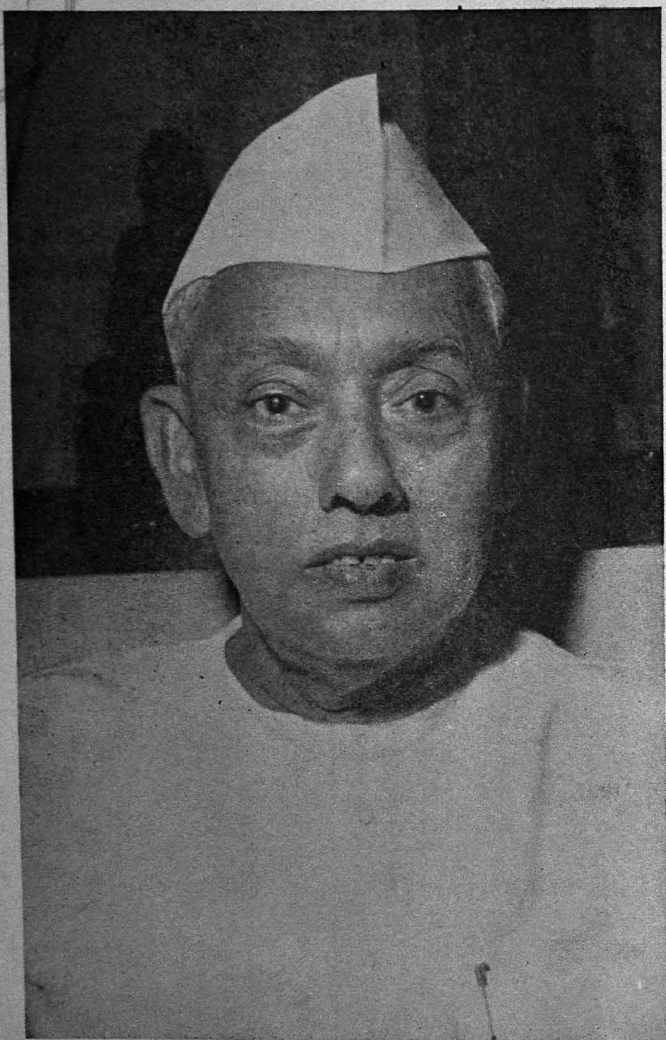
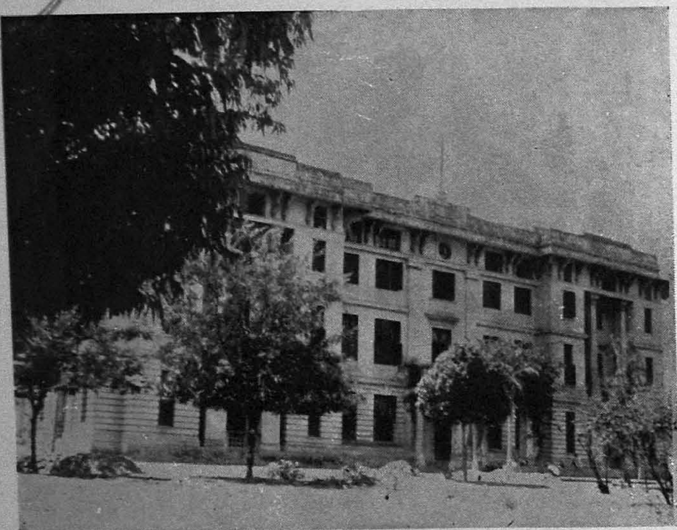


Sri Bishnuram Medhi, Governor of Madras—addresses the Conference.

Sri M. A. Manickavelu, Minister for Public Health, Govt. of Madras—inaugurates the Conference.



Madras Medical College—Venue of the Conference.

Dr. P. Alagasingari Naidu,
Chairman, Reception Committee.

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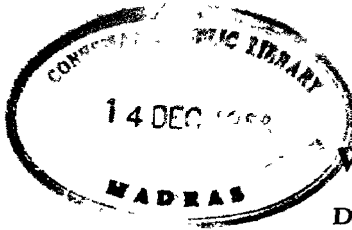
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WELCOME ADDRESS

Dr. P. ALAGASINGARI NAIDU
Chairman, Reception Committee

Your Excellency Bishnuram Medhi, Governor of Madras,
Sri M. A. Manickavelu, Honourable Minister for Public Health,
Dr. T. V. Sivanandam, M.L.C., President Elect of the 13*th Madras State Medical Conference of Indian Medical Association,*
Director of Medical Services,
Dr. Venkappa, President, Indian Medical Association,
Brothers and Sisters in Profession,
Ladies and Gentlemen,

It is a great privilege and honour to me to welcome you today for the 13th Madras State Medical Conference and Exhibition. I take the first opportunity to thank all the members of the Reception Committee who have placed their confidence in me and entrusted me with this honour. I will endeavour to discharge my duties to the best of my abilities.

I extend a hearty welcome to your Excellency and I thank you for accepting to preside on this occasion.

I heartily welcome you, Sri M. A. Manickavelu, Honourable Minister for Public Health. You are taking a very keen interest in all Public Health matters and may I request you to give us a sympathetic and patient hearing. Your acceptance to inaugurate this conference is further evidence of your interest in not only promoting Public Health but also in the activities of our Association.

I sincerely welcome you the President elect of the Madras State Branch of the Indian Medical Association, Dr. T. V. Sivanandam, M.L.C., and congratulate you on your election. I sincerely believe that in you we have a strong champion to fight for our cause. In the course of this welcome address I have taken the liberty to make a few observations which I hope will be of some use to you during the tenure of your office.

I welcome you Dr. Thayumanaswami, the Director of Medical Services, who by your kindness and sympathy endeared yourself not only to the servicemen but also to all of us.

I heartily welcome our beloved Septuagenarian Dr. Venkappa, who now occupies the enviable position of the President of the Indian Medical Association. You have been a fighter all your life and you have sacrificed your lucrative practice for the sake of your colleagues. I thank you sincerely and your presence today in your Home State Conference has given us additional zeal and enthusiasm.

I thank you once again for accepting to hoist the Indian Medical Association Flag today.

It is with great pleasure I welcome to this conference my brothers and sisters in the profession who have taken the trouble to come from the nooks and corners of the State. I request you to join and co-operate with us in the deliberations of this conference and give us your valuable counsel. We have made some arrangements for your stay here. I am afraid these arrangements may be inadequate and we are conscious of our several shortcomings. We request you to bear with us and excuse our faults if any.

It gives me great pleasure to welcome Dr. U. Krishna Rao, Honourable Speaker of the Madras Legislative Assembly. You are one of us and have been a friend, philosopher and guide to us. I thank you for your acceptance to hoist our national Flag.

With unalloyed pleasure I welcome Dr. P. V. Cherian, Chairman, Madras Legislative Council. You, after a brilliant medical career, are guiding our Legislative Council for the second term. I sincerely thank you for your kind acceptance to open the exhibition.

It is with great pleasure I welcome Dr. A. Lakshmanaswami Mudaliar, who had been a teacher to almost all of us. In your experienced hands, Sir, the autonomy of the university and education are safe.

My task of welcoming will not be complete till I welcome our Dean R. G. Krishnan and Vice-Principal, Dr. Janardhanam. I take this opportunity to thank you on behalf of the Reception Committee for your help and advice to make this conference a success.

It is customary in a welcome address given by the Chairman of the Reception Committee to describe our Great City of Madras, being the venue of the 13th Madras State Medical Conference. Madras is the centre of Medical Education and is also the Capital and the seat of the Government. All of you would have been well acquainted with this city in one way or other. I feel an exhaustive description of our city will be superfluous. Therefore, I will confine myself to few points only. Madras has grown very big during the last decade from an area of 13 and odd square miles to 50 square miles. All around the city beautiful suburbs are fast developing. The Marina, considered the second best in the world, is still in the same old condition and we are hoping for a day when the beach is made a continuous belt of foreshore extending from the Napier Bridge to Elliot Beach, attracting tourist traffic during our cold months and incidentally raising an extra and enviable income to our State. The harbour is being enlarged and within a few years it will be one of the biggest artificial harbours in our country. Six crores of Rupees have been sanctioned by the Centre for this purpose. Madras is not an industrial town, but a big business centre. However, the Integral Coach Factory, the Buckingham and Carnatic Mills, T.I. Cycles of India, Western India Match Factory are some of the industrial works in our city. We have a big stadium in Peoples' Park comprising 13 acres of land. The galleries are designed to accom-

modate about 30 to 40,000 spectators. There are six Cricket pitches in the centre and a football and a hockey field on either side. The Stadium with its beautiful pavilion has cost the Corporation of Madras Rs. 7 Lakhs. Madras is the first city in the whole of our country to start Corporation Maternity Services and Midday Meals for the poor Corporation School Children. We are proud to have a first class museum and a good Zoo.

Among the temples of antiquity Sri Parthasarathi Temple at Triplicane, Sri Kapaliswarar Koil at Mylapore, and Sri Thyagaraya Temple at Thiruvottiyur, and the famous Samadhi of the great Saint Pattinathar, are holy places to visit. Among the Churches St. Thome Cathedral at Mylapore, said to have been the first Church built in the city, and where St. Francis Xavier had stayed and prayed, and St. Mary's Church at Fort St. George are well worth a visit. The Walaja Mosque in Triplicane built by the Carnatic rulers is another great place of worship to visit.

The Gandhi Mantap at the Raj Bhavan, Guindy, is another great place of veneration to any visitor to the city. Mahabalipuram, which is about 40 miles from our City, wherein the rock temples of Pallava period dating 600 A.D. to 800 A.D. are situated is a historical place worth visiting. The latest addition—the Museum within the Fort St. George, is another place of interest to visit. I have mentioned only a few places of interest which have been recently developed. Many of you who have not recently visited Madras will be struck with the improvements fast developing.

Coming to the Conference proper, I wish to reiterate that the Indian Medical Association is not a trade Union of doctors, we are bound by the Oath of Hippocrates and our sole aim is to relieve human sufferings and to extend our service to the nooks and corners of our State. Consequent to such an aim it is imperative that the economic condition of the Doctors who are workers in the field must be suitably raised. It is not the intention of Indian Medical Association to embarass the finances of our Government when after mature deliberations they have suggested improved and uniform scales of pay for servicemen attached to the hospitals and a different and independent cadre for those who are in the teaching institutions on an All-India Basis. They have made further suggestions to take part-time officers on hour-basis. The time-honoured but antiquated method of posting servicemen for all jobs disregarding the utility and the services of the honorary system, must stop. The crux of the whole scheme depends on a new order, namely, the burden of work must be shifted to a great extent to honorary and part-time officers from the full-time servicemen. Thus true economy may be achieved and at the same time the Medical Services can be expanded without additional financial burden on the Government. In metropolitan towns where the majority of hospitals are situated, a number of specialists and general practitioners are available. Government must pave the way to induce them into their service either as honoraries or on honorarium basis.

The success of the scheme entirely depends upon the Government's attitude towards the honorary system, which must be considered as the first best

and not as second best. Many senior servicemen will be available to help the upgrading district hospitals, to enlarge them, to equip them with all facilities available in the city hospitals and to form centres for extension of medical services to rural parts. This will also effectively check the ever increasing flow of patients from rural areas to urban hospitals, which, I am afraid is the main cause of overcrowding in the City Hospitals.

Modern medicine and its practitioners are indeed costly. I need not go into the reasons as they are well known to you all. The hope of our government to make the medical treatment cheap and to extend it as widely as possible is indeed praiseworthy. The solution for this does not lie in producing medical men with sub-standard qualifications or establishing sub-standard Medical Schools against the advice of the Indian Medical Council and the University Authorities. We must realise that the health of the people is the wealth of the nation. Modern system of treatment can only be made cheap by large-scale subsidies by the Government. Recently the Honourable Minister for Public Health had assured in the Legislative Assembly that more facilities will be given to general practitioners to attract them to rural areas. It is indeed a positive measure. But I am afraid this will not meet the situation adequately. Government must be more liberal. The question of finances will now arise. May I take the liberty to suggest that as we are collecting library cess on the property tax, an equal amount may be collected for health services by the State. I am making this suggestion fully realising the allergic condition of our people to further taxation. You will all agree with me that health services are more important than running libraries.

There is another measure which is in practice in western countries which prevents the aggregation of medical men in urban areas without any interference in their constitutional rights. In all big cities, places for practice for general practitioners are permanently fixed taking into consideration places of practitioners already practising, convenience of the people of the locality and other relevant factors. A new graduate coming from the portals of the Medical College who desires to set-up as a general practitioner is left with three alternatives. He can either buy up a practice or become a partner to an older practitioner already practising or must migrate to rural areas for his livelihood. This measure has a dual advantage of (1) creating equitable distribution of doctors in course of time and (2) rendering pecuniary assistance to old and ailing practitioners who are unable to work, and to widows and children of medical men who are left unprovided. Believe me, Sir, that except for a handful of medical men the majority's income is such that it is impossible for them to make a provision either for their old age or for their children.

Again in the matter of Employees' State Insurance Corporation, the Corporation itself has declared that the panel system introduced by them in some cities, especially in Coimbatore, the home town of our president elect, is successful. I feel no reason why the same system is not introduced in other places, particularly Madras City, where the service system still persists. This service system has got only negative qualities to commend itself. It cuts down the right of every citizen to choose his own doctor. Secondly, it destroys doctor-and-patient relationship which is

otherwise called "Faith", and which plays a great part in curing at least 50 per cent of the diseases, and lastly it has made some "Haves" medical men into "Have-nots". It is really a violation of our democratic principles and I am constrained to say that our Government has not learnt to carry with them the people and their co-operation, and have yet to realise the bulk of such work must be done by the people, the Government reserving to themselves the task of supervision and direction.

We are happy to hear the Honourable Minister for Public Health has been thinking to change the Integrated College of Medicine at Madras to that of a Modern Medicine. I take this opportunity to remind the Honourable Minister that the Indian Medical Council recently suggested in no uncertain terms that there should be one standard of Medical Education, *i.e.*, M.B.B.S., throughout India, and if Indigenous system of Medicine and Homeopathy are taught, they must be taught as post-graduate studies. I request the Government to respect their suggestions lest we go down hill. To revive our Indigenous system of Medicine and to bring it in line with the scientific system of Medicine, a workable knowledge of this system must be taught in all medical colleges to rouse the curiosity of the students and to show them the greatness of such teachers as Agnivesa, Charaka, Sushruta and Vagbhata. I dream of a day when our ancient medicine brought to the scientific level will astonish the world and reverse the flow of science from East to West.

Sir, while we are pushing the five-year programme, which we all consider as very essential for our national economy and regeneration. I cannot understand why our country should feel shy to utilise the energies of our youngmen for the very same purpose. I mean compulsory national service in other words, compulsory Bharat Sevak Sangh. I am well aware this compulsion offends the fundamental rights in our constitution. I believe this is a serious omission and as our constitution is made elastic the ruling party, *i.e.*, the Congress which is in majority both at the Centre and almost in all States can easily rectify this omission. To those who consider such a procedure an impossibility I can only answer with the famous words of our father of the nation, Gandhiji, who, when his critics told him that by non-violence and satyagraha no nation in known or unknown history had won freedom, replied, "To believe that what has not occurred in history, will not occur at all is to argue disbelief in the dignity of man." This national service will serve a double purpose. It will help our economy, will train and regenerate our people to become good and useful citizens and rouse their patriotism. It is common knowledge that our masses are still apathetic in their love and pride of their country.

Lastly, I wish to draw the attention of our Government that the co-operation of the hospital authorities with the general practitioners is not all that could be desired. We firmly believe such a co-operation is very essential in the interest of ailing humanity. Sir, I trust I may be forgiven for the daring and bold suggestions I have made. But, however presumptuous they might appear to be, you will all agree with me that some such serious steps must be taken to meet the situation that is facing us. I have placed these suggestions before you and the President

Elect for consideration with the fervent hope that they may atleast create thought-provoking reactions. I request the Honourable Minister for Pubilc Health not to consider them in the nature of a catalogue of grievances but as healthy and constructive suggestions. We assure the Honourable Minister for Public Health our full co-operation and support and our request is that you will take us with you by creating Advisory Boards consisting of Members of independent profession to aid you in the arduous tasks facing you. As the Indian Medical Association is the sole representative of medical men throughout our country your task is made easier. In democratic countries the portfolio of Public Health is generally given to non-Medical Minister, as we have in our State, with the idea that the Honourable Minister will come into the field without any biased or preconceived ideas and will have the double advantage of consulting not only Government machinery but also independent Advisory Boards. I sometimes dream, I hope my dream will not come true, considering the present state of our Government, democracy has failed and imperceptibly and silently totalitarianism is creeping on us. God save us from such a calamity and may our Bapuji whose spirit must be watching us, come to our aid and succour and give us saner counsels.

JAI HIND

Dr. T. V. Sivanandam, President, Madras State Branch I.M.A., President of the Conference.

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PRESIDENTIAL ADDRESS

Dr. T. V. SIVANANDAM, M.B.B.S., M.L.C.

Your Excellency the Governor of Madras,
Hon'ble Minister for Medicine & Public Health,
President, Indian Medical Association,
Comrades in Profession,
Ladies and Gentlemen,

At the outset permit me to take this opportunity to thank the One Thousand Six Hundred members of the Eighteen branches of the Indian Medical Association in our State for having elected me unanimously to the office of the President of the Madras State Branch of the Indian Medical Association for this year. I feel it a signal honour done to me by the medical men of our State. I am conscious of the responsibilities of this highest post of our State Medical Association. I assure you of my humble services and that of our two Vice-Presidents, Dr. A. G. Leelakrishnan of Coimbatore and Dr. S. R. Rajaram of Salem, to the Indian Medical Association, the only organisation which can proudly claim to have the majority section of medical men on its rolls, to raise the status of medical men and to keep up the standard of medical education. I request you to co-operate and co-ordinate with all my endeavours in discharging my duties as the President of the Madras State Branch of I. M. A. I appeal to all medical men who are not members of the I. M. A. to join our folds and make the Association stronger and stronger.

Our homage :

It was only five years ago that the State Medical Conference was held in this capital city of Madras State. This year the Madras Medical Association has taken the unique privilege of conducting the 13th Madras State Medical Conference. At this Conference it is part of our duty to pay homage to all those members of our Association who were snatched away by the icy hand of Death during the year and we greatly miss the presence of the following members of our Association :

1. Dr. H. J. Masilamani of Madurai.
2. ,, Mrs. C. G. Herbert of Ootacamund.
3. ,, A. Ramachandra Acharya of Madras.
4. ,, Y. G. Henry of Nagapattinam.
5. ,, S. V. Sankaran of Palani.
6. ,, A. V. S. Sarma of Madras.
7. ,, S. V. Subbiah of Alwartirunagari.
8. ,, G. D. Dawson of Nagercoil.
9. ,, N. Shanmughan of Nagalapuram.
10. ,, V. Iravatham of Madras.
11. ,, (Lt. Col.) C. R. Krishnaswami of Madras.
12. ,, M. R. Guruswami Mudaliar of Madras.

I will be failing in my duty if I do not make a special mention of that great soul, born clinician, a perfect physician, a keen educationist, a true saint, a thoughtful philosopher, a sincere guide and valiant fighter Dr. M. R. Guruswami Mudaliar. Thousands have been benefited through him throughout the length and breadth of our country. His death has created a vacuum which cannot be filled for several years to come. It has been proposed by Dr. M. R. Guruswami Mudaliar's Commemoration Committee to establish a Medical Library of the highest order in the Madras Medical College wherein the eminent doctor had spent most part of his life. It is only befitting on my part to appeal to all medical men assembled here, the philanthropic citizens of this great city, the Madras State and the Country, to contribute as liberally as possible for establishing this library. I also request the Madras Government to come forward to erect a memento to perpetuate the memory of the late Dr. M. R. Guruswami Mudaliar in the most fitting manner in recognition of the sincere services the doctor had rendered to the community at large.

Ourselves :

Ladies and Gentlemen ! We the medical men are passing through the critical stage of struggling for due recognition. At this sputnik age and space period every non-medical man has begun to think in the scientific way, and they even go a step further to criticise and talk on medical problems of the day too. In my opinion it would be better if these non-medical men do not make inroads into the technical and skilled science like Medicine and Public Health. It has become a fashion of late of some to lay stress, to define and to decide the fate of medical science and the problem of medical education. It is needless for me to point out that the medical men can solve medical and public health problems with ease and efficiency for the benefit of the common man. The I. M. A., the State and District branches will always be at the disposal of the Government to give their assistance and help to solve amicably the matters concerning medicine and public health. We request the Government to recognise our existence. Often times we hear the non-medical men talking aloud on platforms declaring the code of our approach towards the sick and suffering. We gratefully thank them for their advice and admonitions and at the same breath may I request them also to observe the same code in their own walks of life ! Let not these remarks go with the familiar phraseology "Don't do as I do, but do as I ask you to do." The same code of "smiling face, the kind manners, sympathetic talks and gentle touch" can and should be practised by all. When and if it is practised in the true spirit it will really raise up the highest standard of human approach in the country and will prevent the unnecessary confusion and misunderstanding in the community at large.

Service Personnel :

The medical men in Government service are not paid adequately for the noble and strenuous work they do. The disparity in the scales of pay of medical men in the Government service and the Local Boards' service must be removed. The Government must see that the medical men are well placed in life with adequate emoluments so that they can meet their barest requirements to keep the family running smoothly. The medical men in service must be paid amply and debarred from having any private practice. The person who teaches non-clinical subjects

must be paid more. Once they are allowed private practice, the justice to their duties will be very much impaired. The temptation to the private practice will be such that some will be forced to resign from Government service. It is a pity that the Gcvernment is still keeping deaf ears to the request of the medical men to raise their status to that of similar posts in other departments. It will be surprising to note that the pay of a Gazetted Civil Assistant Surgeon is far below that of a non-gazetted post in the Madras Secretariat Service. The loyalty of medical men can be very well appraised by their non-agitation, co-operation during epidemics, absence of strikes and sense of duty at the troublous occasions. May I appeal to the Government to look into this vital problem sympathetically and give us the benefit at an early date ?

Honorary System :

The Madras State should be proud of having the Honorary System of Medical Services working efficiently in the City Hospitals and at the District Hospitals. The Honorary Medical Officers are doing splendid work and they are an asset to the Government. Madras Government is a pioneer to start the Honorary System of Medical Services and today they can lead the way to other States. May I request the Government to take advantage of the situation and see that the Honorary System is expanded and minimum number of paid officers are posted at district levels ?

District Hospital :

The District Hospitals are overcrowded. We are in the days of specialisation. The special departments at the district hospitals must be upgraded. The new buildings that are being constructed must have sufficient foundation so as to add more storeys for future expansion, as the ground space available is very much limited in most of the towns. Every district hospital must be provided with an X-Ray diagnostic set, with a deep X-Ray plant also. Some of the district hospitals are not having sufficiently equipped clinical laboratories. Most of the district hospitals and dispensaries are not supplied with adequate stocks of medicine and antibiotics to meet the day-to-day demand. An electric laundry is an essential necessity in each district hospital. It is gratifying to note that every Head Quarters Hospital is provided with a well equipped Blood Bank which is of immense use to the public. I am of opinion that it would be economic to entrust the construction of the buildings to private enterprise, which has proved to be less costly and quicker in completing the construction.

In view of the recognition of the Employees State Insurance Scheme it would be better for the labourers as well as the Government to have a separate labour hospital built at every district head-quarters so that a common man can have easy access to the civil hospitals for investigation and treatment. The chronic and disabled patients have become a problem to the society. Hence separate hospitals to take care of these unfortunates must be opened at the district level too.

The advisory bodies to the district hospitals must be properly constituted with persons who really take interest in the welfare of the institutions. The members of the advisory board and the local politicians should not interfere with the day-to-day administration and routine work of the District Medical Officers which are becoming more often a hindrance than an assistance.

District Medical Officer :

The District Medical Officers are expected to do, in addition to their professional duties, other duties such as attending the Block Advisory Committee meetings, the meeting of the District Development Council and other committees in which the role that is expected to be played by them is very little, but they have to spend considerable time in such meetings unnecessarily. The main duty of the District Medical Officers now is to make elaborate enquiries on the charges levelled upon medical personnel on professional misconduct, corruption and negligence of duty which are mostly frivolous and which are invariably of anonymous nature. The Government can very well establish a cadre of senior medical men in service to be personal assistants to D.M.Os. to look after such matters and allow the D.M.Os. to do their professional duties which are in essential need to the sick and suffering.

Nursing System :

The introduction of male nursing system has brought down not only the efficiency of general nursing but also the good and efficient nursing of female nurses. Only the ladies are gifted with qualities that are essential for good nursing. The gentle and sympathetic look, the kind words, pleasing manners and the soothing touch of a female nurse cannot be achieved by a male nurse however much he honestly attempts. I am confident that the persons who advocate Male Nursing System will not be happy to be nursed by male nurses when they happen to fall sick! Male nurses can very well fit in the places of nursing in male venereal departments, mental hospitals etc., wherein the gentleness of the female nurses will be at the verge of disturbance. I am glad to hear that the Madras Government has temporarily suspended the recruitment of men to the nursing profession. In all fitness of things it will be better if the Government permanently prohibit men applying for nurses' training. The present set of male nurses are persons of good calibre and response. They can be given some more training in preventive medicine and minor surgery and posted as Health Assistants in the Primary and Rural Dispensaries where they can discharge their duties creditably. This will certainly solve to a certain extent the problem of rural medical relief. When this is done a qualified medical man with a mobile medical team can do more efficient work by his periodic regular visits to such centres.

E. S. I. S. :

The Panel System of Employees State Insurance Scheme is working very satisfactorily in Coimbatore. In spite of the good compliments that are being given to the working of the panel system in Coimbatore in our State and in some places in other States also, I am unable to understand the attitude of the Government embracing the Service System even in the areas where Panel System could be started. The introduction of Service System in E.S.I.S. cuts at the root of the fundamental basis of the E.S.I. Act that gives opportunities for the labourer to choose his own doctor. I trust the scheme will be extended to the families of the labourers at an early date.

Medical Education :

It is an accepted fact that we require more medical men in our country. I am glad to hear that the new medical college for which the foundation was laid by the

President of the Indian Union at Tanjore will come up within a couple of years. Our colleges should not be mere factories to manufacture men and women with medical degrees but they must turn out medical men with clinical knowledge to tackle the problems independently when they are called upon to do so. This can be achieved only when qualified and well trained teachers are appointed, lest the standard of medical men coming out of the colleges will go far below the average. The Government must send at least one dozen medical men with good credentials to foreign countries for higher studies every year either taking advantage of the Colombo Plan or Technical Assistance Exchange Programme to acquire the knowledge of recent advances in every branch of medicine and public health. Teachers must be abreast with recent advances; then only the taught will have proper stamina to march with the times.

Rural Dispensary :

It is often said there is dearth of medical personnel to be in charge of our hospitals and dispensaries. Have we taken a survey as to how many medical men are available in our State ? How many of our men have gone to other States ? What are the reasons for this acute shortage of men ? It is evident that there is no proper encouragement from Government and the Government Pay of medical men is not commensurate with the volume of responsible duties they are expected to discharge. In private practice one can earn more than the service salary without any departmental bossings. Often times non-medical men who occupy the helm of affairs ask us to go to villages to work in the rural areas. Have the Government provided the medical personnel with the basic requirements in the villages ? No. The doctor, to his utter despair, finds no house to live in. He tries to get a transfer or deliberately resigns from the job to better his prospects.

Primary Health Centres :

We have seen several primary health centres being opened by the initial enthusiasm of local leaders and we have also seen that those centres are closed or not functioning for several months and even years. Before the primary health centres are inaugurated by the responsible men at the top, they must at least be satisfied to see whether the dispensary has been fully equipped and whether the personnel are available to run the show. We hear that several primary health centres are closed either for want of personnel, medicine or buildings. The doctor must be paid at least one hundred rupees extra as long as he serves in the primary health centres. Proportionately the special allowances to the other medical personnel also must be increased. This will be just an attraction for the medical men to take up the job. Our medical men are trained only on the curative aspect of the diseases. But the medical men in the primary health centres are expected to do much of the preventive aspect of the diseases also. Hence to do an efficient service these doctors who are posted at the primary health centres should be given a short term post-graduate training in the public health aspect of the diseases also ; otherwise the purpose for which the primary health centres are opened becomes nullified.

Integrated System of Medicine :

It is very recently the ministers of our State and one section of the members of the legislature took the novel method of " Pada Yatra " which was never practised

in the history of our State before, in their respective constituencies, to have a closer contact with the people in the villages and to have a first hand knowledge of the things which the villagers require. Villagers did ask for opening of dispensaries of modern medicine only. Hence the panacea to open dispensaries of indigenous system need not be thrust on them.

The huge amount of money that is being spent by the Government every year on the College of Integrated Medicine is, in my opinion, a colossal waste. The graduates coming out of this college prefer to practise allopathic system of medicine rather than the other special systems taught to them. Thanks to the Ministry of our State, an Act has been passed recognising their qualification as a registrable one. This kind of so-called Integrated Medicine will certainly lead to natural demise of indigenous system of medicine. If we really want the indigenous system of medicine to survive and flourish, the indigenous system of medicine must be taught as a post-graduate study to the persons who have obtained the basic qualification in the modern scientific medicine.

We often see the Graduates of the Integrated Medicine have got more faith in the modern broad spectrum antibiotics and hormonal therapy than their own Choornams and Lehyams and Thailams. Hence they occupy a strange position in the country. They are neither recognised by allopathic practitioners nor claimed by other systems of practitioners. Hence it is high time we stop this eye-wash and make this College of Integrated Medicine a full blown Medical College of Modern Scientific Medicine and as a post-graduate and a research institution for the study of indigenous system of medicine.

Family Planning :

One of the several ways by which we have to solve the Socio-Economic problems of our country is 'Family Planning'. Enthusiastic State Government has gone through a long way in establishing Family Planning Centres at every District Head-quarters and doing effective propaganda among lay people about the necessity of birth control and reliable methods of birth control. A separate department has also been created. I hear, as usual, our Madras State is showing the lead, as in many other important matters, to other States in the activities of this department also. I admit that the Family Planning is very essential for the healthy socio-economic progress of our country. It is evident that only poor classes, low income and under-privileged section of the human species require Family Planning, and limitation of the family. Though the figures given by the department are showing encouraging results yet we invariably find that those figures mainly pertain to rich people and highly placed middle class persons. The propaganda of family limitation must be taken to proper quarters wherein solution of the socio-economic problem of our country lies. The best method of family limitation must be the one on Gandhian principle of abstinence or adhering to the time-honoured rules and regulations of religious sentiments. For the successful solving of this problem the family limitation must be based on religion, character and morality; and the scheme when implemented on these lines in quarters where it is essential is bound to prove a great success. For proper dissemination of the objects of the Family Planning propaganda and to achieve the desired end of family limitation,

we non-official voluntary social welfare organisations of women and the members of I.M.A. can successfully play our role and thus solve the socio-economic problem of our country in a great way in this direction.

Life Saving Drugs :

The medical profession is very badly in need of life saving drugs like Morphia and allied products. We are very much handicapped to give relief to the sick and suffering on account of its non-availability in the market. Permits for the import licences are given to merchants who utilise the foreign exchange to import other products which are easily marketable and yield good profits. Hence I request the Government to take immediate steps to issue import permits to medical men, pharmacists and chemists. In the interests of the sick and suffering is it not desirable and also dangerous to limit the supply of the life saving drugs?

Medical Inspection :

The health of our children and youths is very much neglected during their school and college days. They are susceptible to evil habits and diseases. May I request the Government to make the medical inspection in schools and colleges compulsory ? Proper and thorough examination and mass X-Ray photographic examination of the students must be done to diagnose latent diseases. This will prevent many promising students from invalidation at a time when they complete their studies to take up their responsibilities in the community.

D. M. S. & D. P. H. :

No one who is aware of the duties and responsibilities of the Director of Medical Services and the Director of Public Health will ever think of integration of these posts. To the medical personnel people go to get relief of their complaints and for the cure of the diseases. But on the other hand the Public Health personnel go to the common man to take them into their confidence to work with their co-operation and prevent the spread of diseases. Hence fundamental differences exist in both their education, method of working and the manner of the discharge of their duties.

Public Health Museum :

Preventive Medicine is very essential for the healthy progress of any country. The common man must be aware of the cause and spread of infectious and contagious diseases and how to prevent the spread of the epidemics. The best way to achieve this aim is to establish a Public Health Museum in every district head-quarters. The Public Health Museum will not only be instructive but it will also be an educative and impressive one to the lay public. May I appeal to our Government to take up this venture to establish a Public Health Museum in every district head-quarters and become a model to other States ?

There has been a proposal to establish Regional Public Health Laboratories but in some places it is yet to be started. It would be better if the Government speed up the matter so that this laboratory can solve Public Health requirements quickly.

Industrial Medicine—D. O. H. S. :

Our State is rapidly getting industrialised. Several new industries are coming up. With the advent of new industries we must be prepared to face very many industrial diseases and accidents. Occupational diseases, environmental hygiene and social psychology are coming into lime-light. Ere long we have got to recognise the new diseases. I would like the Government with the advance in the industrial development of our State to create a separate department of Director of Occupational Health Services to tackle the prevention, spread and treatment of occupational diseases. In view of the increase in industrial accidents, I appeal to the Government to open an Orthopaedic Unit with trained personnel and good equipment in every district head-quarters hospital to tackle the industrial surgery. I request the Madras Government to show the way to the other States of our country not only to keep the departments of Director of Medical Services and Director of Public Health as separate entities but also by creating another separate department, namely the Director of Occupational Health Services. The existence of these three separate departments will result in efficient work, proper control and good understanding of the various branches of work to which they are responsible.

Medicoes :

Since there are many medicoes assembled here let me open my sincere heart to them. We belong to a noble profession. We must raise the prestige and honour of this noble profession. Our community and country expects us to do our duty sincerely and honestly. Hence you must equip yourself well to deal with the life and death of the sick entrusted to your care. Spare no pains to achieve the high standard of clinical knowledge. Keep abreast with the recent advances in medical science. Be a well disciplined soldier to fight for the preventive and curative aspect of the diseases. Your character and morality must be firm, strong and above suspicion. You must discharge your duties to the satisfaction of your conscience, and the fruits of your endeavours will come to your feet without your asking. I wish you all good luck and success and I dare say that the future health of our country lies in the sincerity, efficiency and the loyalty with which you shoulder your responsibilities. I appeal to all the medicoes that when they graduate themselves and go out of the college they should make it a point to enrol themselves as members of the Indian Medical Association, which is the only Association of its kind representing the medical profession, and thus add more strength to the Association in its efforts to solve the problems confronting the profession.

Ladies and Gentlemen ! I am conscious that I have taken much of your time. Let me conclude with my humble prayer to God and remind you the philosophy of life, the spirit of which we very often forget :

" If wealth is lost nothing is lost
If honour is lost something is lost
If health is lost everything is lost."

JAI HIND

MESSAGES

HIS EXCELLENCY BISNURAM MEDHI
Governor of Madras

I am glad to know that the 13th Madras State Medical Conference 1958 will be held at Madras on November, 29, 30 and 1st December, 1958, and that a Souvenir will be published on the occasion.

A conference of this kind will give an opportunity to the members of medical profession to come together and exchange notes on the various aspects of the diseases, preventive and curative, and the methods to be adopted for effectively controlling the diseases.

I take this opportunity of conveying my best wishes for the success of the Conference.

* * * * *

THE HON'BLE SRI M. A. MANICKAVELU
Minister for Revenue and Health, Government of Madras

I am glad that the State Medical Conference is to be held at Madras at the end of this month. I hope that as a result of the deliberations in the Conference, suitable resolutions will be forthcoming towards better medical relief.

* * * * *

THE HON'BLE DR. K. K. HEGDE, M.B.B.S., M.L.A.
Minister for Health, Government of Mysore

I am glad the 13th Madras State Medical Conference, 1958 will be held at Madras from the 29th November, 1958. I send my hearty felicitations and warm greetings on this happy occasion to all members of the Indian Medical Association, Madras City Branch, and all those who will be attending the Conference. Your Branch of the Medical Association has been doing very useful work in furthering the interests of the profession and in alleviating the suffering humanity. I am sure that, important and interesting subjects will be discussed by eminent persons of the profession at your Conference. I wish the Conference a grand success.

Dr. P. V. CHERIAN

Chairman, Madras Legislative Council

I am glad to hear that the 13th Madras State Medical Conference is being held in Madras and will be inaugurated by our popular Governor Shri Bisnuram Medhi. Medical Conferences are extremely helpful in bringing our brothers and sisters together from different parts of the State so that they may get to know each other better and also profit by the scientific papers and exhibitions.

I wish the function every success.

* * * * *

Dr. U. KRISHNA RAU

Speaker, Madras Legislative Assembly

I am glad to learn that the 13th Madras State Medical Conference, 1958, will be held at Madras. There are many problems which vitally affect the medical profession in our State. There is also the burning question of solving the medical needs of the villages. The integration of the Allopathic and Ayurvedic systems of medicine is another topic of paramount importance. I do hope that this Conference will contribute a lot to the solution of these very important questions.

You have my good wishes.

* * * * *

Dr. V. R. THAYUMANASWAMI, M.S., F.R.C.S., Mch. Orth.

Director of Medical Services

Thank you for your invitation to the 13th Madras State Medical Conference, 1958, to be held in Madras from 29th November to 1st December, 1958.

I am sure the Conference, as usual, will serve a very useful purpose and bring together a large number of Medical men; and I hope their deliberations will contribute to the success and implementation of the various Health Schemes which we are all eager to implement in our State.

I wish the Conference all success.

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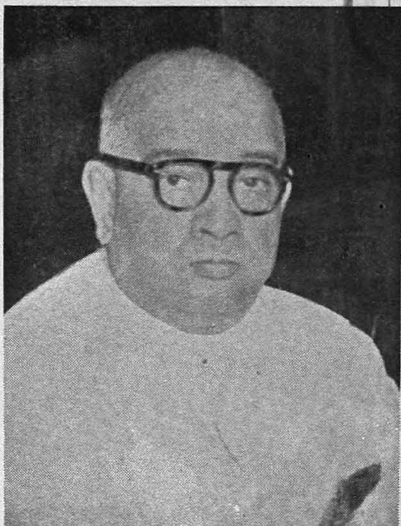
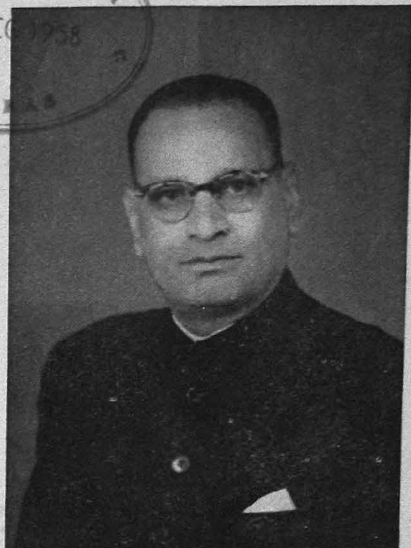
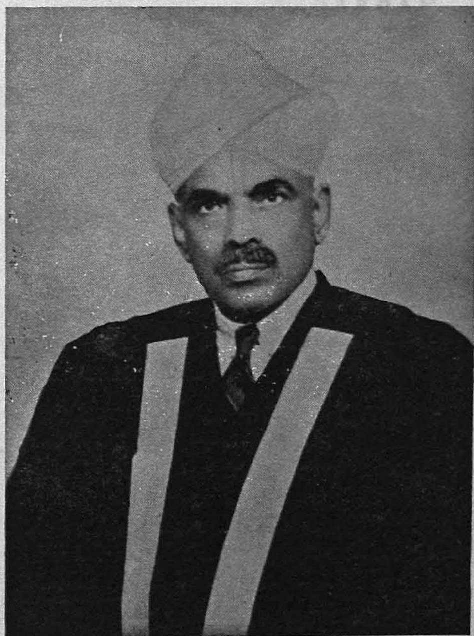


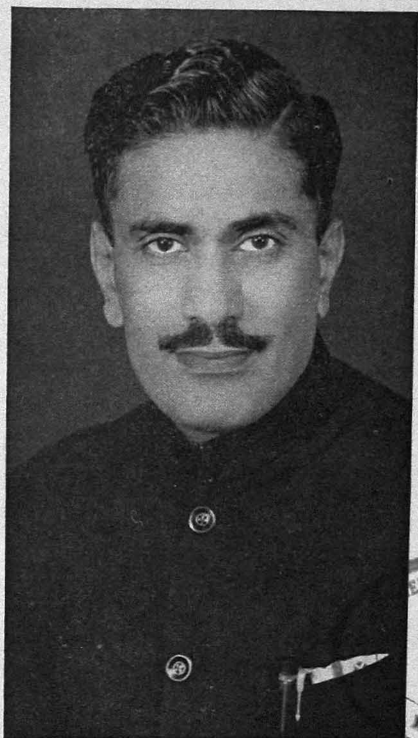
Dr. P. V. Cherian, Chairman, Madras Legislative Council opens the Exhibition.



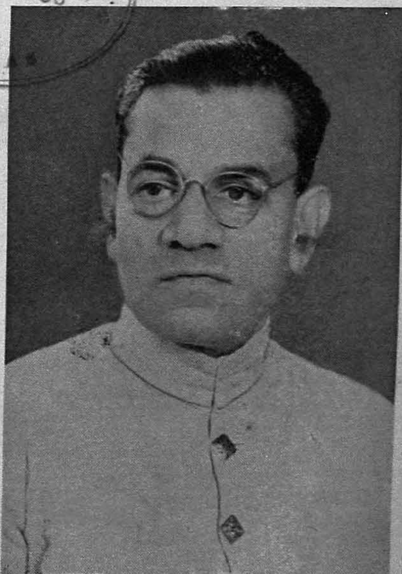
Dr. V. R. Thayumanaswamy, Director of Medical Services, Govt. of Madras presides over Scientific Session.



Dr. A. Lakshmanaswamy Mudaliar, Vice-Chancellor, University of Madras inaugurates the Scientific Session.



Dr. A. G. Leela Krishnan
Vice-President (1958-59)
Madras State Branch.



Dr. S. R. Rajaram
Vice-President (1958-59)
Madras State Branch.

Dr. C. Arumugam, Hony. Secretary, Madras State Branch.

Dr. V. Vijayaraghavan, Hony. Joint Secretary, Madras State Branch.

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Dr. C. N. Santhanam, Hony. Treasurer, Madras State Branch.

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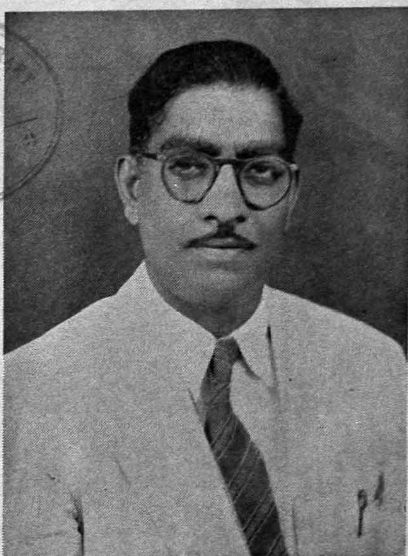
Dr. B. Rama Rau
Hony. Secretary.

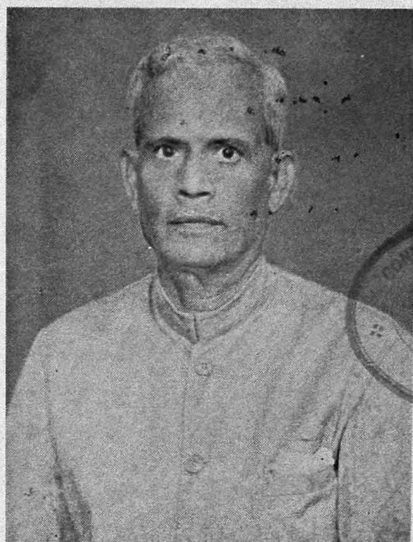


Dr. G. Ramachandra Murthy
Hony. Secretary.

Dr. P. Krishnaswamy
Hony. Treasurer.

Dr. T. Janardhanan
Vice-Principal, Madras Medical College,
Vice-Chairman, Reception Committee.

VICE-CHAIRMEN, RECEPTION COMMITTEE



Dr. K. Narayanamurthy.

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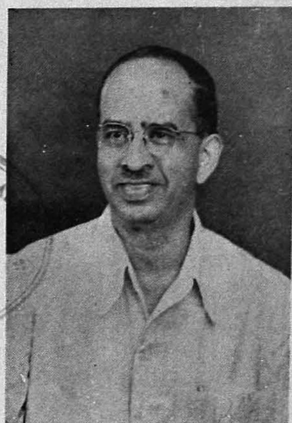
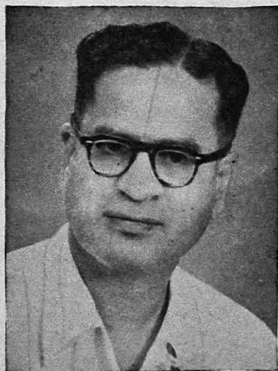


Dr. U. L. Narayana Rau



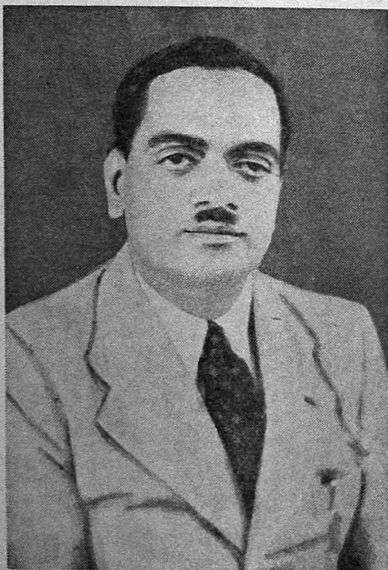
Dr. B. V. Sundara Babu

Dr. V. Samuel

Dr. B. Ramamurthi
Convener—Scientific Section.

Dr. A. M. Sivaraman
Convener—Exhibition.

Dr. Capt. G. Sriramulu
Convener—Souvenir.



Dr. K. Rama Rao
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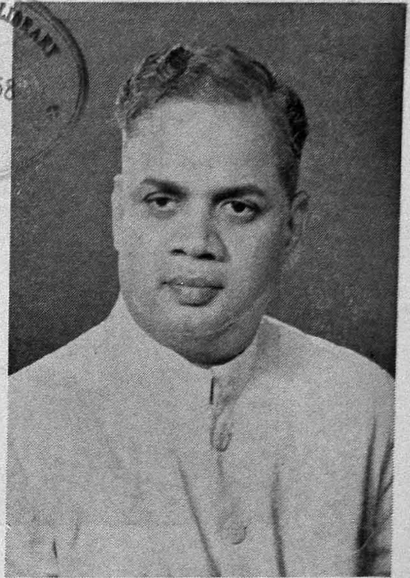

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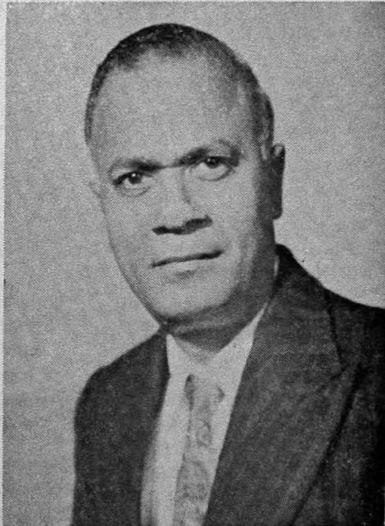

Dr. A. V. Avadhani
Convener-Entertainment

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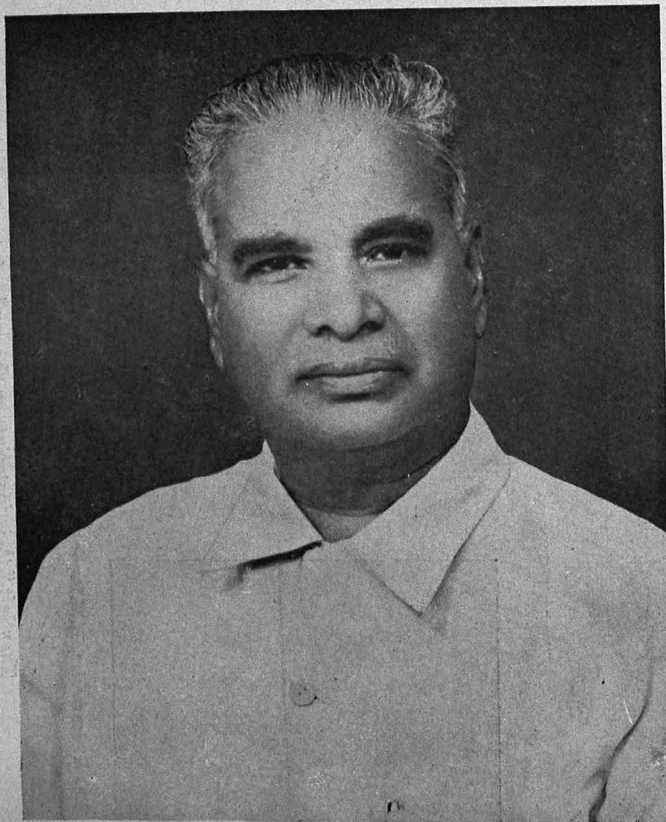
Dr. C. Nathamuni Naidu
Elected—President of Madras Medical Council

Dr. R. V. Rajam, M.B., M.S., F.R.C.P.
Elected—Fellow of the Royal Society of Edinborough



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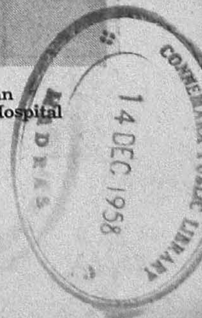
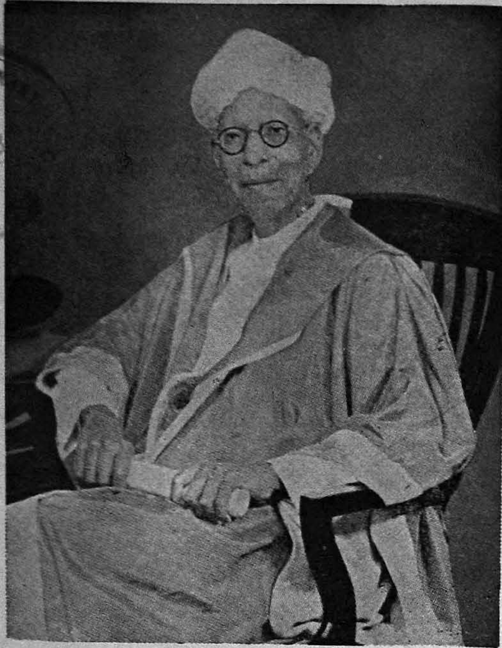




Late Dr. M. R. Guruswamy Mudaliar, M.D., C.M.

Late Dr. V. Iravatham, L.M.P.

Late Dr. Lt.-Col. C. R. Krishnaswamy, M.S.

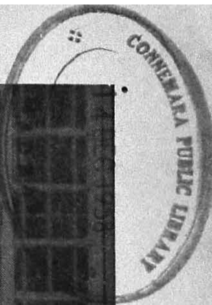
They left " their footprints on the sands of time "

DR. N. PARTHASARATHY, L.M. & S., B.S.SC.
Director of Public Health, Government of Madras

I am happy to hear that the 13th State Medical Conference would be held during the last week of November '58 in Madras. I feel that this is an opportune moment for the meeting of the State Medical Association. Madras State has launched on ambitious mass campaigns like Malaria Eradication, Filaria Control and B.C.G. Campaign etc. in order to bring these major public health problems under control within a comparatively short period. The co-operation of such an influential body of eminent medical men of the State Medical Association would be quite invaluable to the control programmes mentioned above. I have no doubt that such co-operation would be forthcoming in an ample measure in the future as in the past.

I wish the Conference all success.

OFFICE-BEARERS & MEMBERS OF WORKING COMMITTEE OF RECEPTION COMMITTEE



Sitting :—Drs. B. Rama Rao, T. Janardhanam, U. L. Narayana Rao, P. Alagasingari Naidu, M. Santhosham, V. Samuel, V. Vijiaraghavan.

Standing Front Row:—Drs. A. V. Avadhani, K. V. Swamy, K. Rama Rao, C. Rama Rao, K. L. Narayana Rau, A. M. Sivaraman, G. Ramachandra Murthy, P. Krishnaswamy.

Standing Second Row :—Drs. G. Sriramulu, A. Pattabhi, C. Ranganathan, V. S. Selvapathy and A. G. Arumugam.

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PART II

PROBLEMS AND EXPERIENCES IN THE SURGICAL TREATMENT OF TETRALOGY OF FALLOT* †

REEVE H. BETTS, M.D., and N. GOPINATH, M.B., B.S., F.A.C.S., T.D.D.

The surgical treatment of congenital cyanotic heart disease is based on the epochal work of Blalock and Taussig.[1] Approximately 75% of such cyanotic cardiac lesions are due to the combination of abnormalities known as the tetralogy of Fallot or a close variant of it. Numerous large series of cases have been reported from other countries, but there is a paucity of reports in the Indian literature of series from this country.[2] Although our group of surgically treated cases is not large, 47, and of comparative recent date, only seven prior to 1955, it seems worthwhile to record our experiences and some of the problems we have encountered in this modest experience.

DIAGNOSIS

It is providential that the large majority of patients with congenital cyanotic heart disease have the tetralogy of Fallot and that in most instances these cases can be diagnosed without recourse to the more cumbersome and less readily available procedures of angiocardiography, or cardiac catheterization with pressure studies and blood gas analysis. There will always be a border-line group where all the diagnostic facilities will be necessary, but these more complicated tests are not indicated in the majority. Both angiocardiography and cardiac catheterization carry a small but definite risk in the severely cyanotic child. They should be used by all means when there are definite indications but not as strictly routine diagnostic aids.

A history of cyanosis from birth or early infancy, delayed development, limited exercise tolerence and squatting or other similar postures are most suggestive of the tetralogy of Fallot. Episodes of unconsciousness for a few moments, especially after exertion such as crying, are indicative of a severe degree of cerebral anoxia. A normal size heart on physical examination with usually a slight thrill and a systolic murmur maximal in the third or fourth intercostal space to the left of the sternum are to be expected. Examination of the blood reveals an increased hemoglobin content, sometimes over 20 grams, and the red blood cell count is elevated in some instances to 8 or 10 million per cubic millimeter in the more severe cases.

Radiologically the heart is not grossly enlarged, but the characteristic "boot shape" due to the enlarged right ventricle and diminished shadow of the pulmonary conus is found. The vascularity of the lung fields is also markedly decreased in most cases.

* From the Department of Thoracic Surgery, Christian Medical College Hospital, Vellore, South India.

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The aortic knuckle is well seen as there is a relative enlargement of the aorta in these cases. In approximately 20% to 25% of patients there is a right aortic arch and a right descending aorta. It is imperative that the side on which the arch descends be determined by a barium swallow before operation as it influences the side on which the patient is operated upon.

A patient with tricuspid atresia and hypoplastic right ventricle may present much the same picture as a typical tetralogy of Fallot. The absence of any heart border to the right of the vertebral column in the anteroposterior chest X-ray and a left axis instead of a right axis deviation on the electrocardiogram indicate the presence of this condition. Differentiation should be made from the tetralogy of Fallot as the prognosis is not quite as good in tricuspid atresia although the same operative procedures are of benefit.

It is imperative that one differentiate tetralogy of Fallot from pulmonary stenosis without interventricular septal defect. There is usually no confusion unless the patient has an accompanying auricular septal defect with a reversed, or right to left, shunt. These patients have pulmonary stenosis on cardiac catheterization and may be mildly cyanotic. However, they are not benefited by a shunt type of operation like the tetralogy of Fallot patients as this further increases the load of the overworked right ventricle without producing any benefit. Most of these so-called "pure" pulmonic stenotic cases where there is an intact interventricular septum will have a pulmonary valvular stenosis and the post-stenotic dilatation of the pulmonary artery beyond the obstruction shows up in contrast to the small size of the pulmonary artery in the tetralogy of Fallot.

Other lesions with which the tetralogy of Fallot may be confused, such as the Eisenmenger's syndrome, auricular septal defect, ventricular septal defect or truncus arteriosus, are usually distinguished from the tetralogy of Fallot by the differences on physical examination and X-ray. When there is doubt, cardiac catheterization or angiocardiography or both may be necessary.

If a patient has a history of cyanosis from infancy, delayed development, squatting, an essentially normal sized heart, a systolic thrill and murmur in the left third or fourth intercostal space and a "couer en sabot" shaped heart on X-ray with normal or diminished pulmonary vascularity and the electrocardiogram shows right axis deviation, it is inadvisable, in our opinion, to carry out either a cardiac catheterization or an angiocardiography. When some of these cardinal factors are absent or when other signs or symptoms are added, one may need to exhaust the various diagnostic facilities to substantiate the diagnosis. Usually we have found cardiac catheterization with blood gas analysis to be far more helpful than an angiocardiogram. The pressure readings in the right ventricle and the pulmonary artery are the best means of establishing a diagnosis of pulmonary stenosis. Sometimes the cardiac catheter can be passed into the overriding aorta, but more often it cannot and the diagnosis of ventricular septal defect is made on the absence of any

signs of an auricular septal defect by the pressure readings and sometimes by the slight arterialization of the blood in the right ventricle. A right descending aorta is suggestive but not pathognomonic of the tetralogy of Fallot rather than a "pure" pulmonic stenosis with auricular septal defect. It is found in approximately 20% of the tetralogy cases (found in six of our cases) but only rarely in the so-called pure case of pulmonary stenosis.

TREATMENT

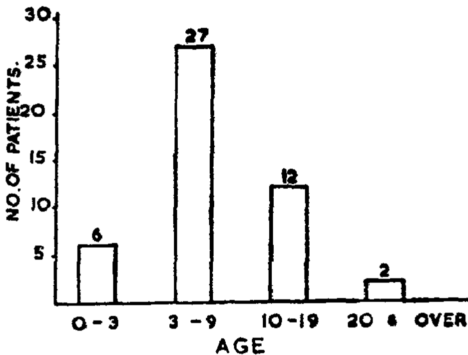
Although an occasional case of tetralogy of Fallot will compensate and be able to lead a fairly normal life over a reasonable life span, most of them are apt to become worse as growth and activity add to the demand on the handicapped right ventricle. It is our opinion that most all cases of tetralogy of Fallot should be helped surgically. When symptoms are more severe and especially if there are periods of anoxic syncope, operation should not be delayed. The ages of our operative cases have varied from two months to 22 years (Table 1 and Fig. 1). In those instances where there was no dangerous increase in red blood cell count or hemoglobin concentration and the patient was under four years of age, we have had the patient return every six months for examination, with operation being delayed until they have reached a more suitable age. From four to ten would seem to be the optimum age for surgical treatment.

TABLE-I

SHUNT OPERATIONS

	NO	FATALITIES	MORTALITY
PATIENTS LESS THAN 3 YRS	6	3	50%
PATIENTS 3 YRS AND OVER	41	3	73%

FIG. I



This chart demonstrates the number of cases in each age group who had shunt operations The six patients operated on under the age of three were severe cases that we thought could not safely be allowed to go on to a more suitable age.

We have a strong preference for the Blalock-Taussig type of surgical procedure. However, we prefer to go in on the side of the aortic arch rather than the opposite as advocated by Blalock in order that a Potts-Smith aorto-pulmonary artery anastomosis can be done should the subclavian-pulmonary artery anastomosis not be feasible. Our early experience was with the Potts type of operation, but the risk of making the anastomosis too large and causing congestive heart failure is a real one and we now prefer the Blalock operation where the size of the anastomosis is limited to that of the subclavian artery.

Our first four operations were the Potts-Smith type and since then we have used this procedure in three other instances, one being a child of two years where the aortic arch seemed placed very high and the subclavian not long enough for a Blalock.

In the other two instances it was thought that the subclavian was too small for an adequate shunt. In one of these the aorto-pulmonary shunt which was constructed was made too large and the patient developed congestive heart failure soon after discharge and later died as will be mentioned below. Although we do not think that the type of operation was at all responsible, the two operative deaths we have had since 1953 both followed a Potts type of operation. We would still use the Potts procedure if confronted with a subclavian artery too small for adequate anastomosis or an arch of the aorta abnormally high making a Blalock operation impractical.

In most of our earlier cases of the Blalock operation we used the subclavian artery after it had given off its first group of branches as we thought that the angle between the subclavian and aorta would be so acute as to prevent proper functioning if we divided the subclavian proximal to its divisions. This appears to be an erroneous supposition in most instances and we now try to use the subclavian before it branches, whenever possible. The aorta and its main branches are usually comparatively enlarged in a tetralogy of Fallot and hence the main subclavian is of good size and will give an adequate anastomotic flow. In those cases where a division was used there has been improvement but not as striking as when the main subclavian before it divides was employed and a larger shunt established. We routinely mobilize the inferior pulmonary ligament to allow the lung to ride up in the hemithorax and it is kept in this position during the operation by placing sponges between the lung and the diaphragm. In some instances we have also sutured the perihilar tissues of the pulmonary hilum to the aortic adventitia to further relieve any tension on the anastomotic site.

We have had no experience in the direct attack on the pulmonary stenosis in the tetralogy of Fallot as advocated by Brock[3] and Baily[4]. It would seem a quite logical procedure in those with valvular stenosis but we believe with Lillehei[5] that if one is to make a direct surgical attack on the stenosis one should close the ventricular septal defect at the same time. Such a procedure entails an open cardiotomy with a heart-lung machine of some type and the mortality in the best of hands is still far higher than in the shunt type of operation. If one relieves the stenosis only without closing the ventricular septal defect, the patient may become worse due to the large left to right shunt that then develops after complete relief of the pulmonic obstruction. No doubt the direct approach will be the adopted procedure in the future, but at present we are content with the results in the shunt operation until the other procedures become standardized.

For the past two years we have been using a mild hypothermia in all our cyanotic heart disease cases. We believe that lowering the body temperature moderately diminishes the risk of cerebral anoxia without materially increasing the risk from cardiac irregularities. We put the patient on the hypothermia blanket as soon as anesthesia has been induced and by the time we are ready to do the actual anastomosis the rectal temperature,

which is measured by a thermocouple, will have dropped to about 35°C (95°F). The tank is then warmed to 43°C (110°F) with the pump turned off and warm water is circulated through the blanket as soon as the anastomosis has been completed. Even with hot water running through the blanket the temperature will usually drift down another two or three degrees before it starts to rise. We like to keep the patient in the operating room until the temperature has started to come up or at least has stopped falling and is stabilized before returning the patient to the ward. With this mild degree of hypothermia we have had no cardiac irregularities. In some instances when the temperature was as low as 32°C (89°F) it has not been possible for the anesthetist to record the pulse or the blood pressure even though the cardiac action seemed quite good on direct inspection. This is a common finding in hypothermia and seems due to the intense peripheral vaso-constriction which involves even the larger arteries when the blood in them is cooled sufficiently.

There has been one serious complication in our series that we have not seen reported elsewhere in the literature and that is the loss of the left arm from ligation of the subclavian artery distal to its primary branches. In several instances in the literature[4] reference is made to the advisability of dividing the subclavian before the branching takes place in order to preserve some collateral circulation in the arm. This is not always possible and still have sufficient length to do a good anastomosis. In one of our patients (G.35446) a 15 year old boy, a Blalock procedure was carried out on the left side and it was deemed necessary to divide the subclavian beyond the first branches. He went through the operative procedure well, but his left arm remained cold and blue after operation and he was unable to move it. In spite of giving papaverine and keeping the arm cool by packing in ice, it was soon obvious that the arm was non-viable. By this time, about 48 hours after operation, he also had a rather marked lower nephron nephrosis syndrome. It was deemed imperative that any toxic absorption from the arm be prevented. So a tight tourniquet was placed around the arm and after he recouped from the lower-nephron nephrosis the arm was amputated. He eventually made a fine recovery as far as his cardiovascular status is concerned and his exercise tolerence is greatly improved. He is badly handicapped however by the loss of this left arm, but fortunately is right-handed. We have not encountered any report in the literature of such a complication before and we have been informed by Taussig[6] that they have not encountered it in the large series of cases at the Johns Hopkins Hospital. In most every instance it is advisable to divide the subclavian before it branches and use the larger vessel for anastomosis. However, there are quite a number of patients in whom the anatomical arrangement is such that anastomosis is possible only by going beyond the divisions. In such instances care should be exercised to see that the circulation in the arm is encouraged to develop and the arm kept cool until circulation is adequate. Several other patients have had temporary pain or inability to move the arm well for a short time,

but these have all improved rapidly after the first few days.

VARIATIONS

As mentioned above, tricuspid atresia with hypoplastic right ventricle and auricular septal defect has certain characteristics in common with the tetralogy of Fallot, but it can be differentiated. This is one other type of congenital cyanotic lesion that is also benefited by a shunt-type of operation. We had one such patient who was correctly diagnosed preoperatively by our cardiologist, Dr. K. I. Vytilingam. The patient was operated on and has considerably improved but not as dramatically as most of the tetralogy of Fallot patients. This is in agreement with other reports.

Everyone recognizes that a patient with one congenital anomaly is apt to have others. We have encountered two anomalies that are of interest, both associated with right descending aortas which have been found in six of our cases. In one instance (I.42132) a girl of 8 years was brought to us with a typical history of tetralogy of Fallot This was substantiated by the findings at cardiac catheterization and on angiocardiography. There was difficulty in establishing the location of the descending aorta, but it was thought that she had a left descending arch. At operation a curious vascular anomaly was found. There was a right descending aorta instead of a left, but the left subclavian artery arose directly from the pulmonary artery through the patent ductus arteriosus. A segment of this artery was resected in order to force more blood into the pulmonary circuit. As the patient had a right descending aorta and this anomalous left subclavian artery, it was not possible to do any shunt-type of operation through the left chest. Unfortunately, definite recording of the blood pressure in both arms was not done preoperatively. (It is now routine !). The patient was clinically improved after operation and we plan to do a shunt-type of operation on the right side should it seem warranted in the future. This patient taught us several things and had resulted in a "check list" type of study chart for all congenital heart cases so that such possible findings will not be overlooked in future.

Another case (R.38754) a ten year old boy was diagnosed as a case of tetralogy of Fallot and it was found preoperatively that he had a right descending aorta. Operation was done through the left chest intending to use the left subclavian after its origin from the left innominate. At operation there was no left innominate and the subclavian arose from the right descending aorta but came around to the left behind the esophagus from lower down. The vascular ring around the esophagus and trachea was completed by the obliterated ductus arteriosus which was markedly stretched out and attenuated. It was divided before the anastomosis was carried out. The patient was very markedly improved by the Blalock type of operation.

ASSOCIATED PULMONARY TUBERCULOSIS

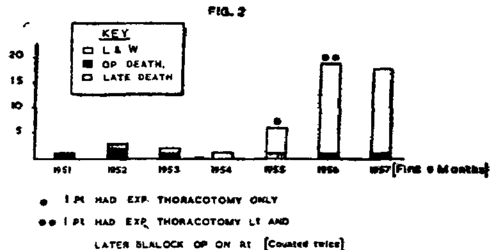
In a locality where the incidence of pulmonary tuberculosis is comparatively high one would expect to encounter a number of cases where pulmonary tuberculosis and the tetralogy of Fallot coexist. We have now

had three such patients. In one (A.43463) the diagnosis of tuberculosis was not made preoperatively although, in retrospect, one can see some abnormal shadows in the left upper zone in the preoperative X-ray of the chest. At operation a moderate tuberculous infiltration of the left upper lobe was found. The Blalock operation was completed and the patient had an uneventful convalescence with antituberculous chemotherapy being instituted in early postoperative period. In a second case the patient (G.43151) a man of 19 years came to the hospital because of hemoptysis of recent origin. He was markedly cyanotic. Examination revealed moderately advanced tuberculosis of the right upper lobe with cavitation and further studies including cardiac catheterization showed the cardiac lesion to be a tetralogy of Fallot. As his exercise tolerence was fair in spite of rather intense cyanosis, we thought that the tuberculous lesion should be treated first. After three months of chemotherapy the right upper lobe was resected. His postoperative course was a bit worrying for the first few days as he developed a partial hemiparesis and facial weakness with inability to talk for 24 to 36 hours. This was presumably due to a spasm or thrombosis of one of the cerebral vessels. With papaverine and other symptomatic treatment the lesion cleared completely. The patient has been advised to return in six months for a Blalock operation on the left side. A third patient (L. 45536) was a 24 year old girl who complained of hemoptysis. Cyahosis had been noted since early childhood and the first hemoptysis had been thirteen years previously at the age of 11. X-ray examination showed a moderately advanced bilateral pulmonary tuberculosis with cavitation. Investigation including cardiac catheterization demonstrated a tetralogy of Fallot. She is now taking chemotherapy and a decision will be reached later as to when the cardiac lesion should be operated upon.

Another recent cyanotic patient was found to have active bilateral pulmonary tuberculosis, but his cardiac lesion has been proved to be an auricular septal defect with a right to left shunt. He is now in a sanatorium being treated for the tuberculosis.

RESULTS

It is only to be expected that the early results of an operative procedure will not be as good as the later ones as experience with the operation should improve the technic. Also in the early stages only the more desperate cases are selected for operation. This has been true in our experience as illustrated in Fig. 2.



This chart demonstrates the number of patients and indicates the operative and late mortality by years. It is only since the beginning of 1955 that any sizable number of patients have been treated. Of the 18 patients operated in the first nine months of 1957, as shown on the chart, one patient (referred to in the text) did not have a shunt operation but only a resection of an abnormal subclavian artery

During the years 1951-1953 only six cases were operated upon. Three of these died within 48 hours of operation. A fourth lived just over two weeks and at postmortem was found to have a complete transposition of the great vessels with other anomalies including a large porencephalic cyst. A fifth survived over a year although she had a hemiplegia after operation. She later died of subacute bacterial endocarditis. The other case, the only long-term survivor in this group, was operated on by Dr. Erik Husfeldt when visiting on a W.H.O. team. This very dark picture has improved materially in the latter period. In the last 41 cases who have had shunt operations there have been two operative deaths and one late death (Table 3). Postmortem examination

TABLE 2

SHUNT OPERATIONS

	NO. OF PATIENTS	OP. DEATHS	LATE DEATHS
POTTS OPERATION	7	5	2
BLALOCK OPERATION	40	1	0
TOTAL	47	6	2

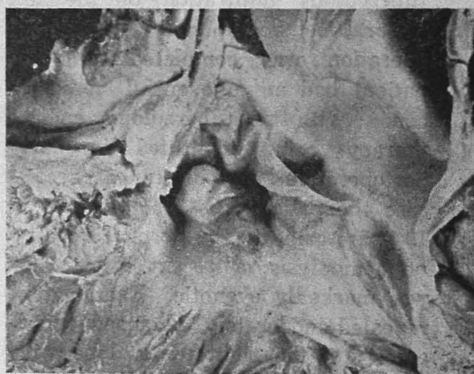
TABLE 3

SHUNT OPERATIONS

	BLALOCK OPERATIONS	BLALOCK FATALATIES		POTTS OPERATIONS	POTTS FATALATIES		TOTAL	TOTAL FATALATIES	
		EARLY	LATE		EARLY	LATE		EARLY	LATE
1951-1953	2	1	0	4	3	1	6	4	1
1954-1957 (9 m.)	38	0	0	3	2	1	41	2	1
TOTAL	40	1	0	7	5	2	47	6	2

was obtained on both the operative fatalities. In one instance (V.39612) it was found that the patient did not have a tetralogy of Fallot but an extremely marked infundibular pulmonic stenosis with an auricular septal defect and must have had a right to left shunt to account for the marked degree of cyanosis. Although we knew that there were certain elements against the tetralogy of Fallot diagnosis, it was thought that the child had this lesion even after an angiocardiogram and cardiac catheterization. We believe that with further experience we would not make this same error in diagnosis again. The other operative death (S.40368) was a two year old boy with evidence of marked right heart strain on the electrocardiogram. The patient died about 24 hours postoperative and the postmortem examination revealed an additional valve cusp below the aortic valve (Fig. 3) which quite obviously would balloon out and markedly obstruct the outflow tract to the aorta. We believe this was the cause of death in this patient.

FIG. 3



Postmortem photograph of patient S.40308. One can see the extra valve placed beneath the aortic valve which must have markedly obstructed the outflow tract.

The one late death in the last 41 cases was in a patient on whom we had done a Pott's operation (P.27491) because of a high placed aorta. On

check up three months after operation she was found to have signs of congestive heart failure. This was undoubtedly due to the anastomosis being too large. Hospitalization was advised but not heeded and we have been informed that death took place not long afterwards. It is well recognized that the one big drawback in the Pott's operation is the tendency to make the anastomosis too large and the heart goes into right-sided failure. In the Blalock operation the size of the anastomosis is limited by the size of the subclavian artery.

In most series of patients the mortality rate for patients in the younger age group is higher than for those that are older. This is mainly due to the fact that most of very young patients are done at an early age because of the severity of symptoms and therefore they are a more seriously ill group. Where possible, everyone prefers that the patients be at least four years of age as the arteries are a little easier to handle when the patients are of this age or older. When the severity of symptoms is such that operation has to be done earlier, then one can expect a higher mortality rate. Gross[7] has reported that in the last three years of their series ending 1st January 1953, there were 135 patients with a total mortality of 11%. For patients under 3 years of age, however, the mortality was 30% and for those over three years of age it was 7.1%. This same finding has been reflected in our results (Table 1) although our series is much smaller. We have had six who were operated upon under three years of age with three fatalities, a mortality rate of 50%, whereas in the three or over age group there are 41 with three deaths or a mortality rate of 7.3%. Whenever possible we prefer to wait until the patients are in the older age group, but when symptoms are severe, and especially in the presence of periods of anoxic syncope, operation should be done without delay. The greater part of the increased risk in these young patients is due to the severity of the lesion rather than to the age itself.

One patient had only an exploratory thoracotomy as he was found to have a valvular pulmonic stenosis and at that time, which was early in our experience, we thought it would be advisable to go in later and do a direct valvotomy. The operation was therefore terminated and the patient was advised to return at a later date, but has not done so.

Another patient (D.29485) was operated on twice. When he was first explored through the right chest it was found that there was an abnormal branching of the vessels from the arch of the aorta. Both the left and the right carotid as well as the right subclavian came off a large innominate, but the branching of the right subclavian was high up in the apex of the chest and the subclavian could not be brought down for a subclavian pulmonary anastomosis on this side. As he had a left descending aorta nothing else could be done through the right thorax. He was again operated on (D.31270) through the left chest and a Blalock procedure carried out without difficulty.

Of the 47 cases in this series we have done a Potts-Smith operation on 7 and Blalock-Taussig operation on 40 (Tables 2 and 3). Although we do not think that the type of operation

was the decisive factor, there has only been one operative death and no late deaths in the group that had the Blalock operation. Thus the mortality rate is an acceptable 2.5%. None of our Pott's type operative cases is alive at present, there being five operative deaths and two late deaths in the seven patients so operated upon. (This is another example of the fallacy of attaching too much significance to figures alone).

SUMMARY

Our experience in surgical treatment of patients with the tetralogy of Fallot has been reported. Forty-seven shunt-type operations were carried out on patients varying in age from under two months to twenty-two years.

During the period 1951-53 inclusive, there were six operations, but only one of the patients still survives.

From 1954 to 1 October 1957 there have been 44 operations on 41 patients. One patient had only an exploratory thoracotomy and another had an exploratory thoracotomy on the right side and a Blalock operation at a later date on the left. A third had a division of a subclavian artery arising from the ductus arteriosus. Of the 41 patients having shunt operations there were three who had the Potts type and 38 the Blalock variety. The two operative deaths and the one late death in the group since 1954 were all following Potts operations although we think that the operative procedure was not responsible except in the one late death from congestive failure. The two operative deaths were due to an error in diagnosis in one instance and an abnormal additional valvular cusp in the second.

We believe that the moderate hypothermia (not below 32°C. 89°F.) used in the past two years has been a beneficial factor.

It is substantiated that in spite of all efforts and diagnostic tests it is sometimes most difficult to be absolutely positive about the diagnosis preoperatively, especially in the younger age group.

Our experience is like that of others in that patients who survive the immediate postoperative period continue to do well and are greatly improved by a systemic-pulmonary shunt operation. We believe that a direct attack on the pulmonary stenosis and the ventricular septal defect is not justified until improved methods have been developed.

BIBLIOGRAPHY

1. Blalock, A., and Taussig, H.B.: Surgical Treatment of Malformations of the Heart in which there is Pulmonary Stenosis or Pulmonary Atresia, J.A.M.A. 128: 189, 1945.

2. (a) Sen, P.K.: Experiences with the First One Hundred cases of Surgery of the Heart and the Great Vessels, Ind J. Surg. 18: 425, 1956.

(b) Betts, R H., Thomas, T., and Gopinath, N.: Experiences in the Surgical correction of Cardiovascular Abnormalities, Ind. J. Surg. 19: 145, 1957.

3. Brock, R C: Surgery of Pulmonary Stenosis. B.M.J. 2: 399, 1949.

4. Bailey, C.P.: Surgery of the Heart, Philadelphia, Lea and Febiger, 1955.

5. Lillehei, C W, Cohen. M., Wardon, H.E., Read, R.C., Aust, J.B., DeWall, R.A. and Varco, R.L.: Direct Vision Intracardiac Surgical Correction of the Tetralogy of Fallot, Pentalogy of Fallot and Pulmonary Atresia Defects, Ann. Surg. 142: 418, 1955.

6 Taussig, H B.: Personal Communication, January 1957.

7. Gross, R E.: Surgery of Infancy and Childhood, Philadelphia, Saunders, 1953.

DIABETES MELLITUS

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Diabetes has been with us for centuries. Classical descriptions of the disease are seen in the earliest recorded medical writings. In India, even the sweet taste of the urine was recognised by Charaka, in the second century A.D.; however it was only in 1776, Dobson demonstrated the presence of sugar in the urine and Cullen added the name 'Mellitus' to the then existing name of "Diabetes". This sweetening of the name did nothing to sweeten the nature of the disease. Diabetes continued to be a dreaded disease till the early years of this century.

With the dawn of this century, the first rays of hope came. Today there is a resurgence of hope in the treatment of diabetes such as was experienced in 1889, when the cause of diabetes was located in the pancreas; in 1924, when the lives of diabetics were prolonged by the therapeutic utilisation of under-nutrition; again in 1921, more strikingly with the discovery of insulin, and today, by the introduction of oral anti-diabetics. Now assurance is added to hope because we know that control of diabetes is possible and worthwhile and that the future of the diabetics is far far brighter than it ever was.

In this paper, it is intended to present some facts about the existing concepts of the pathogenesis and treatment as mentioned in the literature, as well as our experience from a study of the cases attending the diabetic clinic in the Stanley Hospital.

Our knowledge of the Pathologic physiology of the disease stems from Minkowski and Von Merring's experiment in 1889, who produced diabetes by removing the pancreas from a dog. The internal secretion of the pancreas was isolated in 1921, by Banting and Best. At this time the etiological mysteries were thought to have been unravelled. The key to the problem was held to be a primary insulin lack. But Nature yields her secrets grudgingly, and it did not take long to realise that the key opens only the ante-chamber and the insulin lack is only one aspect of the problem. Observations soon accumulated to show the complexity of the problem. The degenerative changes in the B-cells which can be demonstrated histologically are entirely absent in 20 or 30% of diabetics. Even complete removal of the pancreas, can produce only a mild type of diabetes. The influence of pituitary was shown by Houssay and that of the adrenals by Long and Lukens.

Factors which are generally accepted to be of importance are the following :

Heredity : Surveys by Joslin in America and Lawrence in England indicate that 30 to 40% of diabetics

have a family history of this disease.

Obesity: Diabetes is described as the fat man's penalty. Probably continued stress on the islets by over-eating and obesity exhausts them.

Age: No age is exempt, but the disease is commoner in the late middle life.

Sex: After 40 years the incidence is greater in the females than in the males.

Pancreas has naturally held the stage in the pathology of diabetes. The B-cells secrete insulin and diabetes mellitus is due to a deficiency of this hormone. This insufficiency may be brought about by impaired insulin production or due to excessive production of substances which have an anti-insulin activity.

Insulin acts in a number of points in the metabolic chain:

1. It may increase the rate of entry of glucose into the cells by altering the permeability of cell membrane.
2. Insulin may facilitate the phosphorylation of glucose within the cell. This process requires an energy source such as Adenosine Triphosphate and an enzyme hexokinase. Once phosphorylation has taken place, glycosis can proceed down to the pyruvate level even in the absence of Insulin.
3. The next site of possible action of Insulin is in the Kreb's cycle which is the final common pathway for the degeneration of carbo-hydrate protein and fat into carbon-di-oxide and water.

Other endocrines are also known to have a hand. Injection of crude anterior pituitary extract or even purified growth hormone alone can produce diabetes. This fits in with the clinical observation that mild diabetes is sometimes seen in hyper-pituitarism. It may explain why diabetic and even potential diabetic women, tend to have large babies. It has been suggested that the growth hormone leads to growth as long as Insulin is available but leads to diabetes when the supply of insulin is inadequate. The view that the growth and the diabetogenic hormones are identical has however been challenged by recent studies.

The well-known action of adrenaline in raising the blood sugar is probably by de-glycogenation of the liver, and perhaps by blockade of peripheral disposal of glucose. The cortical steroids increase the gluconeogenesis from amino acids and fats and at the same time diminish the utilisation of glucose for the formation of tissue glycogen.

Thyroid: Though it facilitates glucose utilisation the more predominant action is depletion of the liver glycogen and increase in gluconeogenesis.

The influence of the gonads is evident from the fact that diabetes may first appear or if already present become aggravated with menopause. The experimental observation that oestrogens in sufficient doses ameliorates the condition also supports it. We are undertaking a special study of patients who come in this group.

I have discussed in some detail the role of the various endocrines. How far they are of importance in clinical

diabetes continues to remain a moot question.

Not only the endocrines but even non-endocrine agents may have possible importance. Glucose given in large amounts as infusion has been shown to produce permanent hyperglycemia with evidence of initial hyperplasia and subsequent degeneration of the islets. The administration of alloxan, destroys the B-cells and produces a diabetic state. Compounds such as uric acid and di-hydro-ascorbic acid which resemble alloxan, may also produce diabetes. Other substances like fluoro-acetate, quinoline, magnesium and chromium salts and glucagon can all produce diabetes.

In summary it can be stated that the complete removal of insulin sources by pancreatectomy or by the use of alloxan or glucose results in a diabetes which is uniformly mild. Those cases of clinical diabetes which require little or no insulin may therefore be the clinical analogues of these types of experimental diabetes. In those instances of spontaneous human diabetes, which require much larger doses of insulin, we have to look for anti-insulin factors. In some cases these may be an excess of growth hormone or the other factors cited earlier. However there is no evidence of such hormonal factors in the diabetic population as a whole. The low correlation of clinical diabetes with hormonal factors makes the hypothesis of increased insulin destruction particularly attractive.

Diagnosis: The striking symptoms of diabetes include loss of weight and strength, in spite of adequate or excessive intake of food, along with polyuria, polydipsia and pruritis. A good percentage of cases present with symptoms of complications. Many patients especially adults may have diabetes mellitus for months or years without giving rise to any of the above symptoms. In such cases diabetes is recognised only when the urine is examined as a routine, for insurance or other purposes. Indeed today's problem is the recognition of patients with early or even latent diabetes before any of the classical symptoms have developed in order that by earlier institution of treatment many later complications may be postponed or entirely prevented. This stresses the importance of routine urine examination and of blood whenever necessary.

The requirements for a diagnosis of diabetes mellitus are glycosuria and hyperglycemia.

The finding of glucose in the urine at any time should be taken as evidence of diabetes until the contrary is proved. Glycosuria may be discovered by any of the standard methods based on the reduction of alkaline copper solutions such as the Benedicts. The reducing substance need not always be glucose. Salicylates, camphor, chloral hydrate, morphine, ascorbic acid and massive doses of Penicillin can all reduce Benedict's solution and when it is remembered that some of these drugs are very commonly used often by the patient himself the importance of excluding this possibility becomes obvious. Among the other sugars like Pentose, Fructose, Lactose, and Galactose, lactose is the one which is clinically important. Whenever in

doubt further tests may be necessary to establish the identity of the reducing substance.

Once we are convinced that the reducing substance is glucose the question naturally arises—'is it a case of true diabetes mellitus or only a non-diabetic glycosuria?' Glucose can appear in the urine under conditions of stress, as in fever, infections, excessive intake as in IV. glucose therapy, in toxaemia, anaesthesia and in rheumatoid arthritis. Under these conditions the amounts are small and temporary. When glucose appears in the urine, following the ingestion of carbohydrate and becomes less on reducing the carbohydrate in the diet, *i.e.*, when a clear correlation of glycosuria with the intake of carbohydrate is found, it is highly suspicious of diabetes. This can be confirmed only by blood sugar studies.

True glycosuria with normal blood sugar can occur, as a result of stimulation of intracranial nerve centres as in brain tumour and cerebral haemorrhage or as a result of hypertension and chronic nephritis. Whenever in doubt or to be safer still in all cases examination of the blood must be done. A clinically probable diagnosis of diabetes mellitus can be confirmed by examining the blood and urine for sugar, one hour after a meal. The present practice of estimating the fasting blood sugar and examining the early morning urine is not quite satisfactory. In fact in our cases a majority are sugar free in the early morning and their diabetes would be missed entirely if this specimen of urine alone were examined. While doing the glucose tolerance test it is important that patients in whom this test is being done should have received a high carbohydrate diet for three days preceding the day of the test and no insulin for the same period.

Another classical differentiation is from renal glycosuria. It is rare but is benign and requires no treatment and hence it is essential that this condition should be differentiated from true diabetes mellitus. There are a few who believe that this condition is a precursor of diabetes mellitus but this view is not accepted by most authorities. In this condition sugar appears in all the urine specimens but the blood sugar is normal or even low at times. This condition is due to a lowered renal threshold and blood sugar studies are the only means of differentiation.

Interest naturally centres around that group of persons who may be designated as potential diabetics; the fasting blood sugar level is normal but it may go above the upper normal limits one hour after food and sugar appears in the urine. It has been shown that 10 to 15% of these people ultimately developed frank diabetes, such people must be closely watched for the earliest evidence of diabetes —and for them the greatest hopes of treatment are justified.

Along with testing of the specimen for sugar, ketone bodies must be looked for whenever necessary. Gerrhardt's ferric chloride test and Rothera's nitro-prusside test may also be used to detect the ketosis.

In no chronic disease is repeated examination of patients at regular intervals of greater importance than in the case of diabetes. Many of the complications can be discovered in their incipiency in a stage where

successful therapy may prevent serious and long disabling illness.

TREATMENT

The objectives in the management of the diabetic patient may be summarised as follows:

1. Correlation of the disordered metabolism by means of diet, insulin or other drugs;
2. Attainment and maintenance of normal nutrition with body weight approaching the ideal for the age, the height and the sex;
3. Preservation of the remaining insulin producing power of the pancreas; and
4. Prevention, postponement or treatment of complications and sequelae of the disease.

The physician should not only regulate the patient's diabetes and teach methods of maintaining this regulation but also he must sense the very real fears of the diabetic, concerning the impending dangers in the daily routine and what appears to be an uncertain future. Re-assurance represents an important bulwark of the support that the physician can give to the patient, but only when tempered with simple carefully taught facts about diabetes is it effective.

Methods of treatment: None of the major tools in the treatment of diabetes is singly effective. But together their intelligent and continued use will allow the objectives to be reached. As pointed out already the education of the patient is one of prime importance. Perhaps in no other disease is it so much desirable or so much advantageous to let the patient know as much about the disease as possible. The doctor should re-assure the patient that the diabetic symptoms will be removed; that daily activity and occupation need not bê curtailed or altered; that a diabetic diet of today is not one that in most instances a normal expectancy of life is to be anticipated and that infections, injuries, surgical operations and in women, pregnancy are without risk.

Diet: The principles of the diabetic diet are simple but the details of its administration often give rise to difficulties.

Diabetes mellitus primarily a disease of insulin deficiency is accompanied by defects in the metabolism of all the three major nutrients. It follows that treatment must take this fundamental fact into consideration and the total calories whether of carbohydrate, protein or fat origin are to be regulated if diet therapy is to succeed. The needs of the diabetic are identical with those of the normal person. They are influenced by the same factors, age, sex, body weight, occupation, rate of growth and exercise. All authorities agree that obesity should be corrected. The majority of diabetics will do best on 25 calories per Kg. of ideal body weight for height and age. Older patients especially women may not get better until intake is limited 15 calories per Kgm. Similarly under-weight or juvenile patients may be given as much as 35 calories per Kg. of ideal body weight. The diet may be distributed as follows—about 40% of the total calories to be taken as carbohydrates; about 1 gm. of protein per Kg. of body weight and the rest as fat. In addition the diet must contain ample supplements of vitamins. In a country

like ours where the standard of living is very low, it might not be possible for every patient to be put on a diet which is calculated in terms of weights and measures. Another popular notion is that changing over to wheat diet, is sufficient to control diabetes. It is very common to meet patients complaining that they are not alright, in spite of being on a strict diet, not having touched rice for a long time. But on enquiry we find out that they are taking wheat for their principal meals and the tiffins and the total calorific value is atleast 2 to 3 times their absolute requirements. It is to be noted that it is the quantity and not merely the quality that has to be adhered to.

In our clinic it has been our practice to put the patient on a very low calory diet for 2 to 3 days the diet mainly consisting of buttermilk and greens. The urine is examined daily and with this diet most of them become sugar free. At the end of three days the minimum diet for the patient was roughly calculated and according to the dietary habits of the patients a diet was prescribed taking care that nutritive foods like milk, egg or meat find a place wherever possible. If the patient on this diet showed sugar in the urine he is started on insulin.

Here it is important to stress that all patients do not require insulin. It is always safer to treat obese patients by diet alone. Such patients are usually over middle age, and have mild diabetic symptoms or no symptoms. If such patients adhere to a diet of low calorific value their symptoms as well as hyperglycemia and glycosuria are usually rapidly controlled. If the weight is substantially reduced such patients are often able to eat a more liberal diet without a return of the diabetic state. Probably amphetamine may help those who want to control the diet, but whose appetite defeats them. Difficulty arises with over-weight patients who fail to lose weight, especially if they have complications. Insulin must be carefully used in them because there is every chance of the weight increasing further.

Insulin : The prime objective in the treatment is the control of all the signs and symptoms of diabetes. It is necessary to avoid hyperglycemia and glycosuria completely. Although arguments have been advanced pro and con, available evidence today favours the maintenance of the diabetic patient's blood chemistry within normal limits. Not only therefore is it desirable that the blood sugar level should be maintained as nearly normal as possible under all conditions but other chemical components of the body tissues such as cholesterol and blood proteins should also be studied. The protection of the insulin producing function of the islets in the pancreas is aided by the use of Insulin. It has been shown that hyperglycemia itself is a major factor in animals in producing degenerative changes in the islet cells and therefore, the basic rule in treatment today is to give sufficient insulin to control the hyperglycemia and thus protect the islets of Langerhans.

Types of Insulin : A number of insulin preparations are available for use but the basic property of each is same. However they differ in composition and the type of effect on the patient.

Crystalline or soluble insulin—action starts within half an hour; maximum action 3 to 4 hours and duration of action 6 to 8 hours. This is the only type of insulin that can be given Intravenously.

P.Z. insulin—action starts after 4 hours. Maximum action 12 to 20 hours and duration of action 24 to 30 hours.

Globin insulin—usually has got an action midway between soluble and protamine zinc insulins.

N.P.H. insulin is a modified form of P.Z. insulin with an action of shorter duration.

Lente Insulins or Insulin zinc suspensions: This is the latest type of insulin in the market and supposed to be the best insulin available today. Insulin lente starts acting in one to two hours, maximum action is 8 hours, and duration of action 24 hours. Its action is much like a mixture of P.Z. Insulin and soluble insulin. Ultralente which is a longer acting one and semilente which is shorter acting are also available. There is no indication to change the regime of patients who are already controlled with a particular type of insulin. Insulin lente is to be tried in patients who are poorly controlled by other insulins or in those who show allergic reaction with other insulins.

Control of Insulin dosage: The correct dose of insulin can only be determined when the patient is at home and doing normal activity. Patient with normal renal threshold can be controlled satisfactorily by urine tests. In this connection it is to be emphasized that the present practice of doing a single urine test or even a blood sugar under the abnormal circumstances of a visit to the doctor or to the diabetic clinic does not give us a correct idea. In many cases it will be necessary to correlate the urine sugar and the blood sugar in order to have an idea of the renal threshold. To judge whether or not, the patient is well adjusted it is usually necessary to have three or four blood and urine sugar determinations within the course of 24 hours. Where this is not possible, urine must be collected in 2 portions one from 8 a.m. to 7 p.m. and the other from 6 p.m. to 8 a.m. After mixing properly both the samples should be tested separately, for the presence of sugar. Traces of sugar in the urine samples are considered to be not only harmless but even advantageous, as they reduce the changes of hypoglycemic shock. Our experience has been that patients who have mild or moderate diabetes do well with P.Z. insulin alone, provided their insulin requirement is not more than 30 to 40 units. However in severe cases requiring larger doses usually P.Z. insulin requires supplementary doses of soluble insulin. In such cases the N.P.H., Globin or preferably Lente Insulin might be useful.

Oral therapy: The latest advance made in the treatment of diabetes is the introduction of oral treatment. There are two preparations at present available. They are carbutamide or B.Z. 55 and Tolbutamide or D.860. These compounds when administered produce a drop in the blood sugar levels. The one feature which seems necessary to bring out this effect is the presence of a sufficient number of islets of Langerhans to produce a certain amount of Insulin.

Mode of action—The effects of the drug seem to be multiple but the major effect seems to be a stimulation of the islets of Langerhans to release Insulin. Other effects include the possibility that the rate of destruction of insulin in the organism is diminished. Since the pathogenesis of diabetes is not definitely proved and since there is no method of finding out whether there are sufficient functioning islets in the individual, caution must be exercised before we decide to treat patients with these drugs. As it stands today, the drug is most likely to be useful in elderly patients with diabetes of short duration, or who had insulin for relatively short periods. The drug should not be tried in juvenile diabetes and in patients with acidosis or other serious complications.

A useful test that can be probably employed for the selection of patients for oral therapy is the *four hour test.* Insulin must be omitted for 48 hours and the patient tested in the fasting state. The fasting blood sugar level is determined. 3 gm. of the drug are given by mouth and the blood sugar determined 4 hours later. In those who show a drop from 20 to 40% or more in that period of time good results can be expected.

Recent experience has shown that carbutamide or B.Z. 55 though quicker in action can produce some toxic effects whereas Tolbutamide or D.860 is comparatively harmless. The latter has been tried by us in about 30 patients. The response is very slow but is satisfactory. Only time will tell, whether these will aid in correcting the fundamental disorders of diabetes in properly selected cases as well, as does Insulin. But one thing is certain, the oral anti-diabetics are not merely a therapeutic advance; they open up great possibilities regarding the elucidation of the further nature of the disease and perhaps the classification of the disease on a pathological basis.

Complications : Diabetic patients are liable to suffer from a number of complications like skin infections, pulmonary tuberculosis, urinary infections, diabetic ketosis and coma, nephropathy, neuropathy, cataract and various forms of arterial degeneration.

Of all these, diabetic coma is the unique complication of diabetes, resulting from insulin deficiency and failure to diabetic control. We shall confine ourselves to the discussion of a few points of importance regarding diabetic coma.

It is necessary to describe in detail the clinical picture of diabetic coma which occurs in the neglected diabetic. We have already mentioned that some present themselves for the first time in coma. The insidious onset with the return of the main symptoms of polyuria and thirst, the dehydration, the mental torpor quickly passing on to coma, the deep hissing respiration and the smell of acetone in breath, are all wellknown as are the main differential diagnosis. What must be stressed here is the importance of spotting the condition in the precomatose stage which will be often possible by the examination of urine for ketone bodies under all suspicious circumstances and the early and energetic treatment before renal failure sets in.

As soon as the diagnosis is established one must give 50 to 100 units of insulin immediately, and proceed to

hospitalise the patient whenever possible. In the presence of circulatory collapse, the same amount may be given I.V. Correction of dehydration must be undertaken immediately by giving normal saline or 1/6th molar lactate solution. Glucose or glucose saline at this stage only aggravates the electrolytic changes and dehydration. The rate and amount of fluid is to be regulated by the severity of the dehydration and the cardiac status of the patient. At the same time steps must be taken to correct any precipitating cause such as infection. Routine stomach wash helps to control the vomiting, relieves the distension of the stomach and prevents possible aspiration pneumonia. Enemata are also helpful.

During the second hour, insulin may be repeated depending on the results of urine examination. At this stage it is necessary to look for potassium deficiency since the vomiting, the polyuria and the acidosis result in significant potassium loss. This can be easily detected by E.C.G. or blood potassium studies. Unfortunately both are not usually available to us in an emergency. Weakness in spite of an improvement in the diabetic state, abdominal distension and cardiac or respiratory irregularities, strongly suggest potassium depletion. In any case it is better to presume a hypokalemia and after making sure the renal functions are adequate as shown by urinary output of more than 1000 c.c., large quantities of orange juice and Pot. citras or chloride 15 grs. thrice a day orally should be given. Occasionally potassium may be given I.V. cautiously, in the form of multiple electrolyte solution.

Frequent examination of the blood sugar level is the ideal for the guidance of further treatment. When this is impracticable hourly urine examination must be done.

As soon as the initial dehydration is corrected, glucose or glucose saline may be administered because once Insulin begins to act the patient may imperceptibly slip into a hypoglycemic coma, for the glucose reserve of the comatose patient is already low. The routine treatment of shock, the prevention of pulmonary infection etc. are carried out as indicated.

Another complication which is coming into prominence is the diabetic nephropathy or Kimmelsteil Wilson syndrome, where the patient develops albuminuria, oedema, and occasionally hypertension. At this stage the examination of the urine shows the sugar to be diminishing but this is due to a rise in the renal threshold and the blood sugar remains high. Hence only the latter can give us a proper idea of the severity of the diabetes. It is probable that really good control of diabetes, prevents or atleast delays the onset of this complication. However, once established, control of diabetes does not prevent its further progressive course.

I have attempted to paint a picture of diabetes, as it stands today. The picture has obviously not very much changed, a little deepening of the lines, here, repainting a little detail there, and perhaps altering the shades slightly in a place or two. But the main features remain the same. The oral anti-diabetics have opened up a new vista of possibility and with them they have brought an aroma of

hope and comfort and the possibility of taking us nearer the solution of the mystery of the pathogenesis of diabetes. But as ever the control of diabetes continues to rest with the patient; but more so with the family physician. It is his duty to (1) advise against the marriage of one diabetic to another (2) to halt the development of obesity with its liability to diabetes particularly in the relatives of the diabetic before the age of 40 is reached and (3) to discover the new cases by examining the urine and blood of the relatives of his diabetic clientele, among whom the incidence of the disease is 5 or 6 times as great as among those of the non-diabetics.

RESPIRATORY DISEASES IN INFANCY AND CHILDHOOD*

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Respiratory diseases constitute the commonest cause of death in infancy and early childhood, taking precedence even over gastro-intestinal disorders. In Great Britain out of 100 deaths in children below 5 years, 36% is due to respiratory tract infection and 20% to Gastro intestinal disorders. One of the main reasons for this is that respiration is an entirely new and important function that child adopts soon after birth. The degree of maturity of the child, and therefore of the respiratory system, determines whether the child is to survive or not. In a premature child, therefore, the respiratory disorders may be either due to imperfect expansion of the lungs or to superadded infection.

Anatomical Peculiarities of the Respiratory Tract in Infancy: An important anatomical feature is the small size of the air passages. As a result of this even a tiny plug of mucus can cause an obstruction to the bronchi, resulting in collapse of a lobe or a segment. Another anatomical peculiarity of importance is the relative large size of the respiratory lymphatics and lymph glands. As a result of this, mediastinal glandular enlargement is frequently observed in respiratory infections in infancy, particularly in tuberculosis. These glands press upon the soft-walled bronchi causing secondary collapse. The chest wall in a child is very soft. Deformities like pigeon and funnel shaped chest, Harrison's sulcus, sucking in of lower part of the chest during an obstruction to the respiratory passage as in diphtheria, therefore, occur early.

Physiological peculiarities of respiration in childhood is noted particularly with the rate. The respiratory rate is over 40 per minute at birth, reaching 25 per minute at the age of 5 years. Respiration in childhood is mostly abdominal in type. It may be stated here that respiration starts in foetal life. Barcroft has proved conclusively that rhythmic respiratory movements take place in utero in the foetus, the foetus inhaling and exhaling the amniotic fluid. This helps to keep the respiratory mucous membrane moist and lubricated, facilitating expansion soon after birth.

Symptoms common in infancy and childhood

1. *Cough*: This is an early and constant symptom noted. The cough may be due to causes in the larynx and pharynx *e.g.*, tonsillitis or due to causes in the lungs. Recurrent cough is seen in bronchitis, tonsillitis or bronchial asthma. A constant cough is

* Based on a lecture delivered before the Madura Branch of the Indian Medical Association.

more in favour of collapse of a segment or a lobe, large tonsils and bronchiectasis. In whooping cough the cough is spasmodic in nature, followed by a loud whoop. Vomiting occurs after a bout of cough. Cough is not an important sign in tuberculous infection of the lungs in childhood.

2. *Expectoration*: It must be remembered that children usually swallow their sputum. In tuberculous infection expectoration may even be completely absent.

3. *Clubbing of fingers* is most common in bronchiectasis. It may also be present in other chronic respiratory tract infections.

4. *Poverty of growth*: Children with chronic lung infections do not thrive well. They appear short and stunted. In addition to this, they may appear thin and emaciated. But it must be remembered that growth of the head and skull is very little affected by malnutrition arising as a result of respiratory infection. Thus, whilst the rest of the body may appear thin and emaciated, the skull maintains its normal circumference. Till one year of life the circumference of the skull and the chest are the same. At the third year of life the thorax is one inch more than the skull. In a child with chronic respiratory infection this increase in the circumference of the chest may not occur, the child appearing to possess an out-sized head on a small shrivelled body.

5. *Stridor* is another important symptom noted in disorders of the respiratory system in childhood. It is a symptom with which every practitioner must be familiar with. It may occur soon after birth. In such a case it is called congenital laryngeal stridor. It is important to diagnose this condition early and to differentiate it from stridors occurring in early life due to other causes e.g., thymic and thyroid enlargement; mediastinal glandular enlargement; diphtheria; laryngismus stridulus (hypocalcaemia); and laryngo-tracheo-bronchitis. A child with congenital laryngeal stridor is healthy. The stridor is inspiratory in character, and occurs more when the child is excited or crying or during feeds. The stridor disappears as the child grows older and does not require any treatment. In stridor due to diphtheria the child appears ill and toxic and a rise in temperature is noted. A membrane may be seen over the fauces. Stridor may also be a symptom noted with mediastinal glandular enlargement. The commonest cause of such enlargement in childhood is tuberculosis. In this condition wasting is a prominent feature. The finding of tubercle bacilli from the gastric lavage or sputum, and a positive tuberculin (Mantoux) test establish the cause of the stridor. Thymic enlargement and a retrosternal goitre should always be borne in mind in any case of congenital stridor. Thymic enlargement may sometimes be associated with urgent dyspnoea and cyanosis-thymic asthma, or with attacks of syncope, pallor and sudden collapse. Diagnosis is established by radiological examination, when a uniformly rounded opacity will be seen in the superior mediastinum. A lateral picture will show the opacity pressing upon the trachea. In later years of life stridor may be due to bronchial asthma. Attacks of bronchial asthma are characterised by expiratory rhon-

chi, in contrast to the inspiratory difficulty encountered with congenital laryngeal stridor. Laryngeal stridor due to hypocalcaemia (rickets) will be associated with tetany and convulsions.

Respiratory Diseases seen soon after birth

The infant may fail to breathe or the respiration may be very shallow soon after birth. This may be due to either foetal causes *e.g.*, prematurity; obstruction of the respiratory passages by meconium; atelectasis *i.e.* failure of the lungs to expand properly, or to maternal causes e.g. anaemia; delayed labour etc. The child is either pale (asphyxia pallida) or blue and cyanotic (asphyxia livida). Treatment is by means of warmth, artificial respiration after removing the obstructing meconium. The old method of mouth to mouth respiration is now abandoned in favour of gentle raising and lowering of the foot and head ends of the cot, what is called Eve's Rocking Method. Is carbon dioxide to be administered or not?—that is a question which is controversial. 5% carbon dioxide is an excellent stimulant to the respiratory centre, but in an asphyxiated there is already an excess of carbon dioxide in the blood. Even the administration of oxygen by the nasal route is now viewed with disfavour. Paediatricians are of the opinion that intranasal oxygen helps to dry the pulmonary mucosa and promotes the formation of a membrane—the pulmonary hyaline membrane. This membrane interferes with the diffusion of gases in the lungs, thus causing more respiratory distress. Intragastric oxygen is, therefore, recommended in favour of intranasal oxygen.

Congenital malformations of the respiratory tract result in dyspnoea occurring soon or a few days after birth. Tracheo-oesophageal fistula, diaphragmatic herniae, agenesis of the lungs have to be borne in mind as causes of infantile respiratory distress. It may be mentioned here that the suspicion of diaphragmatic hernia should arise if an infant is seen with severe respiratory distress followed by complete intermission occurring suddenly.

Common Respiratory Diseases in Older Children

Obstruction to the respiratory passage: This is an important cause of respiratory illness in older children. Obstruction occurs commonly in children as a result of the following causes:

- (a) the narrow lumen of the bronchi. Even a small plug of sticky mucus can thereby cause obstruction.
- (b) the bronchi are soft and easily compressed, specially by mediastinal glands which are relatively bigger in children and enlarge early *e.g.* in pulmonary tuberculosis.
- (c) the common habit of children of putting various objects in their mouth.

Obstruction due to a foreign body usually occurs at the level of the larynx. Inspiratory stridor and dyspnoea of dramatic onset is the symptom. If the foreign body escapes the larynx it next gets held up at the level of the bifurcation of trachea. Stridor is not a feature. Dyspnoea and as-

phyxia especially on movement is now noted. The onset is less dramatic. Obstruction to the narrow bronchi usually takes place as a result of thick, tenacious mucus which is not brought out easily, as in whooping cough; or, as a result of excessive mucus secretion as in measles. It can also occur during bronchial spasm as in asthma, or when the cough is suppressed, e.g., post-operation. The secretion gathers in the dependent lobes, usually the right lower lobe. If it is not coughed out early it leads to two very important complications viz. collapse of a lobe or segment of a lobe, and superadded infection of the collapsed portion leading to bronchiectasis.

Collapse and Atelectasis

These two conditions do not refer to the same pathological state. Atelectasis means a failure of the lung to expand. The unexpanded lung is still in the foetal state. Collapse refers to an expanded lung which shrinks again and becomes airless. It is very commonly noted in children. Out of 5,000 routine skiagram of the chest of children taken in Edinburgh 854 pictures showed evidence of collapse. The collapse may be massive, lobar, lobular or segmental. When collapse occurs, compensatory mechanism begins to function immediately and correct the intrathoracic pressure. Compensatory emphysema of the healthy portions of the lungs, mediastinal shift to the affected side, a rise in the level of the diaphragm occur whenever there is a partial or complete collapse of a lung. The symptoms of collapse may be nil, or there may be persistent cough, dyspnoea, cyanosis depending on the extent of the collapse. Skiagram confirms the diagnosis. In most of the cases the plug of mucus gradually gets absorbed or is expelled and reaeration of the collapsed portion takes place. Reaeration may fail to take place in whooping cough, tuberculosis, and bronchial asthma. 2% of these cases develop irreversible fibrotic changes and bronchiectasis. It is because of this danger that treatment should be undertaken early. Postural treatment is usually effective if combined with a potassium iodide mixture to soften the thick obstructing mucus. Failing this bronchoscopic aspiration must be resorted to.

Infection

Common cold is more distressing in infants because the small nasal passages are easily obstructed. Moreover, the baby is unable to blow the nose. Difficulty in sucking milk adds to the distress. 1 gr. Menthol in 1 oz. Paraffin; or ½% ephedrine nasal drops often prove helpful.

Acute and Chronic bronchitis are other common respiratory infections. The acute bronchitis may be primary *i.e.*, occur suddenly, or secondary, *i.e.*, following other illnesses *e.g.* rickets, measles, whooping cough. Acute bronchitis is associated with fever, severe cough which is dry in the early stages and moist later on. Dyspnoea, wheezing rhonchi are other features. This condition usually responds rapidly to the oral administration of sulpha drugs and to parenteral penicillin. Repeated attacks of acute bronchitis lead to a chronic condition—Chronic Bronchitis. Such children have been compared by Moncreiff and Evans to a "hothouse plant unable to withstand

any change of temperature." Such children are never free from cough. The treatment of bronchitis is dealt with later.

The Pneumonias: Lobar pneumonia occurs commonly in healthy, robust children and is acute in origin. Broncho-pneumonia occurs usually in infants and children under three years. It is a common complication in marasmic, debilitated and undernourished children. The causal organism of lobar pneumonia is the pneumococcus, while broncho pneumonia is due to staphylococcal or streptococcal infections. Pyrexia, respiratory distress, convulsions, diarrhoea and vomiting, abdominal pain and meningismus are the symptoms usually met with in pneumonia. Respiratory distress is more common in broncho pneumonia, while meningeal and abdominal symptoms are more frequently seen in lobar pneumonia. The course of broncho-pneumonia is prolonged and the mortality rate high, about 54%, in contrast with lobar pneumonia where the mortality is only about 7%. Staphylococcal pneumonia is especially common in children under one year. Lipoid pneumonia occurs mostly in children forcibly fed with cod liver oil, liquid paraffin, or castor oil. The diagnosis of the last-named condition is confirmed by radiological examination. The skiagram will show soft, mottled opacities more at the bases, the shadow persisting for a long time.

The management of acute infections of the respiratory tract is made easier by the introduction of the sulpha group of drugs and the antibiotics. The dose of sulphas is about 0.25 to .75 gm. administered every 4 or 6 hours to children below three years. A higher dose *viz.*, 0.75 to 1 gm. every 4 or 6 hours is given to older children. Crystalline penicillin 10,000 to 20,000 units/lb. body weight per day, (roughly 200,000 to 400,000 units per day) is given with or without procaine penicillin 150,000 to 300,000 units per day. Orally, chlor-tetracycline (aureomycin), tetracycline or chloromycetin may be given in severe cases. The recommended dose is 10 to 50 mgms./lb. per day. This may be divided and given every 4 or 6 hours. Streptomycin 20 mgms./lb. per day may be combined with penicillin for synergistic action.

Empyema is an important complication of lobar pneumonia. It should be suspected if the temperature in the case of lobar pneumonia does not subside in a reasonable time. Signs of pleural effusion *viz.* stony dullness, a shift in the trachea and mediastinum to the opposite side if the effusion is of sufficient quantity, diminished air entry and vocal resonance, enable a diagnosis to be made. The danger of empyema is the formations of adhesions. Aspiration is, therefore, done repeatedly to determine the nature of effusion *i.e.* to note when a turbid fluid changes to thick pus. The development of thick pus is an indication that the purulent exudate is now well loculated and surgical procedures *e.g.*, open drainage can now be attempted.

Bronchiectasis : A cough which persists long after an attack of pneumonia or whooping cough should raise the suspicion of bronchiectasis. Chest deformity, clubbing of fingers and a persistent sinusitis are also important signs for diagnosis. The left lower and lingular lobe are commonly affected.

Treatment comprises in the administration of a suitable antibiotic, postural drainage, and surgical resection in cases which do not respond to medical regime.

Tuberculosis: Tuberculosis attacks the very young child, below three years. The development of the first or primary infection is essential for the protection of the child against the severe forms of tuberculosis. The primary focus is also called Ghon's focus. It is associated with enlargement of mediastinal glands. If the infection is severe, the primary focus may, instead of getting arrested and healing, flare up giving rise to symptoms of tuberculosis. The enlarged mediastinal glands may press upon the soft-walled bronchi and cause the collapse of a segment or lobe. Tuberculous bronchiectasis may develop in the collapsed portion. The conversion of a negative to a positive tuberculin test is of a great help in diagnosis. Since children do not expectorate, gastric lavage must be examined for tubercle bacilli. Streptomycin 20 mgm./lb. per day, isonicotinic acid hydrazide and paraamino salicylic acid 100 mgm. per lb. per day are administered only if there are indications for anti-tuberculous treatment. Such indications are (1) rise in temperature; (2) loss of weight and emaciation; (3) progressive increase in the size of the lesion as seen clinically and radiologically.

With the march of civilization and the consequent development of industries and the conversion of rural into urban areas; with the gradual deterioration in the quality and purity of milk and other food available to children; the better transport facilities which carry infection *e.g.* tuberculosis, to the rural areas and sow it in virgin soil, respiratory tract infections are now increasing among children. It is, therefore, an important problem that medical practitioners in India have to face. This article is written to draw the attention of the medical practitioners to this problem.

MEDICAL ECONOMICS

K. S. SANJIVI, M.D., MADRAS

Since attaining independence, we have heard so much talk about the application of Economics to the different branches of the nation's life. On merely reading the daily newspapers one can note the increasing attention that is being paid to the different possible scientific methods of raising and spending money, of raising, distributing and marketing of food and of providing for the education of children. But as far as the pattern of medical care is concerned, even the possibility that it can be improved has escaped our attention.

Life expectancy in the three countries at the top of economic welfare (U.S.A., Germany and U.K.) with a per capita income of about 500 U.S. dollars is above sixty years; that in the three countries at the opposite end of the scale (Mexico, Brazil and India) with a per capita income of about 50 U.S. dollars, is about thirty-five. Whether we consider the general life expectancy or the mortality from a particular disease such as tuberculosis, the relationship between the economic well-being of a nation and the lesser risk of death is well-established. It is obvious, therefore, that the one answer to improving our longevity is the improvement in our standards of life and the latter is exactly what the Five Year Plans are aiming at.

In this article, however, we will restrict the application of economics to Medicine, to the narrower field of the organization and distribution of medical care, taking into account the facilities we have and the needs of the people in different economic strata of life.

Long ago, Chamousset pointed out that men are the most valuable possession of a state and their health is their most valuable possession. They should have both the means of preserving their health and in case of sickness, they should be able to count on all the aid necessary for their recovery. Almost three centuries back, Daniel Defoe made several suggestions for social security. He proposed the application to the poor of that principle of collective self-help, the insurance principle.

During the last three decades, medicine has advanced so rapidly that it has reached a state in which it could not only save many more lives but keep many more of us well if only we could take maximum advantage of its new developments. Unfortunately, however, many of the new developments are expensive and outside the capacity of not only the poor man but even the middle-class man.

Just as the improvement in the standards of living makes for a healthier and longer life, improvement in the hygienic conditions of the environment must have a salutary effect. But making arrangements for better environmental hygiene is only, in a very limited way, the responsibility of the practising doctor.

We are, therefore, largely concerned with the practical ways in which modern medical aid could be made available to every citizen irrespective of his ability to pay. In this context one naturally thinks of introducing in India a National Health Service on the British pattern. Indeed, our leaders like Raj Kumari Amrit Kaur and Prime Minister Nehru have, more than once, dreamt of providing such a service. Careful thought to the economics of such a service will make one realise how impossible it is of achievement in India. India has 14 times the area of the U.K. with 7.5 times the population and with 6% per capita income. Besides the major part of the population of India is still rural, which means additional cost in working out the scheme.

It is now over ten years since the establishment of the National Health Service in the U.K. During the decade the total expenditure on medical care has nearly doubled itself. The present annual cost is about £730 millions. This sum is about the total budget of the Government of India.

In the Contributory Health Service at Delhi and in the Employees State Insurance Organisations in several parts of the country, the governments concerned have unfortunately adopted a salary system.

It is true that the really good and conscientious person will find in such paid service sufficient incentive for hard and sincere work, but in any large-scale organisation the average should be thought of rather than the outstanding worker. On this view, the NHS in U.K. has adopted for general practitioners the system of capitation payment where the doctor is paid a fixed amount for each person registered with him; this has been acclaimed as a great social invention. "It avoids the possible abuses of fee-for-service or salary; the one invites the doctor to do too much and the other too little." The patients have the choice of their doctors and equally well the doctor who wishes to limit or reduce his work can do so with a good conscience by limiting or reducing his list.

If there is a fear that nationalisation kills private initiative and enterprise, it is most true in the field of medical practice. The personal touch that subsists between the doctor and his patients is not found in other services (*e.g.*, Transport, Insurance or Banking) which may be nationalised.

Kenneth Walker, the famous medical philosopher, says :—"The old friendly relationship between the general practitioner and his patients is in course disappearing. Insured men and women now enter the consulting room to demand their rights and the over-worked and disspirited doctor gets rid of them as soon as he decently can. There can be no doubt rightly or wrongly, that all medical men who are in love with their work are becoming alarmed about the future of their profession. Doctors that come of generations of doctors are now encouraging their sons to break with the family tradition and those few of us that have the prospect of retiring look forward greatly to doing so."

The time has come for the profession in India to assert itself more strongly to ensure that the organiza-

tion of medical care is done on proper lines. In such an organization, what is the place of the general practitioner? Is there a room for him at all or can he be replaced by dispensaries manned by officers "posted" by the authorities? I feel sufficient emphasis cannot be laid on the fact that the general practitioner who functions as the family physician is without any doubt the very foundation of medical practice and medical organization. It has been truly said that he alone could place his patient in the pattern to which he belongs rather than regarding him as a lone sick man who has suddenly popped out from a healthy setting. He alone can bring his judgment to bear upon the life situations as well as on the patient. No amount of most well-equipped and well-run dispensaries and no number of most well-read and well-trained specialists can know the family and its living conditions as well as the family doctor.

A lot has been written about Social Medicine in recent years. It has been pointed out how the patient during his hospital stay is like a "still" picture and how to understand the full story of his illness, we must see the entire movie of his life situation. Likewise, the importance of psychiatry in medical practice is now well recognised. We are now aware that diagnosis is not so much a question of what sort of disease a patient has as that of knowing what sort of patient has a disease.

For the actual practice of Social Medicine and every day psychiatry, the general practitioner is much better equipped than many of his colleagues working inside an institution.

If we grant that urgent steps should be taken to bridge the gap between the brilliant achievements of medicine as a technology and its proper distribution to the public in the low income groups and also decide to ensure and respect the important position of the general practitioner, the obvious course is to provide the latter with diagnostic and treatment facilities of a high quality for his patients who cannot afford such service at private rates.

An experiment in a rather new pattern of medical care is being attempted by the Voluntary Health Services, Madras, registered as a non-profit society. The appeal issued by the Society emphasizes its desire to educate the public on the need for every family enrolling itself with a General Practitioner or "Family Doctor" who will know the family intimately and will do as much as he can by himself, and makes it clear that there will be no direct access to the Society's special services except through the general practitioner who may be either the office doctor or the family doctor or both. The public and the profession in South India will undoubtedly look upon the efforts of the Society with interest and sympathy.

THE PROBLEM OF SURGICAL PAIN

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The choice of the subject

From time immemorial the problem of pain has vexed the minds of philosophers, theologians and physicians. For the physician the problem has been a relatively simple one as without adopting a purely materialistic outlook he could appreciate the value of pain as a warning of disturbed function. In other words pain subserves a useful function, for without it many a disease process would reach an incurable stage before being detected. This means that in medicine the primary problem of pain is how to assess its true significance as a diagnostic agent at the earliest possible stage. The physician however is a humanitarian as well as a diagnostician and so after diagnosing the cause he would try to bring early relief.

The physiology of pain

Pain the signal of danger or damage to the body, leads to reactions designed to minimise it whether they be simple protective reflexes of the spinal cord or the elaborate planning of cerebrum involving such operations as the foundation of hospitals and the prosecution of medical research.

Nerve endings and nerve pathways

Superficial and deep pain: Superficial is somatic and well localised. Deep—visceral is splanchnic—referred pain.

It is clear that any message from the skin will be relatively well localised—the skin has a rich afferent supply, and it can be seen and touched so that the brain has built up a *detailed reference map of the body surface*. The deep structures are not represented in this map. Therefore if a pain message from them shares some of the pathways from the skin, *its place in the map will be referred to the skin.*

The part played by the brain itself in perception and interpretation of pain is not only *fascinating but mysterious*. The cortex no doubt contributes to the perception of pain as a mental state but there is no definite cortical area or pattern. *The cortex and the brain matter itself are painless i.e. do not respond to pain by stimulation of any kind.*

So the reflex apparatus of the spinal cord can render the first aid—it can withdraw the limb, immobilise the injured region and adjust the blood flow. The cerebrum can plan further ahead to avoid renewed danger. But acting alone the individual cannot take effective action to end his sensation of pain anymore than he can always succeed in ending sensations of hunger and thirst. *We must still suffer pain which seems to have no purpose but remind us of our frailty.* By their collective action, however, human brains and minds are capable of devising new methods to aid their bodies. The spinal cord may react to pain by

the flexion reflex—the cerebrum can do so by calling in the doctor to suppress the damage signal and assume responsibility for the danger. *In fact human intelligence developing medicine and surgery has already taken effective steps to prevent unnecessary pain and there is no reason to think that its resources are at an end.*

The visceral pain

Is more important for purposes of diagnosis. It is generally agreed that most of the viscera are insensitive to stimuli that excite pain in somatic structures; such as pressure, cutting or burning. It is not surprising, for as they are not normally exposed to such trauma it cannot be expected that they will be endowed with a nervous apparatus to respond to them.

Abnormal and violent contractions of hollow viscera cause severe pain.

On the other hand the parietal peritoneum, the pleura, the mesentery and blood vessels are sensitive to various injuries, such as handling, stretching and cutting. The pain associated with disease of viscera is believed by some to depend upon stimuli that originate from the structures. In contrast to cutaneous pain, it is usually *more diffuse, less accurately localised, has an aching and sickly character, and is less tolerable* in relation to its intensity.

Pain is so wholly subjective that an accurate estimate of its severity is always difficult. There is no objective measure of pain. The threshold varies from person to person. *Nausea is a part of deep pain syndrome.* It never occurs in superficial pain. It is only found when deep structures are affected and in particular in visceral pain,—angina and biliary colic, ureteric and intestinal colic.

The purpose of deep pain

It has been seen that there is a close relation between skin pain and injury. In the survival of the species it has served a useful purpose in preparing the response to injury. *On the other hand if pain has any function in disease* it is hidden from us. We cannot see that a man passing a minute calculus along the ureter is any better for doing it in torment nor is it revealed to us how the victim of cancer or a cerebral tumour benefits from his pain. Trotter concludes that so far as the interior of the body is concerned, the mechanism of pain appears to be crude, ill-adapted and relatively functionless. In this, the final word on deep pain is "*we don't know*".

Abdominal pain

Is defined as any deep pain felt within the anatomical boundaries of the abdomen. Although it most commonly arises from the abnormal conditions of abdominal viscera and peritoneum—this is not invariably so. Lesions of the chest, spine and abdominal wall are frequently its causes.

The pain is always deep and diffuse and aching in quality unlike superficial pain which is accurately localised. The tenderness observed when a lesion is palpated through the abdominal wall is due to stimulation of the parietal peritoneum brought about by the examining finger. The anterior parietal peritoneum is more sensitive for pain than the posterior peritoneum. Hence the femoral strangulated hernia is most often missed.

Charactet of abdominal pain

Since pain is caused by a peripheral stimulus but felt after passing through three neurones—in the cerebral cortex, its quality will vary not merely with the transmitting agent but with the receiving apparatus *i.e.* with the mental background, courage and general health of the patient. If no description of the nature of pain can approach to accuracy, so also no reliable method of tracing the source is available.

Analysis of pain will allow the separation of the following characteristics of any pain :

1. Nature.
2. Its severity.
3. Its site.
4. Its periodicity.
5. Its time relations.
6. The manner of onset and departure.
7. Its duration.
8. The sites if any to which it is referred.
9. The factors which aggravate and those which relieve it.

Nature

Splanchnic or somatic—whether it is colic of a muscle working against difficulties or spasm of one faced with complete block: somatic may be due to peritonitis—chemical or bacterial.

The severity

Here the scale of values differs—as the threshold for pain varies from patient to patient. Ogilvie is of opinion that the pain of perforated peptic ulcer is the most agonising which the human being is ever called upon to withstand. It is the only one which will make a strong man drop to the ground motionless and voiceless. Second gall stone colic, third ureteric and fourth acute pancreatitis. Moyinihan stressed the agony of acute pancreatitis.

The site

Will often give a clue to the site of origin. Splanchnic pain is felt in an area which betrays the embryonic rather than the present position of the outraged viscus. Midline organs *i.e.* the alimentary canal and all its associated glands refer their pain to the midline; those developed laterally, *i.e.* genitourinary system to the corresponding side. Within these regions the upper portions refer their pain higher than the lower; the oesophagus to the episternal notch; the stomach, duodenum, pancreas and biliary system to the upper epigastrium; the small intestines, appendix and caecum to the lower epigastrium and umbilicus; the large intestines to the hypogastrium; the kidney to the loin, radiating as it gets more severe to the flank and iliac fossa, the ureter to the groin and external genital; the bladder to the suprapubic region in the midline.

Somatic pain is felt in the area of skin supplied by the cerebrospinal nerve, the sensory endings of which in the peritoneum are receiving the painful stimulus. In most cases the site of pain will correspond anatomically to the site of lesion but there is a notable exception in the case of diaphragm. This is a cervical organ drawn down during development—so referred to 4th and 5th cervical nerve area in the neck and over the acromian process.

By the periodicity of pain is meant its distribution in the time sense surveyed over a long period. This periodicity is therefore of importance in

the chronic rather than in the acute lesions of the abdomen. These are almost unnoticed at onset followed by a gradual increasing intensity of pain, an inexorable advance with no appreciable remissions, speaks of cancer. The pain of peptic ulcer will occur in bouts, which rarely lasts less than a month, separated by intervals of freedom lasting anything from a month to several years. During the bout the pain usually recurs regularly day after day with no holidays; during the intervals it leaves no reminder. Gastric pain which disappears for a day or two is rarely due to an organic lesion of the stomach or duodenum. Long remissions favour duodenal, short ones gastric ulcers. Appendicular attacks are scattered irregularly rarely lasting less than 48 hours or more than a week; not often coming more than two or three times a year and showing no constant interval. Gall bladder attacks tend to be of moderate severity at first, but to become more severe with repetition. The interval between them obey no rule. Pain arising in the Fallopian tubes and ovaries tend to follow a regular monthly cycle, usually being at its worst immediately before the period. The pain of any inflammatory focus such as an infected pelvic appendix or colonic diverticulum which lies in contact with the female genital organs will also be increased by the premenstrual engorgement and show the same monthly periodicity.

By the time relations of a pain are meant its behaviour when analysed over a period of 24 hours and especially to environment or to the function of any abdominal viscus. The most familiar example of such a time-table is the regularity with which gastric or duodenal pain follows meals. A clock-like punctuality is suggestive of a duodenal or gastric lesion but it must be remembered that a meal is the signal of awakening peristalsis throughout the alimentary tract the lower ileum empties into the caecum, the colon undergoes mass peristalsis. Any lesion of the large intestines may therefore give the symptom of pain related to meals.

The onset of many abdominal affections is typical : gall stone colic comes with the dramatic suddenness, appearing at any hour of the day or night, unheralded and evoked by no known circumstances, reaching the maximum intensity within a few minutes of the first twinge. At the other extreme, pain of ulcer never appears suddenly, but will take from a week to a month, to pass from the stage of discomfort after meals to that of fully developed ulcer pain. The onset of appendicitis is usually so typical-umbilical splanchnic pain followed by fixed somatic pain wherever the appendix may be that the diagnosis is rarely missed unless the somatic pain is absent.

The history of the way the pain disappeared after an attack may give a clue to the nature of the trouble. An inflammatory lesion can right itself only gradually ; the pain of an ulcer fades way as slowly as it appeared, that of inflammatory appendix can never remain severe for three days and be gone the next. A colic ceases as suddenly as it had started but it is apt to reappear after an interval of a few hours. While the real pain of colic disappears abruptly, it often leaves a soreness behind, which the patient describes as feeling as if he were bruised

or beaten. This cycle—abrupt onset, abrupt termination, residual soreness is typical of pain caused by stones in the gall bladder and the kidney.

The duration of pain may be of considerable diagnostic importance. In general, the short lived pains are due to mechanical causes—*i.e.* they are colics, the enduring ones are caused by organic changes; bacterial or neoplastic.

The factors that aggravate or relieve pain already present will often give further help. Any aggravation or alleviation of pain by function of some organ suggests that that organ is affected by the pain producing lesion or is in close proximity to it. Pain made worse or relieved by food probably arises in the stomach; pain only when the bladder is full and is disappearing when the urine is passed warrants the belief that some inflamed structure such as diseased appendix or a pyosalpinx is lying in contact with its walls. The pain of stones is brought on by standing and tends to become more insistent towards the end of the day; it is relieved by lying down. Sudden falling is apt to start a renal stone on its painful downward passage.

Relief of pain by emptying of a viscus—of the stomach by vomiting and of the colon by the passage of wind points to distension of its muscle coat as the cause and demands an enquiry into the reason behind it. Alkalies relieve pylorospasm not only of ulcer but the reflex spasm due to gall stones or appendicitis. Lastly it must be remembered that the nerves of the visceral sensation run their course along mesenteries, and across ganglia through the diaphragm and beside the thoracic vertebra until they enter the cord to pursue further paths as yet undetermined. The gastric crisis of tabes, and the colicky attacks that accompany tabes mesenterica are reminders that pain of true visceral type may be extra visceral in origin. Similarly, the somatic nerves which supply the abdominal lining supply also its walls and the bones and joints of its framework, and pass on their way along the confines of the thorax. The errors which may arise from the lesions of the anterior or posterior abdominal muscles from diseases of the chest or spinal column or from extrathecal tumours pressing on the posterior nerve roots in the lower dorsal region are familiar from books or from bitter experience. But in the end the diagnosis will depend not alone upon the collection and right interpretation of all possible facts, but upon the judicial shrewdness with which the facts are marshalled, the relative importance assigned to each and the conclusion drawn. Such wisdom is not acquired from books nor yet from discussions but only at the bedside.

About urological pain Dr. Cadge wrote in 1879, "The behaviour of the bladder towards a stone is most peculiar and puzzling. In one case it displays almost perfect indifference and even acts the part of host with an approach to hospitality and allows the guest to grow and stay until it reaches enormous proportions. In other cases no sooner does a stone enter at one portal than the conflict begins and goes on until it is turned out along the urethral passage at the other portal or the bladder is perpetually fretted, worried and inflamed until either the

surgeon or death comes to end the dispute."

"Pain is perfect misery, the worst of all evils; and excessive, overturns all patience":—

(*Milton's Paradise Lost*).

The complaint of Milton in 'Paradise Lost' has without a doubt been uttered by human lips throughout the ages, for pain is as old as humanity—and perhaps even older there is reason to believe that it is inherent to any life linked with consciousness.

The proper management of pain remains after all the most important obligation, the main objective and the crowning achievement of every physician.

Individuals vary greatly in their sensitivity to pain and it is by the art as much as by the science of medicine the suffering is assessed and the right dose of drug is prescribed. We must count ourselves lucky that so far there is no pain-meter or other instrument of precision to confuse the issue.

It takes atleast a year fully to understand the vagaries of a new drug and therefore, since life is short, it is wise to use a few preparations and know them well. It was said of a great clinician that as his experience widened, he reduced his list of drugs so much that ultimately he confined himself to *bicarbonate of soda*!

Analgesics are composed of several heterogenous group of drugs which act on various physiological systems concerned with pain, namely:

1. Those which have their effect on the CNS, as for example OPIATES and other synthetic analgesics like Demoral (Pethidine) and Methadone (Physeptone).

2. Those which exert a local action on the pain conducting system at the site of injection as exemplified by local analgesics.

3. Those drugs which are not analgesics in the usual sense in that they do not directly affect either the perception or the reaction to pain but which are employed in special situations for the purpose of removing the condition giving rise to stimulation of pain receptors., *i.e.*,

(*a*) Smooth muscle relaxants which relieve pain by abolishing marked smooth muscle spasm, *e.g.*, atropine.

(*b*) Skeletal muscle relaxants such as curare and the curare like derivatives—*e.g.*, Flaccidil and Myenesin (in tetanus).

(*c*) Vasodilators such as papavarine and priscoline which increase circulation and abolish ischaemic pain.

"Man made aspirin and morphia came from heaven" is the saying.

Morphia is still the sheet anchor for relief of pain. More patients are saved by morphia than killed by it. Pethidine possesses both atropine-like *spasmolytic action* and morphia-like *analgesic action*.

DIAGNOSIS OF CORONARY THROMBOSIS*

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The incidence of coronary thrombosis has increased from decade to decade in this century. This increased incidence is not only because one is more and more aware of it and many instruments of precision are available in its diagnosis, but also because there is an actual increase in the incidence as recorded by Sir Maurice Cassidy in his Harveizn oration. Unfortunately, there is a tendency amongst doctors to dub all cases of sudden death as due to coronary thrombosis. This diagnosis is used as a convenient jargon where the cause of sudden death is not quite clear.

Coronary thrombosis is characterised by pain, shock and acute left ventricular failure. Sometimes, it may manifest itself as a thromboembolic phenomenon or as cardiac arrhythmias.

The pain is the most arresting symptom of coronary thrombosis. It is one of the most severe types of pain one may experience. It is often described as constricting in nature retrosternal in distribution. The pain comes on generally at rest. This is distinct from angina of effort where the pain comes on with exertion and disappears with rest. If the patient had angina of effort which generally better with rest or with nitrites, he may be dismayed to find that his present pain does not respond to vasodilators and has struck him while he is at rest. The patient may describe the pain as severe in intensity, as if he is caught in a vice. The pain may be so severe that he may toss in bed and fail to find any decubitus which is comfortable. The pain generally tends to radiate to the left upper extremity. In one of my patients, it radiated to the jaws and he presented himself at the out-patient with a complaint of severe pain in the lower jaw. A superficial examination might have landed this patient in the dentist's chair, but a careful examination showed him to be severely collapsed and his ECG showed evidence of posterior myocardial infarction. Quite a number of my patients had upper abdominal discomfort as the presenting symptom, with no evidence of rectrosternal pain. This upper abdominal discomfort is often mistaken for flatulence and many cases are likely to be missed if myocardial mischief is not thought of. Any patient around the age of 40 who complains of gaseous distension and upper abdominal discomfort should be investigated from point of view of coronary thrombosis. A few of my patients had a burning sensation over the chest. The usual teaching that if the pain is not constricting in nature, coronary thrombosis should be excluded, I am afraid is not always true. One can-

* Lecture delivered to the post-graduates in August 1958.

not be dogmatic about the nature of the pain in coronary thrombosis.

It has been said that 10 to 20% of patients with coronary thrombosis may not have pain at all. In the elderly patient, it is some other symptom, other than the pain, *e.g.*, cardiac arrhythmia, which attracts attention to the heart. When coronary thrombosis produces complete heart block and Adam-Stokes' Syndrome, the patient may not complain of pain since he is unconscious. When dyspnoea is intense, pain may not be appreciated. Repeated attacks of coronary thrombosis resulting in increased fibrosis of the myocardium may interfere with the pain fibres and in such cases pain may be absent. Pain is a subjective sensation whose threshold varies from person to person. What is severe pain to one person may be nothing at all for another.

Shock is due to the hypo-systole of the heart. It is characterised by progressive fall of blood pressure, tachycardia and cold and clammy sweats. The temperature may sometimes be sub-normal. In very severe myocardial infarction, the cold sweats are so profuse that one has to change the patient's sheets very often and the limbs may feel icy cold. In such cases, cyanosis is obvious in the tongue and nails. In some patients, the blood pressure may not record a fall for 6 to 12 hours. On the other hand, rarely, it may actually go up, in the first few hours before it begins to register a fall. In hypertensive patients, unless one knows the previous readings, it may be difficult to appreciate the fall of blood pressure.

Acute left ventricular failure is one of the presenting cardinal symptoms of coronary thrombosis. The patient who had been quite normal suddenly becomes dyspnoeic; in the first few hours, there may not be any positive findings on asculation of the lungs. Later on, a few crepitations begin to appear, which may become coarse and pulmonary oedema may supervene.

As already mentioned, cardiac arrhythmias, especially in the elderly, should make one suspect the presence of coronary thrombosis. The commonest complication is auricular fibrillation, whereas ventricular tachycardia is generally fatal.

Clinical examination of the heart is often negative in the first few hours following the attack. If the patient is severely shocked, there may be tachycardia and bradycardia may be present if there is heart block. Sometimes a gallop rhythm may be audible. Pericardial rub, which is often diagnostic, does not occur before 36 to 48 hours are past after the onset of pain. In certain patients, the onset of hemiplegia may be the first indication of a pre-existing coronary thrombosis. There may be myocardial infarction which are entirely silent and which are detected as old scars at autopsy or in the course of routine screening examinations as myocardial aneurisms.

By the end of 48 hours, there is generally a rise of temperature. The temperature seldom goes beyond 102°F. and generally settles down to normal in the course of a week. It is due to the absorption of protein-like substances from the infarcted area. The sedimentation rate begins to get elevated between the third and fifth day of the attack and reaches a maximum by the fifteenth day and it reaches normal by the end of one month.

Of all diagnostic methods, the most important and most dependable one is the electrocardiogram. If repeated cardiographic examinations using all the available unipolar chest and limb leads show normal configuraties, it is unlikely that there is infarction of any size. The usual changes in the ECG in cases of myocardial infarction are the presence of a pathological Q wave, elevation of ST segment and inversion of T wave. In the course of a few weeks, the ST elevation becomes less and less marked until it becomes iso-electric. The T wave which was inverted to start with gradually gets flattened and when the recovery is favourable, it may become erect. The persistence of a Q wave suggests the formation of a scar. If ST elevation persists through months and years, it is suggestive of a myocardial aneurism.

There are certain fallacies which should be appreciated before one can be definite about an infarction from the study of an ECG. Thus a Q wave in V_1 and V_2 is seldom pathological; but if there are R waves in V_1 and V_2, the presence of a deep Q wave in V_3 should be considered pathological. When the heart is vertical, a Q wave may be normally present in AVL and it should not be mistaken for a high lateral infarction. A Q wave in lead 3 is of no significance if a similar pattern is not maintained in AVF. Elevation of the ST segment in V_1 and V_2 should be considered as normal. Pericarditis without any infarction can also produce ST elevation and this should be differentiated from the type of ST elevation observed in acute myocardial infarction; when Left bundle branch block is present, Q wave may be absent.

During myocardial infarction, it has been found that many enzymes are released into the general circulation, the important two being glutamic oxalacetic transaminase and serum lactic dehydrogenase. Serum glutamic Oxalacetic transaminase (S-G-O-T) is widely distributed in animal tissues but is most concentrated in heart muscle. Usually, the S-G-O-T varies from 10 to 40 units per m.m. when tested at room temperature. Levels above this are not seen in patients with infections, neoplastic, metabolic or degenerative diseases in which acute destruction of heart, skeletal muscle or liver tissue are not present. The level of S-G-O-T following experimentally produced myocardial infarction almost invariably rises between 4 to 6 hours of the injury and remains elevated for 2 to 5 days, depending upon the extent of necrosis. In the study of coronary thrombosis, no co-relation between the height of S-G-O-T and age, sex, colour, weight, height of temperature, level of W-B-C count, sedimentation rate, presence or absence of shock, blood pressure, presence of cardiac failure, the mortality rate or the use of anti-coagulants, is observed. There is significant co-relation, however, between S-G-O-T and the electrocardiographically estimated size of the infarct. The higher the enzymatic activity, the larger the infarct. In the study of 74 out of 75 patients who had acute transmural infarction undertaken by La Due and others (Circulation 1955, Vol. 11, p. 871) the S-G-O-T rose to 2 to 20 times the normal. It has been laid that the method of analysis is relatively simple and that the results are comparable with different laboratories.

SOME SYNDROMES OF THE CERVICAL SPINAL CORD*

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Diseases of the spinal cord contribute no small number to neurological disorders. Since the spinal cord is a compact structure containing numerous long conducting tracts, pathological lesions are likely to produce widespread disturbances. An early recognition of these disturbances will go a long way towards amelioration of the symptoms and a cure. In the spinal cord itself, it is observed that pathological lesions tend to be more or less concentrated at the upper *i.e.* cervical segments and the lower *i.e.* lumbar segments. In the cervical region the complement of the nerve fibres is more than at any other level, with the result that lesions of this part of the spinal cord result in gross disturbances of neural function.

We are now in an age when infections are no longer the menace they used to be. The control of infections and institution of improved preventive measures has resulted in giving the individual a longer lease of life. This has led to an increase in the incidence of neoplastic and degenerative diseases. Ability to be independent, to move about freely and to work are some of the things that we treasure most. In nervous disorders these very things are affected early, leading to a progressive disability with the attending psychological consequences. If these conditions are recognized early and appropriate therapeutic measures instituted, many a patient can be restituted to normal. There is a tendency on the part of many practitioners of medicine to neglect the study of neurological problems, considering them as incurable and to be of only academic value. Elsewhere I have pointed out the presence of similar attitude towards yet another aspect of neurologic medicine.

It is not my intention to give you an exhaustive account of all the diseases that affect the cervical portion of the spinal cord, but to confine myself to the commoner syndromes whose early recognition will make all the difference between recovery and progressive disability. In my experience, and I am sure it is in the experience of most people, the commonest diagnosis made in cases of focal spinal cord lesions is neuritis. A history of innumerable injections of vitamin B complex and B_{12} is a very characteristic one. The temptation to diagnose 'neuritis' and to prescribe vitamin B complex is far greater than to submit the patient with muscular wasting and/or root pains to a painstaking neurologic examination and investigation. Not only that, the lack

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of any improvement following 'massive B complex and B_{12} therapy' only makes them persist in it for a longer time, or blame the potency of the preparation without reviewing the original diagnosis. If only more clinicians coming across cases of focal neurologic disturbances look for any local aetiological factor that can be removed or treated effectively then only will the present state of affairs improve. It is always my practice to teach the undergraduates the one important principle in clinical diagnosis *i.e.* to look for any cause, focal or otherwise, that is removable in any disease process before dubbing it as incurable. This if pursued will pay rich dividends. Only a few years ago elderly people developing slowly progressive paraplegia were diagnosed as 'senile paraplegics', an inevitable degenerative process in the spinal cord, a legacy of old age. With Russell Brain's demonstration of the pathology of the cervical intervertebral disc and the secondary spondylitic changes as the responsible factors in many of these cases, much light has been thrown on this problem and its effective management. This proper analytic approach to all clinical problems is real clinical research and will clear the fog around many similar diseases. I shall now consider some common diseases that affect the cervical portion of the spinal cord.

CERVICAL SPONDYLOSIS

The term cervical spondylosis has been applied to the condition where degenerative changes in the cervical intervertebral discs result in the formation of secondary spondylitic changes in the adjacent vertebral margins. This secondarily leads to disturbances of the cervical cord. This condition has been known to both radiologists and clinicians for a long time under such names as, osteoarthritis, spondylitis etc. Spondylitic changes in the cervical region is a very common finding, and according to Valentine Logue, is the commonest cause of spinal cord disease in people over fifty.

The earlier studies on the pathology of the intervertebral disc following the clinical descriptions of disc rupture by Mixter and Barr nearly twenty-five years ago has brought intervertebral disc abnormalities to the forefront. These studies revealed that intervertebral disc rupture consisted in an injury to the peripheral annulus fibrosus followed by a protrusion of the central gelatinous nucleus pulposus through the tear. This condition, often referred to as a disc prolapse or nuclear herniation was most commonly met with in the lumbar region and less commonly at other sites. This type of a disc lesion was often the result of an indirect trauma or strain as in lifting a heavy weight suddenly. In the cervical region, however, nuclear protrusion is not a common feature, instead a protrusion of the annulus fibrosus is more frequent. The other striking difference is that trauma plays little part in initiating the changes in the disc.

The changes in the intervertebral discs is a degenerative one, starting in the third decade of life. The essential change is that of dehydration, the fluid content of the disc falling from about 88% at birth to about 70% in old age. This results in a loss of the normal elasticity of the nucleus pul-

posus and a gradual shrinkage, and an alteration, in its consistency. Pari passu with this the fibres of the peripheral annulus bulge and become coarser and have to bear most of the normal stresses and strains the spine is subjected to. The result of all these is the appearance of new bone (osteophytes) in the form of spurs from the margins of the vertebrae producing the characteristic lipping. These spurs may bridge the intervertebral space forming transverse bars across the posterior part of the vertebral bodies. They may however be localized either in the midline, paramedian position or in the region of the intervertebral foramen.

The shrinkage of the intervertebral disc alters the normal alignment of the vertebral bodies and the intervertebral joints. This produces secondary arthritic changes in them leading to more osteophytes which consequently narrow the intervertebral foramen. The formation of osteophytes between the vertebral bodies and around the articular surfaces of the intervertebral joints constitutes cervical spondylosis. These changes can occur at any of the cervical discs but are more frequent in the fifth and sixth discs probably because movements are greater at this level.

The degenerative changes in the cervical discs that are responsible for the secondary changes in the vertebrae initiate a train of events affecting the spinal cord and the nerves that originate from it. The latter are responsible for the varied neurologic symptomatology seen in cervical spondylosis. The changes in the disc lead to a shortening of the spinal column resulting in an alteration in the normal relationship between the nerve roots and the corresponding intervertebral foramina. Added to this, the narrowing of the intervertebral foramina by the bony spurs, result in a constant rubbing of the nerve roots with their covering sheaths against the bony prominences. The extreme mobility of the cervical spine and the fact that movements occur here during most of the day result in changes in the nerve roots and their covering sheaths. The resultant fibrosis around the nerve roots is referred to as root sleeve fibrosis; the term root sleeve being applied to the attachment of the dural envelope to the sensory and motor rami of the spinal nerves which finally forms the fibrous covering of the nerves. The tethering action of this change on the nerve roots makes them unduly susceptible to injury in the normal day-to-day movements of the spinal column. The presence of root sleeve fibrosis, the importance of which was first drawn by Frykholm, is one of the most important factors determining the clinical symptomatology and the treatment to be adopted.

The narrowing of the intervertebral foramina by the bony spurs is mainly responsible for nerve root symptoms. The spinal cord on the other hand is mainly affected by the protrusion of one or more intervertebral discs and the accompanying bony ridges. The ridges, which may occasionally form a continuous bar across the vertebral canal extending from one intervertebral foramen to the other, may compress the spinal cord against the laminae and ligamenta flava posteriorly or press upon the anterior spinal artery producing ischemia of the cord. The cord changes are particularly

aggravated by the repeated movements of a cord that is more or less fixed by the root sleeve fibrosis, over the bony ridges. Thus the clinical manifestations of cervical spondylosis are mainly of two types—one, that of nerve root irritation and compression *i.e.* the radicular syndrome, and the other, that of spinal cord dysfunction *i.e.* the cervical myelopathy syndrome. Both these may occur together or independently.

Clinical Features

The clinical history of the patient with cervical spondylosis may be spread over a wide period ranging from a week to a number of years. The changes in the cervical spine alone are often symptomless. It is only when the nervous system is involved that the symptoms appear and are taken note of. As already alluded to, the symptoms are due to nerve root compression or cord compression or both. In most instances symptoms of nerve root involvement tend to develop more rapidly than those of cord compression. In three instances of nerve root compression that came under my care the symptoms developed more or less suddenly. One other case had a fairly rapid onset, spread over a week or two, but there had been a previous history of recurrent unilateral cervical pain over a period of six months. The myelopathy syndrome is more gradual in onset. One patient aged about 60 years, started with difficulty in walking some fifteen years ago. The symptoms progressed for about a year and then remained stationary till a few months before he presented himself at the hospital. In another case, a male aged about 40 years, the symptoms progressed rapidly over a period of three months rendering him bedridden. For descriptive purposes the syndromes of radicular and spinal cord involvement will be considered separately. Though symptoms of radicular or cord involvement may predominate both are often affected together.

The Radicular Syndrome

The onset of the symptoms of nerve root involvement may be sudden or gradual. In quite a few there is a past history of recurrent bouts of cervical pain, occurring at the same site and lasting for a few days or a week or two. This history of recurrent cricks in the neck should put one on the guard and exclude any changes in the cervical spine before dismissing it as insignificant. In some cases the pain due to nerve root involvement may be a source of confusion. The pain may be referred to the myotome *i.e.* muscles supplied by the nerve root, or to the sclerotome, *i.e.* bones and joints supplied by the nerve root. Thus the pain may be referred to the chest, neck and upper limbs, mimicking cardiac pain. Pain may be confined to one shoulder giving the picture of a 'frozen shoulder' or periarthritis. I have come across instances of both the above types. The commonest feature is for the pain to be localized to the neck. There is often an associated spasm of the cervical muscles leading to stiffness and a partial immobilization of the neck. In those where the onset is more gradual, pain may not be a feature. It is often replaced by paraesthesia along the upper limb, which is worse at night.

The pain in cases of nerve root involvement has been shown by

Frykholm to be due to involvement of both the sensory and motor components. His stimulation experiments on patients operated under local anaesthesia showed that stimulation of the motor root produces a prolonged aching pain, while the sensory root stimulation resulted in a sharp, shock-like pain referred to the myotome, sclerotome as well as the dermatome.

Next to pain the most important feature, and the one that draws attention to the seriousness of the affection is muscular paralysis. Wasting of the shoulder girdle muscles, especially the deltoid is a frequent feature. In one of my patients the presence of deltoid wasting was the clue to the diagnosis (See Fig. 1). He was referred as a case of rheumatic torticollis resistant to therapy. Muscular weakness and wasting may affect any of the groups. In my cases the following muscular groups have been frequently involved: small muscles of the hand, pectoralis, flexor group and deltoid. The muscle groups involved correspond to the nerve roots affected. Here it is worth noting that there is a wide variation in segmental distribution of the cervical nerve roots. Wasting of the small muscles of the hand is often seen with spondylitic changes between C 7 and 8.

Diagnosis: Diagnosis is quite simple if each and every case of cervical or upper limb pain, particularly if associated with muscle spasm, is investigated for abnormalities in the cervical spine. The presence of muscular weakness and wasting and any sensory disturbances should be a pointer to further investigations.

Radiological examination of the cervical spine is essential in each and every suspected case. Conventional postero-anterior, lateral and oblique views may reveal the abnormalities. In some cases lateral views may have to be taken in both flexion and extension to demonstrate the encroachment of the spinal canal and intervertebral foramina. Osteophytes may be seen projecting into an intervertebral foramen corresponding to the nerve root suspected to be involved. This is clearly seen in Fig. 2, where a prominent bony spur is seen narrowing the intervertebral foramen indicated by the arrow. Similar but smaller bony spurs are seen in the intervertebral foramina above and below. In the postero-anterior view (Fig. 3) bony spurs can be seen between C 4 and 5 and C 5 and 6. The spur arising from the left side of the upper border of the sixth cervical vertebra is particularly striking. In many cases there is a loss of the normal cervical lordosis resulting in a straightening of the spine as seen in the lateral view (see Fig. 4). Narrowing of the intervertebral spaces may be seen in some cases.

In some cases, as happened in one of mine, the conventional films do not show any abnormalities. The patient referred to above, had typical root pains and paralysis of the muscles supplied by the 5th cervical segment. The spinal films were normal. The patient was sent for a second series and by some oversight the radiologist took a soft tissue film and this clearly demonstrated the abnormality. Therefore as Northfield has pointed out, the bony ridges and spurs may not be markedly calcified and may show up only if soft-tissue films are taken.

For a proper radiological evaluation of the cervical spine abnormality in

suspected cases of the radicular syndrome it may be necessary to take lateral, postero-anterior and oblique films, and possibly soft-tissue films. Here a word of caution. The clinical description of the syndrome of cervical spondylosis has resulted in the overdiagnosis of this condition. The presence of spondylitic changes in the spine is no evidence for spinal cord or nerve root involvement. In the absence of incontrovertible signs of involvement of the nerve roots or spinal cord the radiological findings may not be of significance.

Myelographic examination is rarely required but may be occasionally necessary to localize the nerve root affected particularly in cases with extensive spondylitic changes. The characteristic sign of nerve root affection is obliteration of the normal root pouch of the affected nerve.

Treatment : In the acute stage treatment consists in bed rest and immobilization of the neck. With this alone there is a prompt relief in the pain and muscular spasm. Analgesics may be necessary in some cases and their use in most cases will enable the patient to give the much needed rest. The period of immobilization required may be two to three weeks. In some cases where this preliminary rest has not relieved the pain cervical traction may be necessary. In my experience, failure of rest and immobilization was invariably due to it being imperfectly carried out and the impatience of the patient. In such cases traction may have to be employed. In the absence of the proper equipment for cervical traction I employ a modified method using the Glisson's sling as shown in Fig. 5. The traction should be maintained for short periods of time every day; *viz.*, about an hour or two, two or three times a day. After some time it is advisable to prescribe a suitable collar which should be worn throughout the waking period. There are a number of collars available but none are socially acceptable. The plastic type of collar advocated is very difficult or impossible to procure in our country. The alternate one and the one that is very effective is a felt collar that is wound round the neck a number of times. This and similar collars are employed with the object of preventing the normal movements of the cervical spine that increase the damage to the nerve roots and the cord. This collar may have to be worn for about three months. With this almost all patients are symptom-free within a short period. The most important thing to be borne in mind is that the treatment should be persisted to get the desired results.

Surgical treatment for nerve root involvement is more often performed in American countries. The principle involved is to relieve the pressure on the nerve root and may require a laminectomy and removal of the offending bony spur and in those cases with narrowing of the intervertebral foramen, widening of the foramen. The above decompressive procedures are not likely to be of any benefit if there is a marked root sleeve fibrosis. In such cases incision of the root sleeve and dividing any adhesions present may produce the desired result. Surgical measures are to be preferred if the conservative regimen fails.

Since the disease is a degenerative one there is every likelihood of pro-

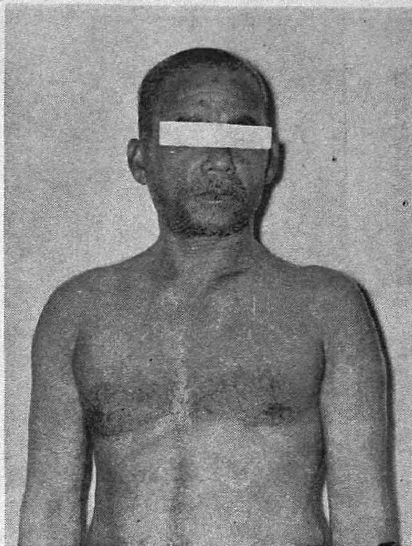

FIG. 1

Photograph showing wasting of the left deltoid.

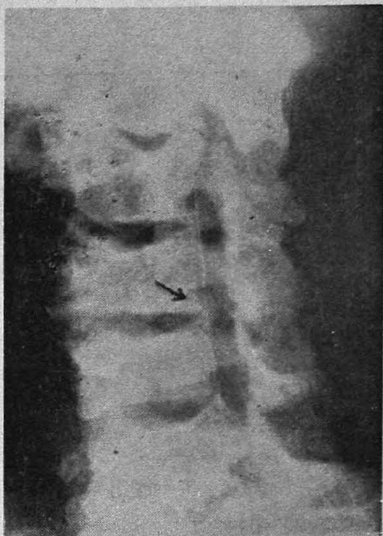

FIG. 2

An oblique view of the cervical spine showing osteophytes encroaching on the intervertebral foramen.

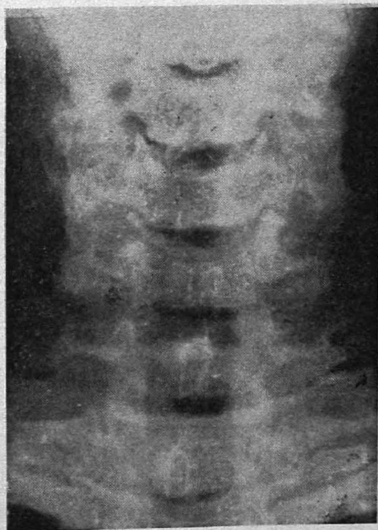

FIG. 3

A P.A. view of the cervical spine showing osteophytes arising from the upper and lateral parts of the vertebral bodies. This X-ray is of the same patient shown in Fig. 2.

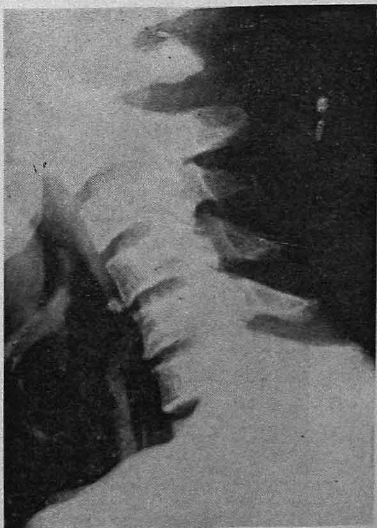

FIG. 4

A lateral view of the cervical spine showing the loss of the normal lordosis.

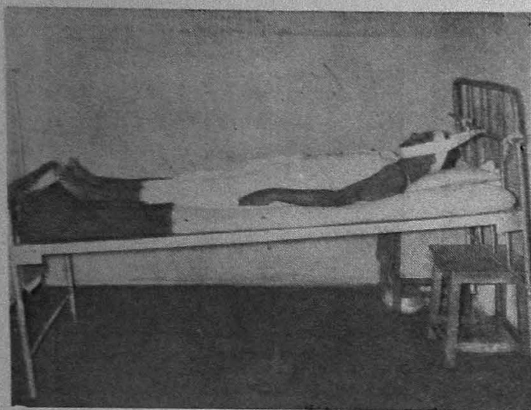

FIG. 5

A photograph showing the method of cervical traction employed.

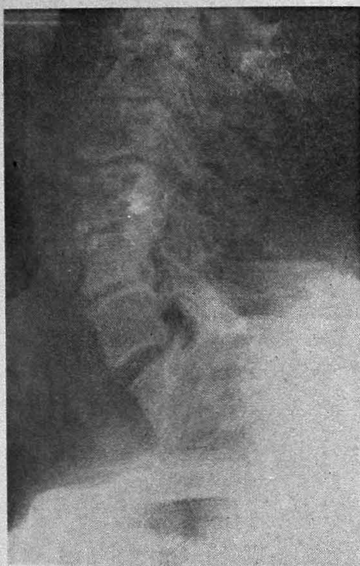

FIG. 6

A lateral view of the cervical spine showing narrowing of disc spaces and spondylitic reaction.

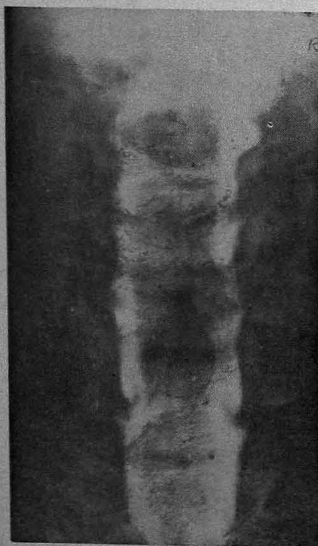

FIG. 7

A P.A. view with myodil in the cervical canal showing the characteristic transverse filling defects in the column.

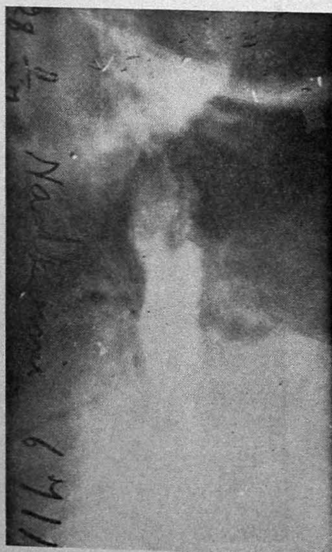

FIG. 8

A lateral view of the same patient shown in Fig. 7, showing the indentation of the myodil column by the osteophytes.

gression of the pathological process and consequently recurrence of symptoms. The conservative measures may have to be persisted for a long time and re-employed whenever the pain returns.

The Cervical Myelopathy Syndrome

The above term is applied to the syndrome where there are predominant signs of involvement of the cervical spinal cord in cases of cervical spondylosis. The onset and progress of the condition is more gradual than that of the radicular affection. In quite a few cases symptoms of nerve root involvement are also present.

A striking feature of many of these cases is an early steady deterioration in the clinical condition with a cessation of further progress at the end of some period. In one case of mine, the symptoms progressed for a year and then remained stationary for a number of years. In another, this initial period of deterioration was only three months.

The clinical features are mainly those of involvement of the pyramidal tracts. The mechanism of this affection has already been mentioned. The main difficulty is that noticed in walking. In one case there was such an amount of spasticity that the patient was bedridden. The progressive disability in walking is the most striking feature. In a few cases the disability may be due to an ataxia, the result of either a posterior column or spinocerebellar involvement. This is by no means a frequent finding.

In many, signs of nerve root involvement are also present, thus giving the appearance of a lower motor neurone affection in upper limb and an upper motor neurone affection in the lower limb. The most frequent finding has been that of wasting of the small muscles of the hand. In a case of mine this was so striking and the absence of any sensory findings raised the possibility of motor neurone disease. Subjective sensory symptoms as paraesthesia of the hands are frequently found. Objective sensory loss tends to be patchy and not clear cut as with spinal cord neoplasms.

Diagnosis: Clinical signs of a progressive pyramidal affection with or without any radicular lesions should raise the possibility of cervical spondylosis particularly in middle age and older people. The pleomorphic clinical picture may result in confusion with many disorders notably motor neurone disease, syringomyelia and spinal neoplasms. Motor neurone disease may simetimes be difficult to exclude without a complete radiological examination. The sensory involvement characteristic of a central cord disease and the age are of considerable help in distinguishing syringomyelia. The characteristic sensory disturbances will help in the diagnosis of spinal cord growths. Differentiation may not be possible without a proper radiological assessment.

The radiological examination consists in taking the usual postero-anterior, lateral and oblique views of the cervical spine. In the lateral views, narrowing of the disc spaces and often an increase in the cervical lordosis are invariably noticed. (See Fig. 6). To determine the disc responsible for the compression myelography is often necessary. It is particularly essential in distinguishing from

other spinal cord lesions, as the presence of spondylitic changes is no indication for it being responsible for the symptoms. In most cases the characteristic myelographic defect is the breaking of the column of contrast material by the transverse bony bars. This can be seen in the postero-anterior view (Fig. 7). In the lateral view the deep indentations by these bony spurs may be seen in the myodil column. (See Fig. 8). In some cases there may be a complete block (Fig. 9). In this case even with the patient at an angle of about 75 degrees Trendlenberg, the myodil column would not enter the cervical curve.

Treatment: The initial treatment consists of bed rest and immobilization of the neck as with the radicular syndrome. Traction may have to be employed and often gives relief. The use of a cervical collar is essential in maintaining the clinical improvement and in preventing further damage.

Surgery has been often employed and consists in doing a laminectomy and section of the dentate ligaments. The role of the dentate ligament in many lesions of the spine has been pointed out by Kahn. The division of the ligament will permit more mobility to the cord. Removal of the bony protrusions from the front of the cord is a more radical procedure and may require to be done at multiple sites. The results of various surgical procedures employed have been reported to be encouraging but the series reported are quite small to permit a proper assessment. The possibility of spontaneous arrest should be borne in mind before embarking on radical procedures.

Fracture Dislocations of the Cervical Spine

That acute injuries to any portion of the spine can result in damage to the spinal cord is too well known. That chronic neurologic disturbances can also result from similar injuries is less well known. The types of lesions are legion. I propose to consider one type, the so-called "tear-drop" fracture of the cervical spine. This type of lesion is often the result of compression of one vertebral body by the one above it leading to a chipping of the anterior part of the body of the lower one. The broken fragment resembles a drop of water or tear dripping from the rest of the body and has earned it the name. The more important one however is the posterior displacement of the rest of the vertebral body into the spinal canal. If neglected, the broken fragment, along with the secondary osteophytes formed as the result of the instability of the fracture site leads to a compression of the anterior part of the spinal cord. The importance of this type of chronic anterior spinal cord compression developing after acute injuries to the cervical spine was pointed out by Taylor in 1929. He also drew attention to the fact that even if the cord has escaped damage during the injury, the projecting bony fragment can result in a chronic compression of the spinal cord. In this, the tethering action of the dentate ligament plays an important role in further damaging the cord.

A case of this type came under our observation recently. This was a Hindu, female, aged 19 years who gave a history of a fall from a height of ten feet some four years before. The patient apparently lost consciousness for some time and when she recovered

consciousness she was found to have a paralysis of all the four limbs. Patient was not given any particular treatment and she made a gradual recovery over a period of one year. The improvement was more in the right upper and lower limbs. Clinical examination revealed a marked weakness, spasticity, exaggeration of deep reflexes, extensor plantar responses and ankle and patellar clonus on both sides. The weakness and hypertonia was more marked on the left side. In the upper limbs there was wasting of the deltoid, forearm flexors and small muscles of the hands, all more on the left side. Weakness was also pronounced in the left upper limb.

An X-ray of the cervical spine showed an old fracture dislocation of C 4, 5 and 6 vertebrae with a posterior mass projecting into the spinal canal. (See Fig. 10). A spinal manometry showed signs of a partial block.. A myelographic examination revealed an obstruction to the flow of the myodil column at the lower border of the 6th cervical vertebra. A small streak of myodil could be seen passing upwards along the right side of the spinal canal. A small amount of myodil can be seen depicting the upper level of the obstruction (Fig. 11).

At operation a left hemilaminectomy of the 4th, 5th and 6th cervical vertebrae was performed by Dr. M. K. Guha. The dura was found densely adherent to the laminae and the underlying cord. The cord was pulsating very feebly. Division of adhesions could not be effectively carried out because of the dense adhesions.

The patient had an uneventful post-operative period, but there was-n't any appreciable improvement a month after the operation.

The idea of describing the case in detail is to bring home the fact that extensive and irreparable damage can be brought by imperfect or lack of prompt and early therapeutic measures. To prevent the complication of cord compression in the acute flexion or the "tear drop" fracture dislocation of the cervical spine it is advised to employ early traction, laminectomy with the patient in traction with sectioning of the dentate ligaments. In old standing cases where there is marked anterior compression of the cervical cord results with surgery depend on the degree of damage to the cord. The bony mass projecting backwards may compress the anterior spinal artery producing permanent ischaemic changes in the spinal cord. Surgical measures employed for these old cases consists in laminectomy, dentate section and excision of the bony growth.

SPINAL CORD TUMOURS

Spinal cord tumours are being recognised more frequently today because of the facilities available for their treatment. In spite of this we do come across cases of spinal cord neoplasms being missed and treated as 'neuritis' etc. I shall not consider tumours of the spinal cord in great detail as this will form a paper by itself. I shall only consider some aspects of the problem so as to enable one to suspect the condition.

Clinical Features

Since most spinal neoplasms grow slowly symptoms are that of progressive neurologic deficit. The possi-

bility of a spinal tumour should be suspected in each and every case of progressive nervous symptoms. In a few cases particularly after a traumatic episode a tumour may be unmasked. From the point of view of clinical descriptions two types of growths are recognised ; an intramedullary tumour, where the tumour starts in the centre of the cord and an extramedullary tumour, where it starts outside the cord and secondarily presses upon it. We do not have adequate statistical material to study the frequency of various kinds of tumours of the spinal cord, as seen in our country. From various reports intramedullary tumours seem to be as common, if not more than extramedullary tumours.

The onset is usually gradual particularly so with intramedullary growths. The earliest symptom may be muscular weakness with or without wasting. The presence of muscular wasting is a valuable localising sign. The weakness often starts on one side of the body, *i.e.*, weakness of a limb. This progresses and soon the opposite limb is affected, the time interval depending upon the rate of growth of the tumour. Pain is strikingly absent in intramedullary tumours, but is a very striking feature of extramedullary growths. The pain is that of nerve root irritation and the location of this pain is a valuable localizing sign. Bladder symptoms may not be seen in the early stages, but later on precipitancy may be a feature. Objective sensory loss is present at some stage of the disease process. Intramedullary tumours may produce the characteristic dissociated sensory loss (loss of pain and temperature with intact touch) seen in cases of syringomyelia. This type of sensory loss is usually segmental affecting the dermatome supplied by the spinal segment affected. Where more extensive sensory loss is seen, sparing of the sacral segments is more characteristic of intramedullary growths. With extra-medullary tumours the sensory loss is likely to give false localising signs. The spinothalamic tract that is responsible for conduction of pain and temperature fibres shows a characteristic lamination. The fibres from the lower part of the body tend to be pushed to the outer surface of the cord by the fibres that enter at higher levels. The result is that in the case of an extra-medullary tumour arising in the cervical region, the sacral fibres in the spinothalamic tract being most superficial tend to be affected early resulting in an anaesthesia in the sacral areas. As the tumour grows it presses more and more on the spinothalamic tract and the anaesthetic area progresses upwards till the segment affected is reached. With intramedullary tumours on the other hand, the pressure will be felt in the more central fibres and less on the outer fibres leading to the characteristic sacral sparing. This is only a generalisation and there may be differences, as happened in one of my cases of an intramedullary growth, an ependymoma, there was no sacral sparing and the anaesthesia was of the ascending type. This was probably due to a pronounced distension of the cord resulting in secondary pressure effects by the vertebral laminae on the spinothalamic pathways.

Alterations in the reflexes such as the absence of one deep reflex and the exaggeration of the lower one will be of localising value. In this connection

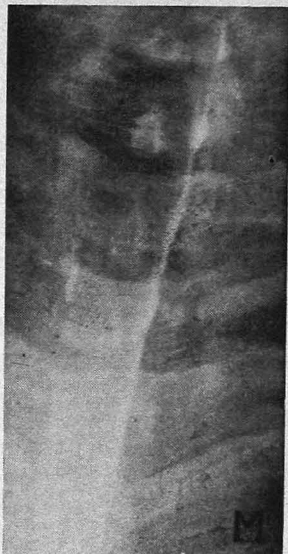

FIG. 9

A myelogram showing complete block.

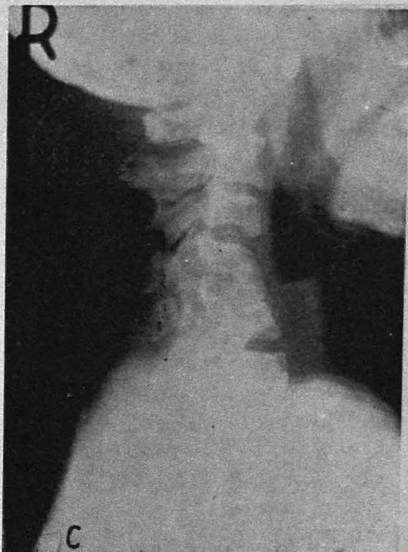

FIG. 10

A lateral view of the cervical spine showing old tear-drop fracture. The bony mass projecting into the spinal canal can be clearly seen.

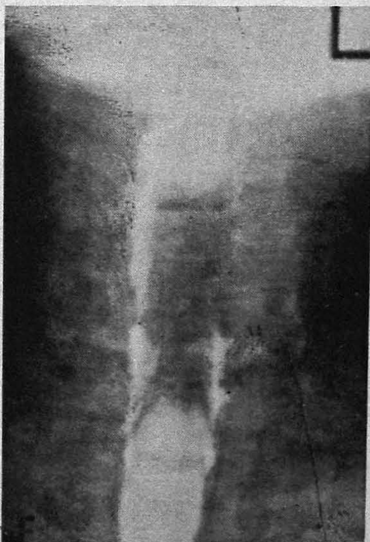

FIG. 11

A myelogram of the patient shown in Fig. 10 showing the filling defect and level of obstruction.

FIG. 12

A mylelogram showing the characteristic filling defect of an intramedullary neoplasm.

one particular reflex change is important. When the normal radial or supinator reflex is taken by striking the lower end of the radius, there is flexion of the elbow. In cases of local lesions of the 5th and 6th segments of the cervical cord, this normal elbow flexion is lost and is replaced by a flexor movement of the fingers. This is referred to as an inversion of the radial reflex.

Diagnosis: Diagnosis in cases of spinal tumours rests on suspicion. Spinal fluid studies, particularly pressure studies may be of help. Signs of complete cord compression are less frequent than those of partial compression. Pressure studies should include the effect of jugular and abdominal compression (done separately) on the pressure. The rate of rise and fall after bilateral jugular compression and release is of great value. The rise in the protein content may not be very great in cases with partial compression. Tumours should be diagnosed before the typical Froin's syndrome develops.

Plain skiagrams of the spine may reveal abnormalities like erosions of the pedicles. Myelographic examination is of great help in not only diagnosing and distinguishing certain types of tumours but is of immense value in localising them precisely. Myelography does not consist in injecting the contrast medium in the wards and taking films. With this, early growths are bound to be missed. The correct method is to instill the contrast material (myodil) with the patient on the X-ray table. The material may be instilled by either a lumbar puncture or cisternal puncture, the former being preferred by me. With the table being tilted gradually, so that the head end is lower than the pelvis and with head and neck in extension, the flow of the myodil column is closely watched. Any abnormalities are noted and spot films taken. In the case of intramedullary tumours there is a characteristic filling defect in the myodil column. As the column approaches the tumour it splits into two, one going on either side of the growth. (See Fig. 12). With extramedullary growths on the other hand there may be a lateral indentation of the column of contrast material.

Treatment: Treatment is essentially surgical. The outlook being better in those with an extramedullary than those with intramedullary growths. Early diagnosis and prompt institution of therapy is the only sure method of getting good results.

CONCLUSIONS

I have tried to touch on some of the common lesions of the cervical spinal cord whose early recognition is very essential in administering the appropriate treatment. The paper was prompted by the step-motherly attitude adopted by most medical men to neurologic problems. The idea of this paper is to kindle the thoughts of people most interested in the care of the sick and to reorient them in their views on neurologic diseases.

ACKNOWLEDGEMENTS

I am particularly grateful to Dr. A. Timmapayya, District Medical Officer, Govt., Headquarter's Hospital, Mangalore for permission to include some of my cases in the body of the paper. To Dr. M. K. Guha, Hon. Orthopaedic Surgeon, Govt., Headquarter's Hospital, and Reader in Orthopaedic Surgery, Kasturba Medical College, Mangalore, for permitting me access to his material, I am very thankful.

SOME ASPECTS OF HYPERTENSION

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With the increasing stress and strain of modern life, there is increase in the incidence of Hypertension. Hypertension is more common in places where people are living under undue stress. For instance, conditions of living in the United States are much more exacting than in U.K. and conditions of living in U.K. are definitely more exacting than what obtains in India. It has been observed that when Americans go over to Oriental countries like India or China, their pressure comes down without any medication. In the West appointments are made, and it has got to be maintained to the split second, whereas no appointments are necessary in India. No Hospital, big or small, insists on patients having appointments before they come into Hospital. Patients sometimes arrive even one or two hours after the hospital is closed and get annoyed if they are not attended to. I narrate below an incident. It may be a bit of an exaggeration, but it serves my purpose to show how things are viewed in this country.

A man arrived at the Station at a particular time expecting the train to come at least one hour late. But he was surprised when he found that the train had arrived in time. So he expressed his surprise to the Station Master about the punctuality of the train, and the Station Master told him the train was due to arrive there not at that particular time but 24 hours earlier. In other words, it was one day late.

In most cases, wages are not paid either weekly or monthly but annually. Nothing serious will happen even if a man attends to his work one or even two hours later than the scheduled time, particularly when he is an agricultural worker. But as the tempo of life is getting faster with the rapid industrialisation, hypertension is likely to become very much more common than what it is today.

In Government General Hospital, Madras, where a separate Hypertension clinic is being run, we see on an average almost 10 to 20 new cases every week, and about 100 patients are attending the clinic weekly. Cases with a systolic pressure of 150 mm. or above, and diastolic pressure of 100 mm. or above recorded consistently are only considered as suffering from Hypertension. A reading taken at random may not give the correct picture, particularly when it is taken for physical fitness or for Life Insurance purposes, but if this happens to be raised and subsequently under quieter conditions the recorded pressure is low, it is called Labile Hypertension.

Recently Prof. Smirk has standardised the procedure. He calls the first reading as the casual pressure. He advises the patient to be at rest in the

hospital for the night preferably with 1½ gr. of phenobarbitone and in the next morning if the patient is still restless he is put on another 1½ gr. of phenobarbitone. The patient and the doctor occupy a secluded room where no telephone, no interruption, and no talking is allowed. The pressure is recorded without any conversation between the doctor and the patient. The pressure is also recorded monotonously at intervals of half a minute—each time the pressure is recorded as Pressure Nos. 1, 2, 3 and so on till it is recorded for 20 minutes. He noticed in quite a good percentage of cases the pressure drops down appreciably. The initial reading he calls it as the casual pressure and the B.P. taken under conditions mentioned above, he calls it as the basal pressure—the difference of pressure between the basal and the casual pressure, he calls it as supplemental pressure. He observed that where the basal pressure is normal they tend to have a good prognosis. He has also noticed irrespective of the supplemental pressure so long as the basal pressure is low the prognosis is good. Even with high supplemental pressures as high as 90 mm. Hg. the prognosis is excellent. Whatever changes are seen with supplemental pressures varying from 0—20 he has seen occurring with supplemental pressures ranging between 40 and 50 mm. or even with higher pressures. In other words he lays great stress in assessing the basal pressure. He considers cases with high basal pressures need alone be treated as B.P. cases. Under conditions in which we now are, it may not be possible to devote so much attention to find the basal pressure. Under the circumstances in which we now are, it is enough if we take the pressure if the patient has rested for the night preferably with sedatives and preferably without any sort of conversation between the doctor and the patient. The whole thing is done in a mechanical way, so that there may not be any emotional episode. It is common experience to note pressures tending to shoot up when problems of emotional nature are discussed in front of the patient and for the same to come down once the patient talks on different topics. The question arises if the casual reading of high pressure is noticed, does he need any treatment ? The answer is the basal pressure has got to be determined in these cases and if the basal pressure is found to be high then they do not need treatment even if no symptoms have been present in the first estimation. The situation, to my mind, is very much like a car going at a high speed, oftentimes we are told by the driver that he has been driving all these days at rash and negligent speed and he has also been getting on without committing accidents, but the chances of meeting with an accident are much greater than the man who drives the car at permissible speed. I prefer in all hypertensive individuals (hypertension of the basal pressure) to be treated, whether they have symptoms or have no symptoms. If they have symptoms with adequate treatment the pressure comes down. Once the pressure is consistently kept at low levels, the symptoms clear, so also some of the changes that are constantly seen as a result of hypertension. The changes that are constantly seen as a result of hypertension which has lasted for some years are a grossly dilated

heart particularly the left ventricle, dilatation of the aorta though not to the same extent as the left ventricle; E.C.G. showing left ventricular strain, eye grounds showing soft and hard exudates, haemorrhages with or without papilloedema. Cases which show haemorrhage and papilloedema should be taken as more serious; so also cases of hard exudates. When in doubt, we always requisition the services of an Ophthalmic Surgeon to test whether there is papilloedema or not. It is not easy even for the Ophthalmic Surgeon to spot out the very early papilloedema. In well-marked papilloedema, the Ophthalmic Surgeon's help though not needed to spot the case, yet he is asked to see them. The idea is to have an independent colleague, to assess the progress. He is totally unconcerned about the drugs used and so his estimate of the fundus changes is an independent opinion.

With regard to the symptoms that occur in hypertension, some symptoms are more often seen after the patient has become aware that he has been suffering from hypertension particularly head-ache, but a dull morning head-ache seems to be the common in hypertension. Other symptoms are lassitude, giddiness, irritability and not being able to go to sleep in time. The symptoms are totally absent in persons with very high pressures. I have often been surprised by some of my markedly hypertensive cases when asked about their sleep, that they slept soundly and do not have any difficulty whatsoever with regard to their sleep. It is also surprising how most of these symptoms are totally absent in a good percentage of cases. The larger the number of cases of hypertension, one sees, one is likely to see symptomless hypertensive cases. In other words there is no particular symptom which can be said to be pathognomonic of hypertension. Often hypertension has been spotted in the course of routine medical examination. B.P. is taken on account of some vague complaint on the part of the patient and it has been found to be raised. Once we record a consistently raised B.P. we quickly eliminate some of the common etiological factors like renal conditions, endocrine disorders and dietetic errors like ingesting excessive salts and things like that. One thing which has struck me as an important feature is the impaired renal excretions, ability to concentrate urine is impaired and it gets markedly affected with the onset of malignant hypertension. Once the kidney fails and non-protein nitrogen has gone above 100, the end does not seem to be far off. Another serious complication that we meet with is the cerebral haemorrhage. In the large majority of cases death within 48 hours is the rule, though an extraordinarily small percentage of cases escape. I have observed in the last few years in the older age group when cerebral thrombosis occur in hypertensive patients, though there is a temporary improvement as a result of treatment fresh episodes of thrombosis occur in different areas and roughly in about 2 years after the onset of first symptoms patient dies as a result of multiple thrombotic episodes occurring at different places and at different times.

Treatment: Having established that we are dealing with cases of essential hypertension, we have the patient weighed. Where he is grossly over-weighed, he is put on a low fat

diet. As the weight is brought down sometimes the pressure also comes down though not appreciably at least by 10—15 or sometimes even 20 mm. of Hg. Substituting sugar for saccharine is of definite advantage in some of these cases. Diet is so adjusted that the weight loss is roughly about 2 lbs. per week. The excessive body weight in some of these people can be controlled by the use of amphetamine though amphetamine may by itself cause in otherwise healthy people a small rise in pressure. In the few obese cases under my care it has not been so and they seem to stand amphetamine very well. Once the pressure has been brought to a steady level and the weight also has started coming down I prefer to put the patient on one of the Rawoulfia group of alkaloids. Most of my cases were on rawoulfia which is available under the trade name of R.S. 51. In cases where the pressure has been high, I have used a combination of all the hypotensive alkaloids, Rawoulfia which is available under the trade name of R.S. Forte. In my series of cases these two products have given consistently better results than reserpin. The side effect that is met with in the course of reserpin may be met with rawoulfin also but the incidence is less in rawoulfin than in reserpin. The side effect that is met with in the use of rawoulfia group of alkaloids is stuffiness of the nose and sometimes in susceptible subjects asthmatic attacks may be precipitated. The sedative effect of the rawoulfia is particularly well marked. When patients are under the action of rawoulfia group of alkaloids they tend to fall off to sleep, particularly when they are engaged in doing monotonous acts like sitting and reading a text-book or listening to a lecture. The main action of the drug seems to be by causing vasodilatation and sedation. This group of alkaloids is not of much value in arteriosclerotic hypertensive subjects. One important objection that has to be borne in mind while administering rawoulfia group of alkaloids is the mental depression of a very profound nature which may be met with. The mental depression will be such as to drive the patient to attempt suicide or even commit suicide. Though the incidence of this sort of thing is less than 0.1 per cent in my own series of cases, this aspect of serious side effect must be kept in mind. The main advantage with rawoulfia group of alkaloids is when it acts it steadily brings down the pressure without causing any untoward symptoms and also it permits the treatment in ambulant cases. The action of reserpin seems to be a bit slow and to record a fall in pressure one should give this drug for 7 to 10 days before the hypotensive effect is observed. I have also noticed that the increase in dose of rawoulfia group of alkaloids does not cause arithmetic proportionate fall of pressure. I have got some of my patients on R.S. 51 for more than 5 years more or less continuously without coming across any adverse symptoms. If for some reason or other the patient discontinues the drug on his own he should be able to have the pressure recorded daily when he is off the drug. Anywhere between 3 to 6 days the pressure starts going up again. The drug should be re-instituted if there are signs of the pressure going up. I personally choose one of the specific alkaloids *viz.*, Rawoulfin or Reserpin

in preference to the whole root extract which has got pressor factors which in some cases cause elevation of pressure instead of depression of pressure. I am sure we have no right to cause any further elevation of pressure in a hypertensive subject though administering whole root extract most times causes a fall of pressure; what is required is not an inconstant fall but a consistant fall in pressure. Where isolated alkaloids are used either it produces a fall in pressure or no fall but never a rise in pressure.

The dose of rawoulfin *i.e.*, R.S. 51 is 1/6 grain t.d.s. before food. This works out to 7.5 mgms. per day. With this dose as I have already stated mental depression is very rare, nor do patients complain much about having had dreams of a disturbing nature. With higher doses the side effects are more prominent without achieving a remarkable fall in pressure.

Where the rawoulfia group of alkaloids have not achieved a desirable fall of B.P. or where they have totally failed the next thing to do is to start the patient on ganglion blocking agents. The ganglion blocking agent which we have been using with considerable advantage is Pantolineum tartrate, available under the trade name Ansolysen. We start our patients on 10 mgm. t.d.s. before food and every second or third day the dose is increased. The dose is increased upto 80 mgms. morning—40 mgms. noon and 80 mgms. night. Since most of our patients are treated on ambulant basis we do not increase it beyond this level. Even in inpatients we have found this dose as the safe upper limit. With this dose, we do not get any unpleasant symptoms. The ganglion blocking agents act on the ganglion and they are used to block the sympathetic ganglions but they not only act against the sympathetic but also against the para sympathetic. It is their action on the para sympathetic that brings about the unpleasant features of the drug. The unpleasant features that may be met with are postural hypotension resulting in fainting of the patient when he suddenly assumes an erect posture or if he stands in one position for a long time. The action of para sympathetic results in dryness of the mouth. It interferes with the gastric peristalsis as also gastric secretion with the result the appetite is impaired and sometimes the patient may vomit. For instance it causes slowing down of the peristalsis and leads on to constipation. If administered indiscriminately it may lead on to paralytic ileus, particularly in the elderly patients who are likely to have mild prostatic enlargement. Without much of a clinical manifestation administration of these drugs are likely to result in retention of urine. Most of the side effects can be controlled by suitable anti-dotes for instance the postural hypotension is considerably reduced if the pressure is checked always in the erect posture, preferably having the patient standing and having the arm resting on a couch. If the pressure is recorded in this posture that is the lowest pressure that is likely to record. By this device one can avoid giving a dose which is likely to cause severe postural hypotension. Sometimes in spite of taking this drug postural hypotension can occur and this can be avoided by the patient either by sitting, or lying flat. This causes elevation of pressure and clears the

fainting attack. With regard to the dryness of the mouth Pylocorpine nitras 1/15 gr. administered once or twice or sometimes thrice after food clears any dryness of the mouth. With regard to constipation I prefer a sénna preparation like the Pursennid to be given the previous night. About half an hour before the expected time as Prof. Smirk has advocated administering 15 mgm. of Prostigmine considerably relieves the constipation. Sometimes instead of constipation diarrhoea may occur. I have had a patient under my care who used to alternate between constipation and diarrhoea. Luckily these cases are rare. The main drawback with regard to Ansolysen—I have noticed—is the uncertainty of absorption. One can never be sure about what is likely to happen with a given dose. When the drug is well absorbed there will be a well marked hypotensive manifestation but where it is not absorbed there may not be any appreciable clinical result. This can to some extent be avoided by giving it in the form of an injection which is an impracticable procedure in a large O.P. practice, and particularly where the patients are so illiterate they cannot be trained to take the injection for themselves. The combination of Rawoulfia with Ansolysen allows us to achieve what we desire; by using a very much smaller dose of ansolysen than by what will otherwise be needed. No patient of mine has been treated purely on Ansolysen.

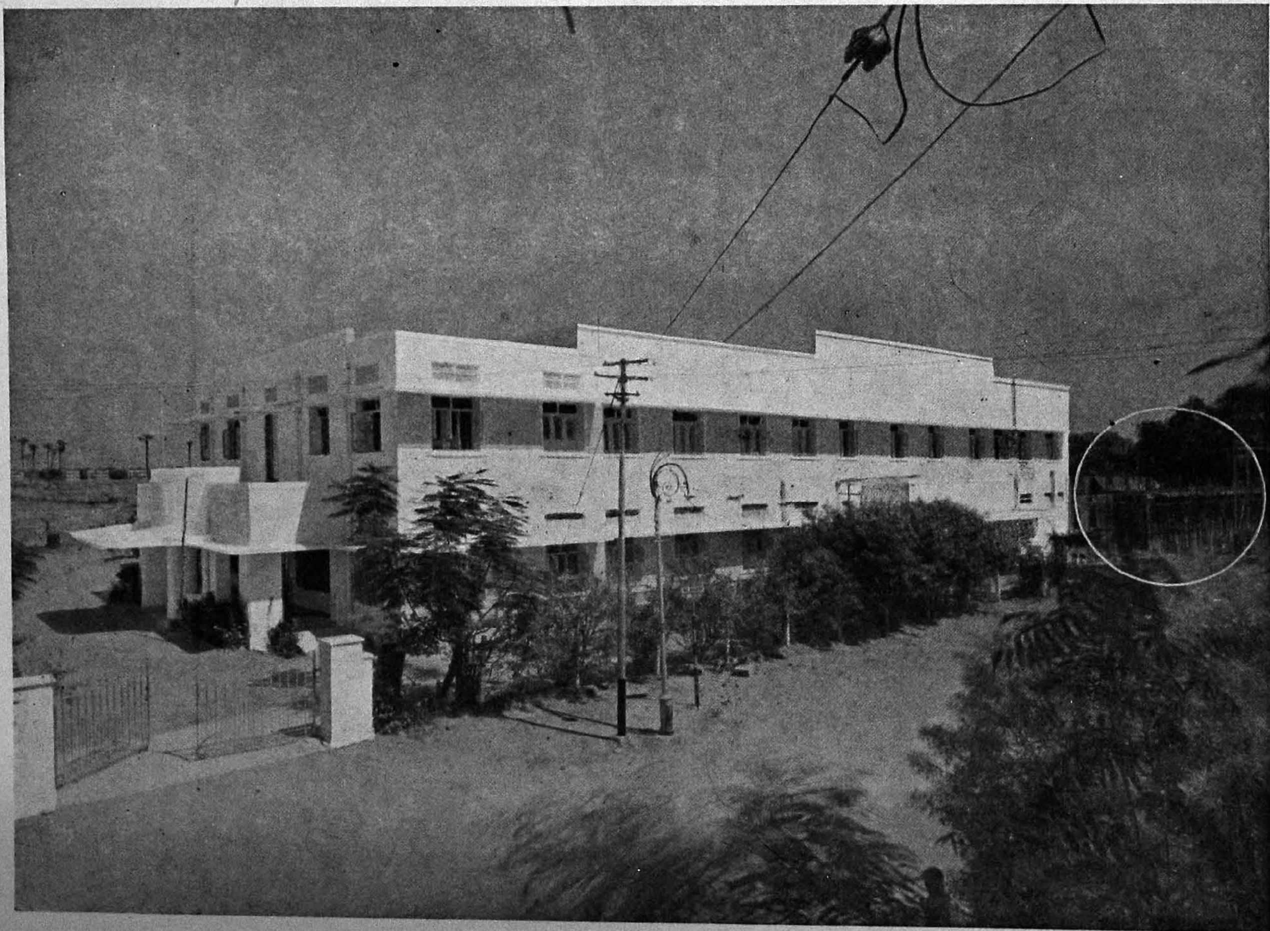
On account of the uncertainty of action of Ansolysen we were on the look out for some other drug which will give more certain results and at this stage Mecamylamine was introduced into the market under the trade name Mevasine. This is totally different from ansolysen—being a secondary amine, it is available as 10 mgm. tablets. We start our patients, on $\frac{1}{4}$ tablet once a day in the morning. This can also be combined with R.S. 51 or R.S. Forte. This combines with ansolysen as well. The great advantage of this drug is that it is well absorbed even on oral administration and the action lasts for quite a number of hours say 6 to 8 or even more. The side effects are definitely more common than with Ansolysen. So we do not use this drug as a routine. We reserve this drug only for cases who have failed to respond to ansolysen. All the side effects that I mentioned under ansolysen also occur with Mevasine in a more intense form. The drug when it is administered for the first time may sometimes cause a precipitate fall in pressure. So it is preferable to institutionalise the patient for whom administration of Mevasine is contemplated. After the first few days of therapy patient gets accustomed to this. Constipation is much more severe with Mevasine than with Ansolysen but the fall of pressure is more consistent with Mevasine than with Ansolysen. The dose of Mevasine is increased at intervals of 4 to 5 days by $\frac{1}{4}$ tablet morning and noon *i.e.*, twice a day before food if the result *is not satisfactory. Most of my patients* are given $\frac{1}{4}$ tablet twice a day with very good results. If the postural hypotension is too marked at a particular time say an hour after breakfast then the dose of Mevasine can be administered $\frac{1}{8}$ pill before breakfast and $\frac{1}{8}$ pill after breakfast so that the precipitate fall at one stage is avoided and thus prevent fainting attacks.

Recently a diuretic agent has been made available under the trade name *Chlortide.*

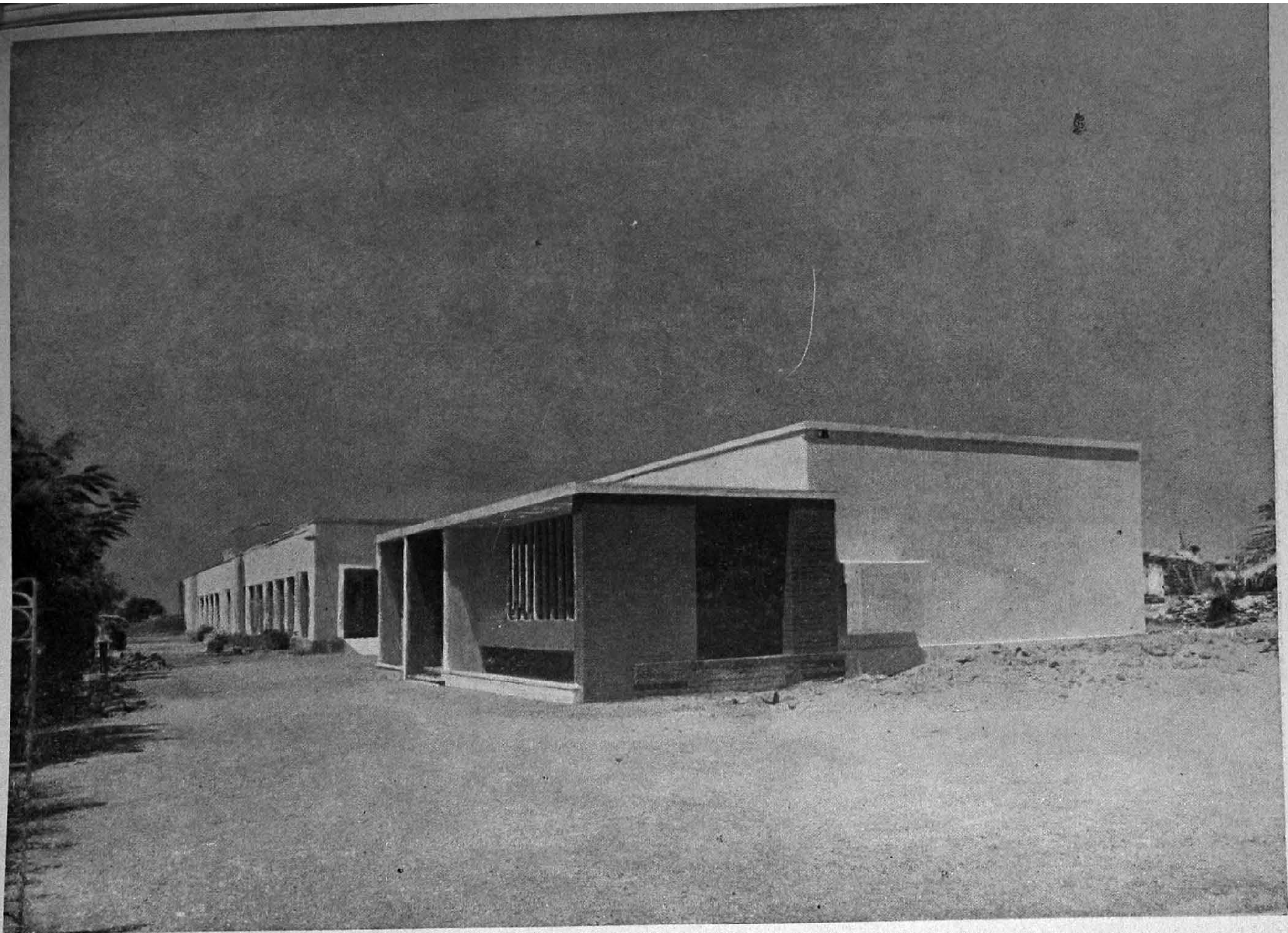
Chlortide-chlor-thiazide. It has got the peculiar property of preventing tubular re-absorption and eliminating or excreting both the sodium and potassium ions. Since it eliminates sodium ions it causes fall of pressure. By eliminating potassium ions it is likely to precipitate hypopotassium symptoms. So while administering this drug it is necessary that the patient should be given oral supplementation of potassium salts preferably as Pot. citrate 10 gr. twice a day. The drug gives very satisfactory results in cases of essential hypertension or even in malignant hypertension but the results are very poor, where the urine volume is lowered on account of kidney damage. The great advantage with this diuretic is, it is not contraindicated. even in the presence of liver damage.

Two grams of thiazide produces the maximum diuresis—the same amount of urine output as with 2 c.c. of mercurial compounds given as i.m. injection. When chlor-thiazide is used and once the oedema is controlled it is enough if half a tablet is used on alternate days in the mornings after breakfast. The sodium loss effected by chlorthiazide is more than what is obtained with mercurial diuretics. With the administration of mercurial diuretics chloride loss is appreciably more than the sodium loss. The drug is marketed as 0.5 gm. tablets, and our patients seem to stand best the dose of half a tablet twice a day. It can be given continuously for a few days when the oedema is well marked. The toxic effect of chlor-thiazide is due to potassium depletion. The symptoms of potassium depletion may be weakness or malaise and loss of deep reflexes. E.C.G. changes can also be recorded. These changes can be prevented by simultaneous administration of potassium citras, to patients getting chlorthiazide. Administration of potassium is particularly indicated when chloride is administered to patients suffering from cirrhosis liver. It has been claimed chloride supplements the action of hypotensive agents like Mevasine and Ansolysen. Where the hypertension is high—the supplementing action at any rate in my cases has not been very marked though it does reduce the pressure in cases where the pressure has not come to stay. Though we have not had it for a sufficient time and for a large number of cases, our experience has shown that this is a very good diuretic agent available for oral administration.

A judicious combination of rawoulfia with the ganglion blocking agent supplemented with chlortide and symptomatic treatment of such symptoms as insomnia, constipation and cough by the use of hypnotic agents, laxatives and demulcents which promote intestinal peristalsis—most cases can today be controlled and maintained at normal pressure levels. Often I have been asked whether it is necessary to treat hypertensive cases when they are symptom-free. My answer is, it is necessary to treat such cases as they are more liable to cerebral vascular catastrophies, anginal attacks or acute pulmonary oedema than normotensive individuals.



Cancer Institute, Adyar, Madras



Cobalt Block—Cancer Institute, Adyar, Madras

ASHOK VIHAR—HEALTH & RECREATION CENTRE CORPORATION OF MADRAS

DR. P. S. SAMBANDAM, L.R.C.P. (Lond.), M.R.C.S. (Eng.)
Director.

ASHOK VIHAR, Health and Recreation Centre, was started in 1948 and is being maintained by the Corporation of Madras. The State Government is also pleased to sanction an annual grant of Rs. 10,000.

The aim of the centre is to provide both preventive and curative health services to families of the low income groups residing in and around the slums in close vicinity to the centre. Each family pay a monthly token membership fee of one anna.

The following services are offered to the member families:

1. Periodic health check up.
2. Free Laboratory investigations, screening and X-ray of chest.
3. Periodic immunizations against common infectious diseases.
4. Dispensary service.
5. Maternity service (ante natal, natal and post natal).
6. Family planning advice.
7. Health education through talks, demonstrations and film shows.
8. Home visits by the doctors and social workers.
9. Recreational facilities for all age groups and both sexes together with vocational training.
10. Day nursery for children of working mothers.

There are about 350 families getting the services offered at the centre.

In addition to the services to the member families, the dispensary, maternity and recreational facilities are thrown open to the public and the centre is being used as a teaching and training centre for personnel in the medical and social fields.

Lately a peripheral wing of the Pediatrics Dept., of the General Hospital has been started for the benefit of sick children.

During the 10 years of its existence the centre has been aiming at optimum standard of health among its family members. More than 1200 families have had the benefit of the services rendered. The centre lays stress on the preventive aspect of health service and instills the members to cultivate and promote health. Due to the health education and frequent contact of the members very few of them have had serious illness. The Health Check-ups revealed very many early diseases especially pulmonary tuberculosis, kala-azar, nutritional defects and worm infestations. Arrangements were made either at the centre or in other hospitals for treatment of the above cases.

The recreation sections have been of use to the members in usefully occupying their leisure hours and at

the same time preventing them from taking anti-social activities. The recreation section also provides facilities for vocational training like book-binding, sewing, needle work. Quite a few have been trained in the above vocations. The centre have also developed a histrionic section where members stage dramas and farces from time to time.

Creche

The creche has a capacity to admit children between the age groups of 3 months and 5 years of working mothers. This service is also extended to mothers who are chronically ill and to widowers' children.

The children are provided with daily bath, change of dress, food three times, nursery education, medical aid, rest and recreation. This is a very popular section and the children are given care and affection. At the end of the 6th year they are admitted in schools or institutions.

Finally the centre helps the members in securing jobs, admissions to schools, boarding in Institutions etc.

The one striking feature that is worthy of mention about the families of this centre is their community action either for work connected with the centre or any help required by a family or individual. Thus the centre is achieving in slow degrees the object with which it was started, *viz.,* to promote health, physical, mental and social among its member families.

MANAGEMENT OF THE OBESE UNCOMPLICATED DIABETIC

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A person who is more than 10% above the "Standard" weight for people of the same age, sex and race is considered to be Obese. Obesity is graded as mild, moderate and severe. The life expectancy of a man 25 lbs. over-weight at the age of 45 is said to be 25% less in years than that of a healthy man of the same age. The hazards involved in being obese are many :—one of which is Diabetes Mellitus. In diabetic clinics it may be noticed that the majority of the patients are obese. So it follows that all the complications of obesity such as degenerative vascular disorders, gall bladder disease, Bronchitis, Arthritis, etc., are likely to affect this type of Diabetic patient in addition to the complications of Diabetes Mellitus itself.

Diagnosis

Usually the Diabetic comes to the doctor when he develops one of the complications and that is the time when Diabetes Mellitus is diagnosed. A few of the patients may be detected during routine medical check-ups. The majority of the unknown asymptomatic Diabetic patients, however, cannot be uncovered by the routine urine analysis and Blood Sugar Estimations. An obese patient, or one with a family history of Diabetes should suggest the possibility of an undetected Diabetes. Glycosuria in the past, Glycosuria during pregnancy or during the course of an infection may give a clue to the diagnosis. Duncan suggested that a concentration of sugar in a specimen of patient's blood generally taken after 8 to 14 hour fasting in excess of 130 mgm. per 100 c.c. or a value exceeding 170 mgm. after a hearty meal is usually due to Diabetes. Blood Sugar value above 140 mgm. *two hours* after the ingestion of 100 grammes of glucose using the technique of Standard Glucose Tolerance Test will also reveal Diabetes. A value below 120 mgm. at the end of two-hour period is definite evidence against the diagnosis. If the Blood Sugar value falls between 120 and 140 at the end of the two-hour period, the diagnosis should be reconsidered and the test done again after two weeks.

The Glucose Tolerance Test should be preceded by a normal diet including 300 grammes or more of carbohydrates for at least three days. In the absence of such preparation, the liver may put forth more sugar into blood than usual and a false positive "Diabetic Type" of Glucose Tolerance Curve may be obtained. Soskin demonstrated by animal experimentation *that it is not* the Pancreas, but

rather the Liver which is essential to the regulating mechanism responsible for the normal Glucose Tolerance Test.

The Carbohydrate metabolism should be studied in the light of :

(*a*) Presence or absence of Hepatic disease.
(*b*) Any infections.
(*c*) Pregnancy.
(*d*) Thyro-toxicosis.
(*e*) Intra-cranial lesions.
(*f*) Hyperpituitarism or Hyper Adreno Cortical activity
and
(*g*) Psychogenic Dysfunction.

MANAGEMENT

The obese Diabetic usually has a mild form of the disease unless he has been over-eating and taking Insulin treatment. These diabetics do not require insulin. The main objectives should be to reduce weight by adequate control of the diet. Whether it is a severe or mild type, if the person is poorly controlled, complications may set in. The benefit of a low calory diet and weight reduction in the management of obese diabetics cannot be over-emphasized. This would mean that there is practically no difference between the management of the over-weight diabetic without acute complications and that of the over-weight person without diabetes. However it should be borne in mind that in persons having acute complications the diet should be provided with adequate calories to maintain body weight and insulin should be employed in dosage sufficient to control the diabetes as long as there is danger of Acidosis. The ideal diabetic weight is 5 to 10% below the standard weight. The obese diabetic seldom goes into acidosis.

The principle of the dietary management is :

(*a*) To provide less calories than his metabolism would demand.
(*b*) Sufficient Protein to prevent endogenous protein breakdown.
(*c*) Adequate fat for palatability.
(*d*) Sufficient minerals and vitamins.
   (1) To obtain the total calories required standard age and height tables should be referred to, which will give the body weight in lbs.
   (2) This standard body weight is to be multiplied by 10 to give the calories required.
   (3) If physical activity is less no further adjustment is necessary—for greater activity 20 to 25% of calories may have to be added.

Recent trends are towards a more liberal allowance of carbo-hydrates and more restriction of fat particularly animal fat. A diet providing less than 40 grammes of fat may not be palatable enough for the patient. 50% of the total calories should be provided by carbo-hydrates up to the maximum of 200 grammes. The proteins should make up 15 to 20% of the total calories and the rest of the calories should be provided by fats. The 24 hour allowance of calories may be distributed in a manner most suitable to the patient's circumstances. It is always wise to give in writing the specifications.

Vitamins and Minerals

I do not wish to stress the need for vitamins because at the moment I see that there is a tendency for giving too much vitamins. I imagine that most of these will be running down the drain, the excess being excreted by the patient in his urine. I should mention something about minerals. Any patient with Polyuria loses salt. If there is polyuria, over-enthusiasm to limit salt intake may act adversely. In the face of diabetic coma if the patient's kidneys are excreting, some Potassium Citras by mouth should be given.

The place of insulin in the case of the Obese Diabetic

Though the obese diabetic does not require insulin ordinarily, the patient should be warned that during an acute illness, the urine should be tested daily for sugar and advice obtained from the physician. If he develops acidosis, insulin must be administered in adequate dosage. When the acute infection has subsided, Insulin can be stopped and he may go back to the dietetic regime.

• The diabetic who is only slightly over-weight at the onset of his disease is more likely to need insulin than the person with severe obesity. In this case a single small dose of intermediate insulin given before breakfast each morning will suffice.

Drugs: Drugs aimed at weight reduction namely Thyroid and Amphetamines are practically valueless. On the other hand they may do harm occasionally.

Carbutamide (BZ 55 "Invenol" in "Nadisan") : This is a Sulphonamide used by a number of people since the last three years. Edinburgh, Wittington Hospital, London, Kings College Hospital, and Middlesex Hospital in London have published reports after using the drugs extensively. In general the reports are not very encouraging. It is clear that this drug can be used only on a diabetic over the age of 40, obese in type and free from any complications. The degree of Diabetes should be mild. Prolonged use of the drug has been known to produce toxic side reactions some of which fatal such as Blood Dyscrasias and allergic in response.

Tolbutamide (D. 860, "Rastinon" etc.) : This is also a *sulphonamide* but differing from Bz 55 in having a Methyl Group *instead of Amino* Group on the Benzene ring and therefore has *less* tendency *to* produce Blood Dyscrasias. It is excreted in the urine as *oxidation product* which may give a false positive reaction in the the Sulpho-salicylic acid test for Proteinurea. Its mode of action is *still not certain.* It however lowers the blood sugar in many mild, stable, *middle-aged* diabetics. Some of these patients who need insulin to keep the Blood Sugar at a reasonable level may be able to dispense with injections and take tablets instead. But unfortunately while lowering the Blood Sugar they do not correct the other metabolic disturbances of diabetes. The British and American experience suggests that Bz 55 is a dangerous substance unsuitable for long term control of Diabetes Mellitus. D. 860 may prove safer but prolonged observation may be needed.

Hyperglycaemia in obese diabetics can also be controlled by Tolbutamide, but it will slowly increase the

obesity. The correct treatment for such persons is dietary restriction which by itself will correct the obesity and restore Blood Sugar to normal levels. The dose of Tolbutamide ranges from 1 to 4.5 grammes in divided doses. No one dose should exceed 1.5 grammes. It is better to be taken after food instead of in empty stomach.

Hospitalisation : Although it is not essential to admit a patient in the hospital for weight reduction, it may be profitable to have him in the hospital for the first 4 or 5 days, thus removing him completely from the pleasures and pressures of his usual life.

Psychogenic Aspect : Attempts should be made to investigate why the obese patient over-indulges in eating. A patient listening and sympathetic understanding will be necessary. Some of these obese people may have emotional disturbances and may be quite unhappy inwardly. Try and explain to them the advantage of reducing weight and try to get their cooperation.

References :

1. Joslin, E. P. and others, "*Treatment of Diabetes Mellitus.*"

2. Duncan, G. G., "*Diabetes Mellitus*", W. B. Saunders Co., Philadelphia.

3. Prof. Dunlop, D. M. and others, *B.M.J.*, page 433, dated Aug. 25, 1956.

4. Arnold Bloom, M.D., M.R.C.P. and others, *B.M.J.*, page 440, dated Aug. 1956.

5. H. E. Holling and others, *B.M.J.*, page 325, dated Aug. 10, 1956.

6. *B.M.J.*, Editorial page 466, dated Aug. 25, 1956.

7. *B.M.J.*, 1957, page 343.

MODERN TREATMENT OF LEPROSY

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Leprosy is one among the few diseases that had baffled medical men of all countries and of all ages so much so the treatment of this dreaded disease has remained an unsolved problem till very recently *i.e.*, the second decade of this century. The treatment of this disease has been so difficult and the results achieved so poor that when one goes into the history of the previous methods of treatment of this disease, one is amused at the various drugs that were used in the past. Even an eminent Leprologist like Dr. R. G. Cochrane states "Probably there is no disease which calls for more ingenuity in the matter of treatment on the part of the physician than Leprosy" (Practical Text-Book of Leprosy by Dr. R. G. Cochrane). So long as there is no specific treatment for a disease, it ceases to interest the medical men and becomes a humanitarian problem. Hence it is, that all the pioneering work in the relief of this section of the community had been left in the past to the Missionaries.

It will not be out of place here for me to make a passing reference to the various unsuccessful methods of 'Treatment' that were 'inflicted' on the unfortunate patient of leprosy in the past and hence the disease remained an 'Enigma' till the advent of the sulphones in the second decade of this century.

During the end of the 19th century Radcliffe Crocker advocated the use of Mercurial preparations. It will be interesting to note that many Indian Indigenous remedies used in Leprosy contain Mercury.

Potassium Iodide

This does not do any good to any patient except to precipitate dangerous reactions and consequently damage to vital organs and nerves.

Arsenic

The earlier physician notably Danielssen used it in the form of Fowler's solution. Hasson (1922) reintroduced it but it fell again into disfavour.

Antimony

Antimony was being used even as recently as 1925 by Rodriguez and Eubanus and Cawston (1920). It is found to be of no value for the treatment of the disease per se but still it is being used for the treatment of lepra reaction.

Copper

Copper which was also being used in colloidal form has now been given up completely as it was found to be of no value.

Sera and vaccines

In 1890 Carrasquilla advocated serum therapy. Needless to say, no sera or vaccine has ever been found

to be of use in the treatment of this disease.

Dyes (Trypan Blue, Brilliant Green etc.)

These drugs were used presumably due to their Antiseptic properties, but they have also been found to be of no value.

Sulphonamides

Sulphonamides which revolutioned the treatment of pyogenic infections in the Thirties of this century has also been found to be useless in the treatment of this disease.

Hydnocarpus oil

Hydnocarpus oil came into the arena of treatment of leprosy during the earlier part of this century, which can be regarded as a definite step forward though not a satisfactory achievement. Hydnocarpus oil was used parenterally [Intra muscular injection as well as into the Macules (intradermal)]. The results of the therapy were very slow and the relapse rate was very high and hence was not found to be as satisfactory as it was expected to be.

The Modern Treatment of Leprosy is with Sulphones

The sulphone era of Leprosy treatment which dawned very recently (1943) has revolutionised the treatment of the disease and has brought hope and cheer to the many patients who were steeped in despair. Incidentally with a specific treatment for the disease, the interest of medical men in the disease has increased and so, nowadays, Leprosy has come into the purview of medical man (of course humanitarian agencies have still their part in giving relief to the social and economic problems which many of these patients still face).

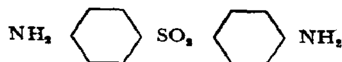
Introduction

The modern treatment of Leprosy with Sulphones was ushered in by Faget in 1943 in Carville, America. The first sulphone (derivative of the parent drug D.D.S.) used in Leprosy was Promin by Faget et all at Carville Leprosorium. The drug was given as a daily intravenous injection in doses of 2 to 5 gm. for a prolonged period and it was observed that the result achieved with this drug was better than with any drug used before.

Subsequently other workers *i.e.*, John Lowe in Nigeria, Sowza Lima in Brazil, Fernandez in Argentina, Molesworth in Malaya, Muir, Cochrane and Dharmendra in India worked with these drugs and all workers came to the unanimous conclusion that the sulphones are the best known treatment of leprosy. Thus the sulphones have become firmly established in the treatment of leprosy and it has been truly said of the sulphones that "they are no longer mystery drugs nor yet miracle drugs".

Pharmacology of Sulphones

The parent sulphone is 4 : 4—Di-amino Diphenyl Sulphone (D.A.D.P.S. or D.D.S. for short) which was first synthesised in 1908 in Germany by Fromin and Witmann and was subjected to animal experiments by Buttle et all in 1937. D.A.D.P.S. is represented chemically as follows :



They found D.D.S. effective against Streptococci and also against B. Tuberculosis. In 1939 Rist also reported from Paris that the drug was found

to inhibit the growth of human and Avian Tubercle Bacilli in Vitro. One should remember that since the Lepra bacilli cannot be cultured, experiments in Vitro with this Bacillus cannot be made with any drug. This inability to culture the organism outside the human body has been a stumbling block in the advancement of our knowledge of the disease. But unfortunately the drug was found to be too toxic for human use and also relatively insoluble. This lead to further investigations to find out a derivative of the parent sulphones which would be less toxic but yet retain the Therapeutic activity. Thus came into existence a series of derivatives of D.D.S. namely Promin, Diasone, Sulphetrone, Promozole etc. Promin is the first derivative of D.A.D.P.S. which was used in the treatment of Leprosy at Carville by Faget et all (1943) who gave a preliminary report which was very encouraging and thus began the Sulphone era of Leprosy. Next year E. Muir published his report about the use of Diasone in the treatment of leprosy which was found to be very good. Then came the introduction of a soluble preparation Sulphetrone by Buttle which was found to be relatively non-toxic and an equally effective preparation. Subsequently John Lowe in Nigeria and Dharmendra in India proved that the parent compound D.D.S. is itself quite safe if the dose of the drug is kept low. Thus at the end, the parent drug which was once regarded as too toxic has been brought back into the treatment of leprosy and now it is established as quite a safe and effective method of treatment if the dose of the drug is kept within certain limits.

Mode of action of sulphones in leprosy

Though we know that sulphones are definitely effective in Leprosy still the precise method of action of the sulphones is not clear. Some workers believe that these drugs are bactericidal to leprosy bacilli as sulphonamides to the Pyogenic Bacteria. It is with this idea that the dose is increased as quickly as possible so that there is an optimum blood concentration for effective action against the bacilli. Yet some others have injected the drug so that the drug may have a direct lethal action against the bacilli in the skin. If it is to be regarded as bactericidal we should be able to say at what dose and at what blood level the drug is bactericidal and the period of treatment required for effecting the death of the bacilli and also be able to prove that the bacilli are dead. We are still not clear about these points. Hence there is difference of opinion among the leprosy workers as to the exact nature of the mode of action of these drugs in Leprosy. It is found that though clinical improvement is rapid, the bacteriological improvement is slow. Johensen states that this slow disappearance of the bacilli shows that the sulphones have no direct bactericidal action on the bacilli. Muir said that sulphones are effective in leprosy by their power of preventing the origin of new lesions. Cochrane thinks that the sulphones possibly alter the environment of the M. Leprae so that the tissues become unfavourable to its adequate growth and multiplication. Under these adverse conditions the bacilli assume the Granular form, the exact significance of this form of the bacilli is still not understood. J. Low said that the granular form of Lepra

bacilli achieved with sulphone treatment may be taken as degeneration of the bacilli, though not, as death of the bacilli. Also if the bacilli are killed by sulphones, then the drug should also be expected to be prophylactic. But then, there is no unanimity of opinion about this also. Hence, suffice it to say that the drug is effective in Leprosy and so has come to stay, till, probably a still better drug comes into the arena of the treatment of Leprosy.

Effects of treatment

(a) *In a Lepromatous case* :

Bacteriological changes : The bacilli in a patient before treatment are acid-fast rods seen in clusters ("cigar bundles") and in advanced cases form Gloea. After the administration of sulphones in course of time, the bacilli begin to lose their "rod shape". They begin to fragment and ultimately become granular. Even the striking 'acid-fastness' of the bacilli also becomes less, so that at the end of a few years treatment (2 to 3 years) they are seen as acidfast granules. Continuing the treatment still further leads ultimately to the disappearance of the acid-fast granules also from the skin. Cochrane states that when this state is reached, the bacilli are still found in the subcutaneous nerves. The patient who can be regarded as really cured of the disease, should be able to get rid of the bacilli even from these subcutaneous nerves. The question, now arises, as to the exact nature of these acidfast granules seen after treatment with sulphones. We are not sure whether these granules represent an attenuated form of the bacilli or whether they mean the death of the bacilli. One is not sure, about this because a few patients who became negative bacteriologically after sulphone treatment and had stopped the treatment, had subsequently developed lepra reaction and these patients broke down after reaction with plenty of lepra bacilli of a normal acid-fastness and rod shape. This feature of the disease is confusing —nay makes us come to the conclusion that the treatment should be continued on a maintenance dosage for ever.

Clinical effects

Clinical effects of sulphone therapy are striking and very encouraging. The clinical improvement is so rapid that the bacteriological improvement does not keep pace with it.

As soon as a patient is started on sulphones the first change noted in a patient is that the skin loses its excessive oily nature and becomes drier. Then the nodules become flattened, then subside and later on completely disappear.

The 'Nasal ulcers' heal up and consequently there is cessation of Epistaxis and relief of nasal congestion.

The infiltration of the skin begins to disappear. In the course of say 3 to 4 years of continuous treatment, the patient who had a hideous look due to nodules etc., is now cleared of these lesion and he looks almost normal. The clinical improvement with sulphones is so good that after a few years of treatment with this drug, the patient looks almost normal except that the bacteriological improvement is not in proportion to the clinical improvement.

(b) *Non-Lepromatous Leprosy* :

The Macules fade away after a few years of treatment. The Neuritis unless

very advanced also subsides. The origin of new lesions is prevented. The Anaesthesia if due to damage to big nerves cannot be recovered from and trophic ulcers do not improve with sulphones.

Cases suitable for sulphone therapy

When the sulphones were first introduced in the treatment of leprosy it was first tried in the malignant type *i.e.*, lepromatous type and was found to be very useful. As regards the non-lepromatous cases, Cochrane was of the opinion that the sulphones were not only of no use in these cases, but may even lead to dangerous complications by precipitating severe reactions. But then, subsequent investigations have proved these views as being untenable. Nowadays, sulphones are used for both Lepromatous and Non-Lepromatous cases and it is found that they are beneficial in the Non-lepromas also.

I will now consider *et seriatum* the method of treatment of leprosy with various sulphones.

Promin : (Chemical name : Didextrosesodiumsulphonate) Trade name : Promin.

This is a derivative of sulphone which was the first drug used in the treatment of leprosy by Faget et all (1943).

Mode of administration : Daily intravenous injections over a prolonged period. Dose 2 to 5 gms. (in 5 c.c. solution) daily for 6 days a week. As this method of treatment is tedious, cumbersome and practically difficult it has been given up long ago and is now only of historical importance.

Sulphetrone : (Chemical name : Tetrasodium diphenylsulphone tetrasulphonate) Trade name : Solapsone.

One of the derivatives of D.A.D.P.S. which is relatively non-toxic, cheap, effective and quite a safe one, which could be given to those who are weak and who may not tolerate the parent drug D.D.S.

It is marketted as follows :

Trade name.	Manufacturer.	Packing.	Method of treatment.
Solapsone			
Tablets.	Burroughs Welcome & Co. Ltd.	100 tablets.	Oral.
Granules.	Do.	500 gm and 1000 gm tins.	50 per cent Aqueous solution for injection.
50 per cent solution.	Do.	20 c.c. vials.	Injection.
Novotrone			
Tablets.	Bengal Chemical and Pharmaceuticals Ltd.	—	Oral.
Granules.	Do.	100 gm tins	50 per cent Aqueous solution for injection.

Mode of administration : Oral and injections.

Orally it is started on 3 tablets a day and the dose is then gradually increased to 6 tablets a day. 90% of the drug given orally is not absorbed and the cost of the treatment is quite high and so oral treatment has been given up. Parenteral route is the ideal method of administration of this drug.

Sulphetrone is given Parenterally (Intramuscular injections) twice a week as a 50% Aq. solution.

Dose: Started with ½ c.c. twice a week, increased by ½ c.c. every week till a maximum dose of 3 c.c. twice a week is given or 6 c.c. a week.

This method of treatment had been tried in a selected group of 35 patients in this centre for 6 years and found to be effective and quite safe. (Refer 'Six years of Aq. Sulphetrone Therapy in a rural area' by Dr. V. Ekambaram, M.B.B.S.—Leprosy Review, London—April 1958).

Preparation of Aq. Sulphetrone for injections

Preparation used

Sulphetrone granules	.. Burroughs Welcome & Co. Ltd.	Supplied in 500 and 1000 gm. tins.
Novotrone granules	.. Bengal Chemicals and Pharmaceuticals Ltd.	Supplied in 500 and 1000 gm. tins.

The solution used is of a 50% strength and is made up as *follows*:

50 gms. of Sulphetrone granules are dissolved in hot distilled water and the final volume made up to 100 ml. No preservative is used. This solution is then placed for 10 minutes in boiling water bath as a substitute for autoclaving, as no facilities exist in this centre for autoclaving. This solution is acid in reaction and gives rise to excessive burning sensation at the site of the injection, hence is neutralized by the addition of sodium carbonate (about 1.5 gm. of sodium carbonate to 1 litre of the solution). The solution is then transferred to a sterilized transfusion bottle from which it is drawn into a syringe for injections.

To avoid deterioration, only about 8 ozs. of the solution are prepared at a time so that the solution is not used for more than two weeks before a fresh solution is made. The period of treatment with any drug for Leprosy is not definite. Usually it is between 3 to 5 years.

Diasone: (Chemical name: Disodiumformaldehydesulphoxylate diaminodiphenyl sulphone).

Diasone is another derivative of D.D.S. It is marketted in the form of tablets by Abbot Laboratory. Dr. Muir found Diasone to be very effective and relatively less toxic.

Method of administration: Oral.

Dose: *1 tablet 3 times a day.*

Period of treatment: Prolonged.

Comments: This drug is better absorbed than Sulphetrone but then the cost of treatment is very high (Rs. 40 per bottle of 100 tablets). This is only of historical interest as this drug has also been given up in the treatment of leprosy.

I have described the use of the various derivatives of the parent sulphone in the *treatment of leprosy, but* then since the parent sulphone itself has now been found to be quite safe, effective and cheap, the standard treatment of leprosy is now with D.A.D.P.S. I shall now describe the methods of treatment with D.D.S. (D.A.D.P.S.).

D.A.D.P.S. or D.D.S. for short

Diaminodephenyle sulphone is the parent Sulphone from which all other drugs mentioned above are derived. It is also thought that most of the derivatives of sulphone act by virtue of their being converted into D.D.S. in the Alimentary Canal. D.A.D.P.S. is marketted as follows by the following companies.

Name of firm	Trade name.	Packing	Mode of administration.
Imperial Chemical Industries ..	Avlosulphone Tablets —	100 mgm.	Oral.
	„ Powder —	100 gm. tins	25 per cent suspension in oil for injections.
Bengal Chemical and Pharmaceuticals Ltd. ..	Novophone Tablets —	50 mgm.	Oral.
	Novophone Y (with Yeast) Tablets —	50 mgm.	Oral.
	Novophone powder —	100 gm tins.	25 per cent suspension in oil for injections.
Albert David Co. ..	Siosulphone tablets —	25 mgm.	Oral.
Burroughs Welcome & Co., Ltd. ..	Dapsone tablets —	100 mgm.	Oral.
Biddle Sawyer & Co., Ltd. ..	Croysulphone tablets —	100 mgm.	Oral.

Mode of administration

(*a*) *Oral*: The tablets are given orally. The administration of D.D.S. has been found to be quite safe and very effective provided judicious care is taken as regards the dose and other aspects. This drug has now become so popular that it has become the sheet anchor in the treatment of leprosy.

The tablets can be given by the daily method or bi-weekly method. In the daily method the patient should take the prescribed dose every day after food.

In the bi-weekly method the required dose for the whole week is taken on 2 days in a week say Tuesday and Saturday. In practice in rural areas, it is not possible to get the patient to come for treatment more often than once a week even which is difficult. Hence if a patient is on the daily dose method, the total dose of the week is given to the patient when he attends the clinic with instructions to take the drug every day as advised. If he is on the bi-weekly method, half the weekly dose is administered to the patient the day he attends the clinic and he is asked to take the remaining half 3 days later. In practice, the bi-weekly method is found to be good for rural patients as the chances of their loosing the tablets or forgetting to take the medicines or over-dosing themselves is less by this method as ½ of the weekly dose is directly administered to the patient by the doctor at the clinic.

Dosage: *Oral.* Start with a small dose say 25 mgm. a day for about a month and then if there are no untoward signs or symptoms increase the dose to 50 mgm. a day and continue this dosage for about a month. Then if the patient still does not show any untoward signs or symptoms increase the dose gradually to 100 mgm. a day for 6 days a week *i.e.*, 600 mgm. a week for ever afterwards till the patient becomes bacteriologically negative in a Leproma and the clinical lesions disappear and remain so at least for a year in non-lepromatous case. (Neural leprosy). *This should not be exceeded in the case of Indian patients.* As regards children (under 12 years) half this dose should be given.

The dosage schedule of the bi-weekly method is as follows:

50 mgm. a week for 3 weeks.
100 do.
150 do.
200 do.
300 mgm. a week for always.

The above is a schedule of treatment outlined for patients when they exhibit no untoward signs or symptoms *during the sulphone therapy.* But *if* any of the complications mentioned are found (see heading "Complication from treatment") then sulphones should be stopped temporarily and should only be started after the complications have been treated.

It should also be borne in mind; that in general Lepromatous cases and reacting neural cases should be started on smaller dosage and the dose worked up very gradually.

D.D.S. by Parenteral route has also been tried and is still in vogue in the hands of the French workers in West Africa.

Method of administration

D.D.S. powder being insoluble in water or oil, a 25% suspension is prepared in Arachis oil or coconut oil. This oily suspension is given as a deep intra muscular injection once a week.

Dose

Start with 1 *c.c.* once a week and increase dose gradually to 4 c.c. But in practice, I found that the safe dose is 2 c.c. a week for our patients.

Relative advantages and disadvantages of oral and parenteral methods of treatment with D.D.S.

Oral	*Injection*
1. Easy to administer in field clinics as it does not require any appliances for injections (specially in rural areas).	1. Difficult and cumbersome for work in field areas as it requires facilities for injections—not available in rural clinics where a large area is covered by mobile clinics.
2. The patients are liable to loose, over-dose and forget to take the tablets.	2. As the drug is directly put into the tissues of the patient, the dangers of loosing or misusing does not arise.

Oral	*Injection*
3. The patients are not impressed with such oral treatment (small tablets for a big disease!) especially so in a rural area. Hence likelihood of irregularity and falling into the hands of Quacks who promise miraculous cures with costly injections (usually coconut oil pumped into the credulous patient).	3. The patients are very impressed with injections and so, are more regular for treatment.
4. There is no danger of abscesses from this treatment as it is oral	4. There is always the danger of injection abscesses and consequent suffering to the patient.
5. The drug given orally is comparatively quickly eliminated from the body.	5. Due to the formation of a Depot by this method, the patient gets the benefit of the drug for a prolonged period.
6. In case of lepra reaction or Anaemia or Sensitisation Dermatitis, the patient can be advised to stop D.D.S. and thus grave dangers can be avoided.	6. Unless a medical man supervises the treatment and unless he is very careful once an injection is given to a patient, the complications which may arise later may become worse due to the Depot or the drug already in body.
7. The relapse rate question is controversial subject and the question of relapse, in a disease in which we are still not sure about the period of treatment required, does not arise.	7. A few workers state that the relapse rate is very low with this method.

In the end I should state that D.D.S. oral is preferable for mass therapy among rural folk for centres like this which work under very difficult conditions (the clinics are held under the shade of trees mostly, as no buildings are available in many of these areas). Also the results achieved are very good with oral D.D.S.

The period of treatment required depends upon the type of the case treated. In lepromatous cases the present consensus of opinion is that it takes any period from 3 to 7 years for a patient to become clinically and bacteriologically negative. But as we are still not sure that there is no danger of relapse, it is wise to continue the treatment on a maintenance dose say 200 to 300 mgm. a week for ever. In non-lepromatous cases, the lesions disappear unusually in about 2 to 3

years. But then again it is wise to continue the treatment for a year after all the signs and symptoms have disappeared. During this period it is better to keep him on a full dose and then to tail off the dose gradually to a maintenance dose of say about 100 mgm. a week. Remember it is always wiser to keep a patient of leprosy under observation. Better err on the safe side!

Other drugs used in Leprosy

I have described the modern routine treatment of leprosy. The following drugs are also used in leprosy under certain circumstances.

Thiosemicarbasone

This drug was first used by Gordon Ryrie and very good results have been reported by him. Since then other investigators (Dharmendra) have tried the drug with equally good results. But later workers have not found the drug so effective as to replace the sulphones. The present consensus of opinion is that this drug is useful when the patient does not tolerate sulphones or when he gets repeated lepra reaction etc. with sulphone therapy. In such cases Thiosemicarbasone is a good drug. Also in the reactional states of leprosy like the Border line type or the reacting Tuberculoid, Thiosemicarbasone is a good drug. But it is always safe to change over to sulphone once the reactional state is tided over. It is marketted as follows:

Trade name.	Name of firm.	Packing.	Mode of administration.
Thiacetamide	.. Boots Pure Drug Co. Ltd.	25 mgm. tablets	Oral— 25 mgm. a day for 1 week.
Contiben	.. Bayer & Co.	Do	Oral— 50 mgm. for 1 week 75 mgm. for 1 week
Siocarbasone	.. Albert David, Ltd.	Do.	Oral—100 mgm. for a week. 150 mgm. a week thereafter.

The dosage schedule given above is a little rapid for our patients and hence it is wise to keep a patient in one dose for 2 weeks before his dose is increased *i.e.*, 1 tablet a week for 2 weeks, 2 tablets a day for 2 weeks etc.

Isonicotinic Acid Hydraside

This drug was tried by many workers and the first reports were encouraging but then it has not stood the test of further trial. It is not very much used now except in certain cases where the patients do not tolerate sulphones. As a matter of fact, I.N.H. orally with sulphetrone parenterally is under trial in this centre for patients in reactional states of leprosy. The results cannot be assessed now but suffice it to say, that it appears to be promising.

Dose: 50 mgm. 3 times a day.

Method: Oral.

It is always better to give Nicotinic acid along with the drug.

Streptomycin

Streptomycin is reported to be good for reactional states of leprosy. Cochrane *et all* have reported very recently of the good results achieved by a combined treatment of I.N.H. with Strepto-

mycin. It should be borne in mind that more time is required before any conclusions are arrived at about the use of these drugs.

Cortisone

This is not useful for the treatment of disease per-se but has been found to be very efficacious in the treatment of lepra reaction when other routine measures fail.

After having described the modern treatment of leprosy, I shall now state in brief the complications of leprosy and the treatment thereof. The complications of leprosy in the lepromatous type is discussed first and then the complications of the non-lepromatous type (only complications which are still seen even with Sulphone Therapy).

Complications—Lepromatous type

Lepra reaction is a very common complication that occurs in the lepromatous type with or without treatment. It is still vaguely understood but every one is agreed that it is a kind of toxic phenomenon arising out of the breakdown of the leprotic foci and consequently the patient is very ill and so requires rest and careful treatment.

It is characterised by fever coming on every evening with multiple subcutaneous nodules all over the body or sometimes Roseolar evanascent rashes. There is very severe Arthralgia of the big joints and sometimes very painful Neuritis of the peripheral nerves *e.g.*, ulnar or peroneal. Yet sometimes eye complications may also be precipitated by a lepra reaction.

Treatment

1. Sulphones should be stopped.
2. The patient should have absolute rest in bed.
3. The bowels should be kept open with salines.
4. He is given an alkaline mixture or Mist. Soda Salicylas and Aspirin or Codopyrin for relief of pain.

After a few days if the condition does not subside with this treatment, he is given intra-venous injection of Potassium Antimony Tartrate on alternate days for 6 injections as follows :

1st day	P.A.T	02 gm	dissolved in 5 c c. of distilled water or Glucose 10 c.c.
3rd day	,,	.02 gm.	do.
5th day	,,	.02 gm.	do.
7th day	,,	04 gm	do.
9th day	,,	.04 gm	do.
11th day	,,	04 gm.	do.

P.A.T. is marketted by Smith Stanistreet & Co., Calcutta in the form of tablets .04 gm. each.

In case, the lepra reaction does not subside even with this, then Cortisone effects a miraculous improvement.

Neuritis

Neuritis of peripheral nerve trunks *i.e.* peroneal and ulnar does occur sometimes as a manifestation of lepra reaction when it should be treated as for lepra reaction. In case there is no improvement with this method, then injection of 75% solution of Alcohol ms. 3 to 5 directly into the nerve sheath or deep X-ray treatment (say not more than 6 exposures) or injection

of adrenalin (m. 10 in m. 30 of saline) or Ephedrine may be tried. In some cases I have seen great relief of the Neuritis by desheathing of the nerves despite the fact that many text books state that desheathing is not of much use in this type of leprosy.

Lesions of the eye, nose and larynx are also met with in lepromatous leprosy.

Eye complications consist of either generalised or localised lesions. Generalised lesions take the form of diffuse Episcleritis with redness, watering and pain of the eyes.

Circumscribed lesion takes the form of a lepromatous nodule in the Sclera. The cornea may also be affected with the production of a superficial Keratitis, Pannus formation, deep Karatitis and corneal ulceration. The Iris may get involved with the production of Iritis, subacute Iritis or chronic Iritis and the inflammation may spread deeper and may result in Irido cyclitis. Opacity of the lens with cataract formation has also been observed in leprosy. It is now understood how many of these untreated cases of leprosy become blind due to the ravages of the disease.

Treatment of eye lesions

One should remember that most of these complications are very rarely seen nowadays with sulphone therapy. A few neglected cases still come with these complications and I am indicating below the general principles of the treatment of these conditions as it is not possible to write in detail about this in this short article.

Epi-Scleritis

Irrigate the eye with saline and apply Cibazol or Aureomycin eye ointment.

In circumscribed l e p r o m a tous nodule, I have found sulphone therapy give very poor results. The older physicians advocate excision under local Anaesthesia.

Iritis and Iridocyclitis are still seen occasionally. The principle of treatment of this condition is as follows:

1. Treat as for reaction if these are signs of reaction.
2. Give Aspirin or Codopyrin for the relief of pain.
3. Locally Atropine eye ointment does give a lot of relief.
4. Lately cortisone eye ointment has been found to be very good.
5. Dark glasses or an eye shade for the relief of photophobia.

Keratitis is seen very rarely nowadays.

The treatment advocated in olden days was Dionine (2% solution) locally, increased gradually to 4%. Subconjunctival injections of saline starting from 2 c.c. gradually increased by week till 1 c.c. a week is given. Lesions of the Nose are hardly seen in patients under sulphone therapy. In untreated cases the following lesions are seen.

Nasal congestion, then ulceration, scar formation and ultimately destruction of the cartilage with depression of the nose. Epistaxis is a common symptom in these patients.

Treatment with regular sulphone therapy, the nasal condition disappears, unless too far advanced. The instilla-

tion of the following nasal drops is very useful.

Creasote	..	2 oz.	For use as nasal drops.
Camphor	..	20 grs.	
Hydnocarpus oil	..	1 oz.	
Olive oil	..	2 oz.	

Lesions of the Larynx are acute or chronic in nature resulting in hoarseness of voice and stenosis of the Larynx with consequent respiratory distress. This complication after the advent of the sulphone therapy, is not met with and hence is not discussed here.

Complications—Non-Lepromatous Leprosy

The common complications of the Non-lepromatous type of leprosy are as follows:

Reactional state does occur in a few forms of the non-lepromatous type—particularly the Tuberculoid type.

When there is reaction in a Tuberculoid type, the lesions become Erythematous, swollen and new lesions also might arise. The regional nerves also become inflamed with consequent thickening, tenderness and pain which if untreated, may result in wasting of muscle or deformities *i.e.*, contraction of fingers or drop foot. The patient may also have Pyrexia. Sometimes the reaction is so severe that the lesions may even ulcerate and eventually disappear leaving scars (Lazarine Leprosy).

Treatment

It is always wise to treat these cases very carefully. Instead of sulphones, it is better to administer during this period Thiosemicarbasone. Vitamin B complex and Vitamin B_{12} in heavy doses also help in this condition.

Muscular paralysis is very common as a complication of non-lepromatous leprosy *e.g.*, Facial Palsy, Lagophthalmus. If treatment is started early, such complications can be prevented.

Claw hand and Drop foot

Claw hand and drop foot are still seen in leprosy and they deserve mention here. These complications are due to the damage of the peripheral nerve trunks. If treatment is started as early as possible and judicious care is taken in the administration of sulphones to prevent the precipitation of reactions, these complication should not occur.

The treatment of claw hand now consists in Physiotherapy and surgical correction of the deformity.

General principles

1. Suitable advice to the patients not to engage in occupations which are likely to result in accidental injuries to the hands.
2. Keeping the joints supple by daily massage with oil and exercises and immersing the hands in a wax bath at a temperature of 40° C. for 10 minutes.

The surgical procedures now adopted for the corrections of these deformities consist of tendon transplantation operations.

Drop foot

Drop foot requires special boots and care in preventing injuries, to the foot.

Drop foot is also corrected surgically by tendon transplantation operations.

Trophic Ulcers

Trophic ulcers are perhaps the problem still unsolved in these patients and are very common in the feet. Trophic ulcers are not caused by the disease *per-se* but it is caused by a combination of factors namely the anaesthesia due to the disease and the lack of care by the patients to protect their feet from accidental injuries. Hence the first thing to be done in preventing the formation of trophic ulcers is to advise the patients to take great care of their feet by wearing proper shoes and also preventing accidental injuries.

The following principles help in the treatment of trophic ulcers:

1. Trophic ulcers without involvement of bones:
   - (a) If the ulcer is very dirty and with maggots, then dress with Turpentine for a few days till the maggots disappear.
   - (b) Next dress with *Mag. Sulph.* Glycerine till the slough clears.
   - (c) After that any antiseptic dressing which will heal the ulcer say Eusol. Rest to the part is very essential for the recovery of these ulcers.

2. Trophic ulcers with involvement of bones:

   The treatment of these ulcers is removing the bones involved and then treatment as above.

General

Some workers have had good results by dressing with dye-stuffs when the ulcers have become clean. Yet some others have advocated the encasing of the foot in Plaster of Paris for a few weeks (which actually means complete rest to the part).

We have been using Novolep a bye-product in the manufacture of D.D.S. (Bengal Chemical & Pharmaceutical Works Ltd.) for treatment of these ulcers and the results appear very encouraging.

Neuritis

Neuritis of peripheral nerve trunks *i.e.*, ulnar and peroneal are still met with which calls for energetic treatment.

The nerves are thickened, tender and very painful. The pain may be so severe as to make the patient writhe in agony.

Treatment of neuritis in non-lepromatous leprosy consists of giving anodynes, and operative procedure involving the removal of the sheath of the nerve. This operation can be done under general or local anaesthesia and it gives good results.

Complications attributable to sulphone therapy

One should remember that sulphones are not an unmixed blessing and certain complications are met with during the course of therapy with sulphones which require to be watched and treated. They are:

1. As soon as sulphones are administered to a patient minor gastro-Intestinal disturbances are felt, *i.e.*, nausea, even vomiting, a burning sensation after taking the drug but, then, these symptoms disappear after a few days.

2. *Lepra Reaction*: The nature of this condition and the treatment has already been discussed (*vide* supra). Lepra reaction is the commonest complication met with during sulphone therapy. Some workers regard lepra reaction as beneficial but there is no agreement on this among all workers. It is wise to stop the administration of sulphones during a lepra reaction.

Anaemia: D.D.S. is a toxic drug and its use may result in a Haemolytic anaemia especially when the general standard of health of the masses is already low. Hence it is wise to examine a patient and treat any anaemia he has, before starting sulphone therapy. During the course of sulphone therapy, it is better to keep a watch for any signs of anaemia and treat the same. In this connection I have to state that if the drug is given in small doses the chances of the patient developing Anaemia is very rare and so the need for any adjuvant drugs *i.e.*, Haematenics is not felt to be necessary for mass therapy with D.D.S. Due to the fear of Anaemia, in the earlier days of the trial with sulphones, regular and periodical Haemotological examinations were done, but now it is not regarded as necessary.

Treatment: The anaemia that may develop in these patients should first of all be investigated to see if there are other factors responsible for the anaemia *e.g.*, Ankylostomiasis and if so, suitably treated. The Anaemia due to D.D.S. is Haemolytic and treatment with Iron and Liver Extract quickly restores the condition to normal.

Neuritis

Neuritis may be precipitated by sulphones due to a Lepra reaction or increased reactivity in a non-leproma. Hence it is always safe to increase the dose of sulphones gradually and keep a watch on the patient during the sulphone therapy. The treatment of this condition has already been discussed.

Hepatitis

Hepatitis has also been met with in a few patients but then one is not able to put the blame completely on sulphones for this condition.

Treatment: Sulphones must be stopped during this period and treatment for Hepatitis instituted. No serious damage to the liver has been recorded with sulphone therapy.

Sulphone Sensitisation Dermatitis

This condition is akin to sulphanilamide dermatitis. The condition begins insidiously with a feeling of general discomfort and Itching and burning of the exposed parts and flexures. Then there is Erythema and then ultimately a condition similar to ex-Foliative-Dermatitis. The patient is very toxic and absorption of toxic products may lead to grave complications and sometimes a fatal termination.

Treatment: One should always be watchful of any signs of this condition in a patient under sulphone therapy as evidenced by *e.g.*, Pruritis without any apparent cause *i.e.*, Scabies or Tinea and should stop sulphones immediately any such symptoms are seen.

2. Absolute rest in bed with liquid diet.
3. Sodium Thiosulphate injections daily.

4. Vitamin C and Vitamin B_{12} injections in large doses.
5. Glucose in large doses intravenously.
6. Penicillin crystalline to prevent infection of the skin.
7. Locally zinc cream and hydnocarpus oil.

The subsequent treatment of leprosy of these patients should preferably be with Thiosemicarbasone.

D.D.S. Psychosis

As the name implies is met with only in patients under treatment with the parent sulphone.

The patient who had been on sulphones for a considerable time develops this condition. The patient is depressed mentally, does not take any interest in his surroundings and may be lost in brooding thoughts. The depression may be so bad that even attempts at suicide may be made. There are no signs of reaction or any fever.

In some the condition takes the form of an acute Mania when they are noisy, turbulent and dangerous to themselves and others.

Treatment

1. Sulphones should be stopped.
2. Glucose intravenously in large doses.
3. Vit. C - 500 mgm. a day } Parenterally.
4. Vit. B_{12} - 500 mgm. a day } Parenterally.
5. Sedation with paraldehyde or with Barbiturates.

CONCLUSION

I have in this paper described in brief the modern treatment of Leprosy with sulphones. It is no exaggeration to say that the sulphones have revolutionised the treatment of leprosy and brought light and cheer to the millions of the unfortunate sufferers of leprosy who till recently were steeped in gloom and despair. But it is wise to remember that the sulphones though very effective is still not the last word in the treatment of leprosy. Much yet remains to be solved and the Medical men should come forward to find a still better drug by which the treatment should be quicker than at present and the cure definite than what it is now. May we hope that such a Golden Age will come soon !

BEFORE TREATMENT

AFTER 9 MONTHS' TREATMENT

Note Lepromatous nodules on left arm almost subsided after 9 months' treatment c̄ D.A.D.P.S. oral

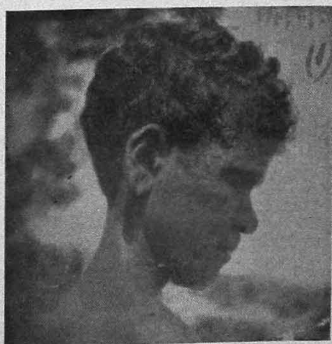
BEFORE TREATMENT

AFTER 1 YEAR TREATMENT

Note Tuberculoid Macule on left shoulder, disappeared after 1 year treatment with D.A.D.P.S. orally

BEFORE TREATMENT

AFTER 1½ YEARS' TREATMENT



Lepromatous Leprosy

Note Nodules on Ear and Cheeks with lepromatous infiltration cleared after 1½ years' treatment with D.A.D.P.S. orally

RECENT ADVANCES IN MEDICINE

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Modern medicine has developed vastly with the growth of technology, for example, space medicine. Man's desire to probe space—sputniks and baby moons—involved considerable medical research and coordination of various branches of science.

Rapid progress has been made in the fundamental researches in micro-biology, chemistry, physics (Atomic energy), mathematics etc., in relation to the maintenance of higher standards of medical education, medical practice and of medical care that we are giving for the suffering humanity. Science transcends all geographical and national boundaries. But so far as India is concerned enough progress has not been made in research. This slackness may be due to several causes. No research in any country can achieve outstanding results unless the Universities, Foundations, Private Research Institutes, Nonteaching Hospitals, Group Clinics, Industrial and Social Organisations, Scientific and Professional Societies and Indian Council of Medical Research, co-ordinate their efforts with the full support and assistance from the government. No country can keep itself aloof and idle when the science of Medicine is developing at a fast speed. The development of speedy air travel, with the radio, and television facilities, time and space have been conquered. The world looks much smaller than before. The advanced techniques adopted in the treatment of several diseases in the U.S.S.R., and in the U.S.A. are now available in our country.

The treatment of the diseases have been so revolutionised that what we are achieving today is marvellous. This achievement can be attributed to the discovery of such drugs like Chemo-therapeutic agents, the anti-biotics, the Cortisones, the Vitamins, the Hormones, Insecticides, Fungicides, Herbicides and above all the Radio active isotopes. The applicability of these 'wonder' drugs have today changed the concepts of diseases in man. The determination of the molecular structure of proteins in blood brings sight to know how disease attacks the body. The doctor, the psychiatrist, neurologist, psycho-analyst, the psychologist—all form a team to assess the proper evaluation of the disease. Thus psychosomatic medicine is establishing itself in scientific precision and now-a-days some gastric ulcers, migraine headaches etc., have both emotional and physical basis. The Industrial and Social medicine, Rehabilitation hospitals, and the War medicine, have all contributed towards the rapid development of our medical practice. The Rehabilitation centres with the Pediatric hospitals hardly exist in our country. If they are started, the children of the school going age will be prevented from their delinquency and adults from serious mental breakdown. If the patients discharged from the tuberculosis and

mental hospitals, and the injured and the crippled are properly rehabilitated, they not only retain self-respect in society, but also save the required pension, and the state would have a valuable workman. Thus our democracy will improve in all directions.

The application of the Radio-active isotopes in medicine both in the diagnosis, localisation and treatment, in agriculture and Veterinary sciences and in industry has proved a great boon. For the correct understanding of the cell division and its activity, the micro-biologist uses radio-isotopes. Radiocardiography, the blood flow in the four chambers of the heart, faulty valves, congenital defects, unborn child receiving food through the placenta, the efficiency of the human liver, the manufacturing or synthesizing the plasma proteins in health and disease can be observed with great precision. An intensive cancer research programme is carried on with the isotopes when they are incorporated in such drugs as Nucleic Acid, Adrenal Cortex hormone and the sex hormones. The nature and the cell division in cancer and the influence of the enzymes over it and the genetic and hereditary factors have considerably influenced the concept of cancer. The electron microscope for the first time made it possible to identify cancer virus *viz.* breast cancer in mice.

Approximately a hundred varieties of radioactive isotopes have been produced from 60 elements. Short lived isotopes have been produced by the Philips' Synchro-Cyclotron in the Institute of Nuclear Research in Amsterdam, which I visited recently. It may be of interest to note that the isotope of phosphorus is active for 14 days, Carbon 14, for 5,000 years and a special isotope of Pollonium lasts for a millionth of a second. Some isotopes like iodine, phosphorus, sodium etc., are supplied to practitioners in Europe by certain companies. With the isotopes cancer tumors of the thyroid, blood diseases like the Leukaemias, and Hodgkin's disease are localised and tackled.

In Tropical Medicine spectacular results are achieved in Malaria, filaria, leprosy, virus infections like small-pox, infectious diseases (water borne) like typhoid, dysentery, cholera etc. These are the problems of public health, sanitation and nutrition. There are a few Pasteur institutes in India but we require many research institutes like the one of Tropical Medicine at Calcutta.

Many advances have been made in surgery. Leaky valves in the human heart, congenital defects of the heart where the main trouble is lack of blood circulation in the lungs, coarctation of aorta, are now better treated by transplanting or grafting of the artery by Dr. Gross "artery Bank"; removing a thrombus in a heart attack by Dr. Lovis Bazy, Surgeon of St. Louis Hospital, Paris, the use of the artificial kidney, the artificial plastic heart, aneurism of the aorta repaired with plastics,—a polyethylene film—manufactured by DUPONT—are some of the phenomenal advances made. The recent knowledge acquired by us confirms that many patients with arterial disease and heart damage would have led normal lives but for the fright unnecessarily created. In certain other cases, the danger they had to face was not properly told. In the field of obstetrics and gynaecology, equally great

advances have been made. In one of the hospitals in Stuttgart in Germany with 450 beds, no post-partum haemmorage, no jaundice in the new born, and painless labour without injury to mother or child was reported. There it was felt that there was no need for the application of forceps or still less of doing a ceaserian section. This wonderful achievement is due to the anti-natal care bestowed during pregnancy and the confinement taking place in the hospital.

In radiology, striking advances are made in miniature radiography, encephalography, arteriography, deep X-ray with high penetration, and radio isotope of Cobalt, which is a very good substitute for radium.

In pharmacology vitamins, hormones, anti-convulsants, amino acids ACTH, cortisones and other cortico steroids, blood derivatives namely proteins, globulins, Petroglutamic Acid, Glutaneural, Nitrogen Mustard, hypnotics, Xylocaine and several other anesthetics have come into use. Anesthesia has become a specialised subject-freezing the heart etc., and trained men are necessary to help the Surgeon. Experimental research on heart, chest and brain have to be done, before good results could be achieved on human beings.

The pharmaceutical industry is making a rapid advance in the discovery of new and better drugs in collaboration with the research workers—medical and scientific—employed in their laboratories.

Fresh problems are arising in India as anterior poliomyelitis, nutritional diseases, tuberculosis, Mycosis etc., and with the rapid industrialization many more problems for a Surgeon—plastic or orthopaedic are bound to arise.

The medical research objectives in the Second Five Year Plan include (1) Strengthening the research facilities in existing institutions, (2) creating new research institutes for research in specific fields, (3) training of research workers by a system of fellowships, (4) providing opportunities for maximum display by the research workers, (5) creating conditions by which young and promising scientists may take research as a career, (6) Initiating programmes for the solution of the many urgent problems in the field of medicine and public health and (7) maintaining close cooperation with the national and international agencies in all matters pertaining to research.

This ambitious programme can be achieved only if all the scientists in India are grouped into an All India Service Cadre like the civil and the police, and the medical profession is nationalised.

I will conclude by referring to a comment made by Dr. Doechez on his acceptance of the Kober Medal from the Association of American Physicians, he said: "The recent rapid parade of the medical discoveries was startling". "In the past," he added, "the public had expected the physician to achieve 'miracles' ...... and now he is performing."

UPPER RESPIRATORY TRACT
VERSUS
LOWER RESPIRATORY TRACT IN DISEASES

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The two parts of the respiratory tract, the so-called upper and lower, arbitrarily divided at the level of the larynx, are closely connected in many aspects. As a rule the entire respiratory tract functions as one unit and diseases affecting the various parts of this unit have a very close inter-relationship. Developmentally both the tracts are derived from the foregut. Anatomically both are lined by the same type of ciliated epithelium and there is a continuity of structures from one end to the other. Lymphatic connections between the nose and chest have been demonstrated. There is a continuity of the air column in both these units, the air in the upper respiratory tract going into the lower respiratory tract and the air from the lower respiratory tract passing through the chambers of the upper respiratory tract, the latter being the gateway of entrance and exit for the lower respiratory tract. Physiological reflexes between these two units are well recognised. The naso-pulmonary reflex is a notable example. Sneezing and caughing, in which the entire respiratory tract reacts to a stimulus applied to the nose or pharynx, are mechanisms which demonstrate the unity of purpose underlying the working of the different units of the respiratory system.

Such being the close connection it is not difficult to understand that diseases affecting one unit produce changes in the other unit. This spread is brought about by aspiration, continuity of structures, lymphatic spread and spread by blood stream.

Aspiration of infected material from the upper respiratory tract to the lower respiratory tract is a well known phenomenon. Development of lung abscess and bronchiectasis due to aspiration of infected material in operations on upper respiratory tract as in tonsillectomy and dental extraction is an example. We are warned of the possibility of the development of lipoid pneumonia if oil or oily drops are put into the nose of infants in the treatment of common cold. These fat globules have been demonstrated in the lungs, during post-mortems on such infants. This phenomenon of aspiration is particularly fecilitated during general anaesthesia and sleep where the protective cough reflex is under abeyance.

Direct continuity of structures affords another valuable route for spread of infection from one region to another in the respiratory tract. "A cold running down the chest" is a common example in everybody's experience.

Spread of infections in the respiratory tract by lymphatics and blood stream is not widely accepted. Development of tubercular lesions in the larynx in cases of pulmonary tuberculosis is believed to be due to a spread through the lymph channels. Spread of septic thrombi and emboli from operated areas in the upper respiratory tract forming deposits in the lungs leading to the development of lung abscess is a possibility that has to be kept in the mind.

Nose is correctly described as the air-conditioner for the lungs. This function is disturbed in every disease affecting the nose. When deprived of this protective benefit of the nose as a result of nasal obstruction, the lower respiratory tract becomes an easy prey to infections.

Among the various lower respiratory tract diseases which are caused, maintained or aggravated by affections of the upper respiratory tract, bronchiectasis is a standing example. It is a common observation that many patients with bronchiectasis have some degree of chronic sinus infection associated. There may be a difference of opinion as to whether chronic sinusitis can start by itself a bronchiectasis, but there is no doubt that a chronic sinus infection maintains very effectively the bronchiectasis and frequently even aggravates it. It is aptly remarked that as long as the tap above is open, the basin below will never be empty.

Chronic sinus infection leads to frequent attacks of pulmonary infections. Infection leads to fibrosis and distortion of the bronchi with resultant loss of elasticity of the pulmonary structures. This coupled with stagnation of infected material leads to a weakness and dilatation of the bronchial tubes thus laying the foundation for the development of bronchiectasis. So chronic sinusitis may at times also be an important causative factor in the development of bronchiectasis. It is futile to treat bronchiectasis without recognising this role of chronic sinusitis in initiating and maintaining the lung condition.

It is doubtful whether a lung abscess can be produced by a chronic upper respiratory tract infection. But occurrence of lung abscess due to aspiration of infected material after operations on the upper respiratory tract has already been stressed.

An important cause of chronic tracheo-bronchitis is a chronic sinus infection. An ordinary cold when untreated or improperly treated leads to chronic sinusitis. With each flare up of the sinus infection this leads to an attack of acute tracheo-bronchitis which in time becomes chronic tracheo-bronchitis. The recognition of this entity of broncho-sinusitis is very important in the management of all chronic respiratory infections.

The role played by certain anatomical and pathological factors in the nose in the production of the syndrome of bronchial asthma is another example where the upper respiratory tract affections are responsible for the lower respiratory tract diseases. This relationship between nose and asthmatic attacks has been clearly demonstrated in animal experiments as well as by clinical observations. Anatomical abnormalities such as spurs and deflected septum or pathological factors like chronic sinus infection or infected teeth and tonsils are all important

factors in initiating attacks of bronchial asthma. Treatment of these nasal factors results in the subsidence of asthmatic attacks and a recrudescence of the nasal trouble results in a recurrence of the attacks.

There are also some upper respiratory tract diseases whose existence is controlled by a previously existing lower respiratory tract affection. Tubercular affection of the nose, pharynx and larynx is always secondary to a pulmonary lesion. Submucosal lymph channel between the two regions is probably the main route of spread of infection. Contact with infected sputum during its passage through the upper respiratory tract is also a potent factor.

In cases of bronchiectasis again, chronic sinus infection is known to occur as a sequela. When a radiograph of the sinuses is taken twenty-four hours after doing a bronchogram, lipoidal is seen in the sinuses. Hence there is no surprise that in bronchiectatic patients the nasal sinuses get infected by the infected sputum entering them during coughing and sneezing.

Sometimes a disease affecting one region can simultaneously affect the other half as well. Common cold is a good example. It is important to recognise the involvement of the entire respiratory tract during the course of a common cold. When symptoms persist beyond the usual period, the entire respiratory tract must be investigated for any lurking focus of infection.

An individual with an allergic diathesis can show its evidences in the nose or lungs or both. Allergy predisposes to infection by increasing the tissue permeability and enabling the irritating factor to act with greater ease, and by lowering the tissue resistance and leading to frequent exacerbations, retards recovery.

Thus we find the close connection between the diseases of upper respiratory tract and those of the lower respiratory tract. An appreciation of the role of the individual units in the production, maintenance or aggravation of the affections of the other half of the respiratory tract is very essential for a successful outcome of the treatment. Sometimes we find that a disease in the upper respiratory tract can be the cause of that in the lower respiratory tract or may be a sequela of it. This point has to be carefully adjudged in every patient. A good example is the connection between chronic sinusitis and bronchiectasis. Sinusitis can be primary to bronchiectasis or bronchiectasis can be the primary one and chronic sinusitis only a sequela of infected sputum passing through the chambers of the upper respiratory tract and infecting the sinuses. Bronchiectasis which is a sequela of chronic sinusitis is always bilateral and is confined to the lower lobes, gravity playing a large part in its production and maintenance. When bronchiectasis is unilateral, other causes like foreign bodies and aspiration of infected material must be looked for. So also we find that infection may start at one end of the respiratory tract and spread to the other end; or the same infection may simultaneously affect the entire respiratory tract. When any infection persists beyond the usual period in spite of adequate treatment the possibility of its lurking in some portion of the

respiratory tract should be kept in mind and the whole of the tract should be investigated irrespective of the original infection being in either half of the respiratory tract. Cough is a symptom of affection of either half of the respiratory tract and sputum can also result from infections in either half. Patients sometimes come for a symptom referable to one unit whereas the disease is entirely in the other half. For example, patients with a mediastinal mass come for change in the voice which is a result of a vocal cord paralysis due to pressure on the recurrent laryngeal nerve. Again patients with chronic sinus infection sometimes present themselves with low grade fever and cough particularly at nights, and get investigated and treated for pulmonary tuberculosis with no improvement. Like this experiences can be recounted in legion. All this is only to stress that the diseases of the upper respiratory tract and those of the lower respiratory tract are very closely interlinked and that both units of the respiratory system must be investigated and the importance of every symptom and sign correctly assessed in every disease of the respiratory tract.

References :

1. Dr. P. Narasimharao, "Bezwada Medical Association Silver Jubilee Souvenir."

2. Sir St. Clair Thompson and V. E. Negus, "Diseases of Nose and Throat."

Stop me if you have heard it, but can you ?

The Doctor concluded his examination "I can't find the cause for your trouble. Offhand, I would say it's due to drinking."

The patient shook his head understandingly. "Perhaps I better come back, Doc, when you're sober."

COMMON SKIN DISEASES

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INTRODUCTION

Anatomy of the Skin

Two layers—

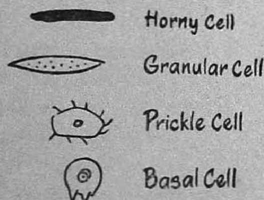
(1) Epidermis

(2) Dermis

(1) The epidermis contains (*a*) Stratum Corneum, (*b*) Stratum Lucidum, (c) Stratum Granulosum, (*d*) Reti Malphigi and (*e*) Basal Cell layer.

(2) The dermis contains the important structures, *viz.*, the sweat glands, hair follicles, sebacious glands, the important plexus of nerves and the blood vessels ; the lymphatics are also present in the dermis.

The various cells in the epidermis:—



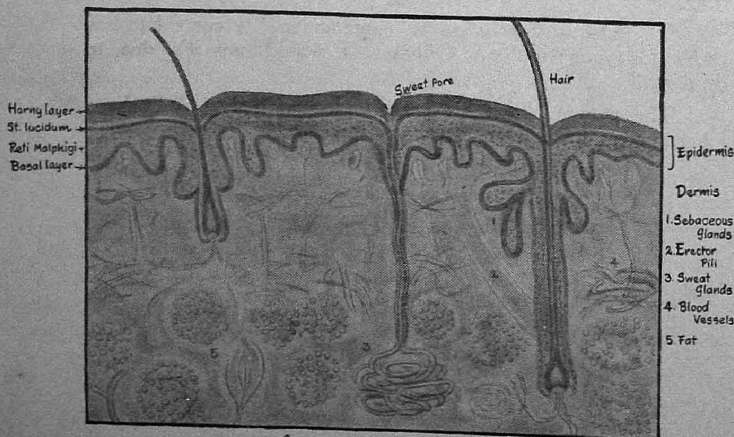
The stratum corneum or horny layer is higher in the palms and soles. The cells have no nucleus.

The stratum lucidum is intermediate between horny layer and the granular layer and has no nucleus. The layer is seen only on the palms and soles.

The stratum granulosum is lozenge shaped and contains granules of eleidone.

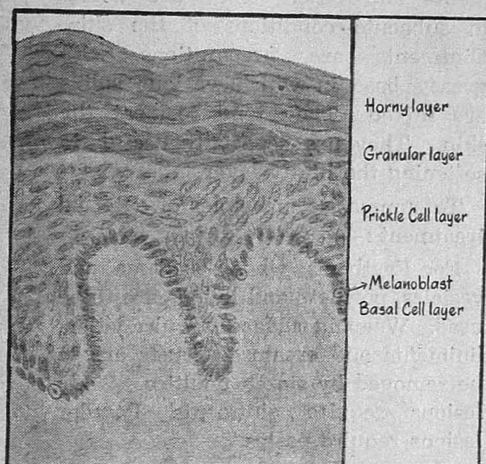
The prickle cell layer (Reti Malphigi) is polygonal with filamentous process connecting one another.

The basal layer contains cylindrical cells and in this layer certain special cells are seen which are responsible for pigmentation of the skin. These melanin forming cells are similar to neuroglial cells and have dendritic processes which end in a cup shaped or button like end. The melanin is manufactured in these cells and is liberated, *via* the cup-shaped ends



Anatomy of the Skin

and passes to the surface as well as carried inside by cells called chromatophores. The melanin formation is under the control of the melanocytic harmone of the anterior pituitary.



Anatomy of epidermis

The various appendages of the skin

(1) The nail is an appendage of the skin and contains the following points :—

(2) Hair follicles are developed as downward prolongation. The tube contains an inner and outer root sheath and the former is adherent to the hair. The hair itself contains a cortex and medulla.

(3) The sebaceous gland contains fat-containing cells attached to the hair follicle by a short duct. These are found all over the body except the palms and soles.

(4) The sweat glands are developed from narrow epidermal invagination which descends deeply into the corium. The sweat glands are present all over the body surface including palms and soles. There are two types of sweat glands, the eccrine glands which produce the sweat and the apocrine glands which discharge products of cellular disintegration into lumen and are found in the axillae, nipple and pubic area.

Functions of the skin

(1) The skin is a protective organ for the body, (2) It regulates temperature and (3) It serves as an excreting organ.

Dermatological Alphabets

1. Erythema is a reddish area over the skin.

2. Macule is a lesion flush with the surface. It is neither raised nor depressed. The patch may be erythematous, pigmented or discoloured. In measles, macules are erythematous.

3. Wheal is a special type of lesion which is raised above the skin. It is evanescent with a pale area in the centre and erythematous area at the periphery.

4. Papule is a solid elevation not more than 5 m.m. diameter.

5. Vesicle is an elevation containing fluid not more than 5 m.m. in diameter.

6. Bulla is an elevation containing fluid more than 5 m.m. in diameter.

7. The pustules contain pus in the centre.

8. Nodule is a solid elevation more than 5 m.m. in diameter.

9. Crusts are produced by coagulation and desiccation of plasma and serous exudates on the skin.

10. Fissures are produced by mechanical splitting of the epidermis.

11. Plaques are hard areas of skin.

12. Lichen is an aggregation of papules.

ALLERGY

This is a hypersensitivity reaction. The skin may be used as test organ for detecting allergic states.

Principles of Treatment of Skin Diseases

The first principle in dermatological therapy is the removal of crusts wherever found on the skin. Various methods can be used. I will describe one method.

Starch boric poultice is the most effective method.

One teaspoonful of boric acid and 4 tablespoonsful of starch are mixed with cold water and add to it, a pint of boiling water till the starch bursts and forms into a cream. When this is cold, pieces of gauze soaked in this are applied. The poultice is renewed 3 or 4 times till the crusts are removed.

Soap is not useful for skin condition since it produces desquamative action. Soap is useful in Acne Vulgaris but is harmful in Eczema.

Powders: Simple powders are useful in protecting the skin from external irritation and are to be used over dry skin for exudative skin lesions. Hard cakes are formed.

Lotions are used for cooling, astringent, antipruritic and antiseptic effects. In using Calamine Lotion, the water evaporates producing soothing and astringent effect. The zinc oxide and calamine are protective and glycerine makes them adhere.

In inflamed and weeping skin lesion, it is best to use Calamine oily liniment producing cooling effect without much drying. Creams have similar effect as liniment and are useful in inflamed skin lesions. Pastes are preparations having equal proportion of solid ingredients and base. They are useful in subacute conditions of the skin. Ointments are preparations with greasy base in which various solids are mixed. They tend to heat the skin. They are especially useful in softening the horny layer.

To summarise principles of external treatment :—

For Erythema, wheals, oedema and vesicles, powders and lotions are to be used. Weeping surfaces require lotion, liniments and creams. Crusts are to be removed by starch poultice. Scaly lesions require ointments. Papular lesions require pastes.

Various antibiotics and chemotherapeutics can be incorporated in these preparations but they should not be used more than 7 days to prevent the risk of allergic hypersensitiveness.

Physical methods

(1) Cold :—Co.2 snow is useful in the treatment of warts.

(2) Heat :—Galvano cautery is similarly useful.

(3) *Ionisation* :—Copper ionisation is useful in fungus infections of skin.

(4) U.V. Rays are useful in Psoriasis, Eczema and Acne Vulgaris.

(5) Superficial X-Ray therapy is beneficial in local Eczema.

DISEASES OF THE SKIN

I. Erythematous Eruptions :—

(1) *Non-specific types* :

Dermatitis medicamentosa is a cutaneous manifestation produced by

drugs, *e.g.*, Penicillin, iodides, luminal, sulphonamides, etc.

Arsenic produces a variety of lesions

(i) Nine day Erythema.
(ii) Exfoliative Dermatitis.
(iii) Rain drop pigmentation.

The various measures are:—(1) to stop the drug, (2) to use externally liniments or lotions and (3) to use in the case of Arsenic poisoning BAL.

(2) *Specific Erythemata*:

(*a*) *Erythema multiforme*: The lesions are erythematous papules and plaques. The commonest sites are back of the hands and forearms, front of knees and back of feet, back of neck.

These types of eruptions with mucous membrane involvement and toxemia are known as Stevens Johnson Syndrome. Bullous and purpuric lesions may also occur.

Aetiology

(i) Systemic infections as pneumonia and meningitis.
(ii) Viral infections.
(iii) Drugs as quinine and sulphonamides.

The measures are (1) Corticosteroids systemically and (2) Calamine Liniment externally.

(*b*) *Erythema Nodosum*: The lesions appear as bright red nodules on the front of the legs and posterior aspect of the forearms. They are symmetrical in distribution. Fever, sorethroat and joint pain may accompany the lesion.

Aetiology

(i) Tuberculosis.
(ii) Rheumatism.
(iii) Iodides.

The measures are (i) Rest, (ii) Salicylates, (iii) Causative treatment.

Erythema Iris: (Iris: Rainbow)

The lesions are concentric, with various coloured rings. The centre is rosepink and may be vesicular. Around this occur two or more zones, occur alternately dark or purple. The usual size is ½ to 3 cm.

The causes and management are similar to erythema multiforme.

Cutaneous Lupus Erythematosus

The disease most commonly affects the face producing a butterfly distribution over nose and malar regions. Patches may occur in other areas also. The lesions are brightly erythematous and follicular plugging occurs. Lupus erythematosus may be chronic discoid or disseminated variety. The disseminated variety is characterised by (1) Skin lesion, (2) a verrucous endocarditis of Libman Sacks, (3) Leukopenia, (4) an increase of serum globulin, (5) Demonstration of L.E. Cell, (6) Nephritis.

Aetiology

(1) Cause of localised type is not known. (2) Disseminated lupus erythematosus is a collagen disease.

Treatment

Internal—Chloroquin 0.3 gm. thrice daily.

External—Calamine lotion.

In generalised or systemic Lupus Erythematosus, A.C.T.H. or Cortisone is to be given.

URTICARIA

This is a symptom complex characterised by the occurrence of wheals which appear and disappear.

Aetiology

The causes may be exogenous and endogenous.

Exogenous—

(1) Contactants.
(2) Ingestants (Food).
(3) Injections (Drugs, insect bites).
(4) Inhalation (House dust).
(5) Physical stimuli (Climate).

Endogenous—

(1) Micro organisms.
(2) Parasites.
(3) Psychogenic stimuli.

Cutaneous tests with various allergens are helpful in finding out the cause of allergy.

Treatment: Specific measures are directed against desensitisation against offending allergens.

(2) Non-specific measures are
(*a*) Autohaemotherapy.
(*b*) Peptone.
(*c*) Calcium gluconate.

(3) Symptomatic measures are
(*a*) Antihistamine.
(*b*) Corticosteroids.
(*c*) Externally: Antipruritic lotion.

VESICULAR AND BULLOUS LESIONS

(1) Herpes simplex occurs in the circum-oral area and genitals. The disease is caused by filtrable virus. A variety of precipitating causes favour the action of the virus namely, pneumonia, meningitis, leukemia and gastrointestinal upsets.

The measures are externally 1% aqueous methylene blue and directed against precipitating causes.

(2) *Herpes Zoster*: Groups of vesicles occur in connection with this due to virus infection. The lesions are unilateral. It may occur at any age and in either sex. The occurrence of zoster confirms immunity against varicella. The lesions are preceded by neuritic pain. In some cases the pain will occur after the lesions subside called post-herpetic neuralgia. The skin changes are explained as due to vascular dilatation produced by stimulation of the posterior nerve roots; but the actual virus has been recovered from the skin lesions.

Herpes zoster may also occur in relation to the cranial nerves *e.g.*, Trigeminal (Gasserian ganglion) and Facial (geniculate).

Complications of Herpes zoster

(1) Paralysis (temporary or permanent) due to the involvement of the nerves.

(2) Secondary infection of the skin lesions.

(3) Involvement of the eye in zoster Ophthalmicus.

The various methods of treatment

External:

The principles involved are:

(*a*) care of the affected area
(*b*) reducing pruritis
(*c*) preventing secondary infection.

A mild antipruritic lotion is beneficial.

General treatment

(*a*) Posterior pituitary extract 0.5 c.c. S.C. daily for 3 days prevents the onset of post-herpetic neuralgia.

(*b*) Broad spectrum antibiotics: Tetracycline 250 mgms. thrice daily for 5 days.

(c) Antiinflammatory measures with corticosteroids as Prednisolone.

(3) *Dermatitis Herpetiformis or Duhring's disease*

A polymorphic eruption containing erythema, vesicles and bullae. The cause of the disease is not known. It has been suggested that it is due to toxaemia.

There are 4 features: (1) The eruption is polymorphic; (2) It is attended by intense itching; (3) It is recurrent; (4) The patient's health is good.

The commonest sites are limbs but any part of the body may be involved.

Treatment

Till recently Sulphapyridine was given orally in doses of 0.5 gm. thrice daily. Today the best remedy is Sulphone (Dapsone) 25 mgm. daily.

PEMPHIGUS

This is essentially a bullous lesion. The varieties of pemphigus are: (1) Pemphigus vulgaris, (2) Pemphigus vegetans and (3) Pemphigus foliaceus.

1. *Pemphigus Vulgaris*: Bullae arise on the mucous membrane and skin. The bullae arise characteristically from normal skin. Any lateral tension easily detaches the bulla. This is called Nikolosky's sign.

2. *Pemphigus Vegetans*: The eruption tends to be distributed on the groins and pubic area and axillae. There are vegetations and bullae. The latter are not conspicuous. Unless carefully looked for, it is possible to miss the diagnosis.

3. *Pemphigus Foliaceus*: In this variety the bullae rupture readily leaving exfoliated skin.

Aetiology

The cause is not known. It is said that a pathogenic enzyme produces the histological picture of pemphigus. Toxaemia is blamed to produce the lesion. The nature of the toxin is not known.

Biochemical changes in pemphigus

(*i*) A progressive fall in serum proteins. Fall in albumin and relative increase of alpha globulin and gamma globulin.

(*ii*) An increase in fibrinogen.

(*iii*) Increase in sedimentation rate.

(*iv*) Fall in serum chloride, sodium and calcium.

Pemphigus is a grave disease and requires energetic measures.

Treatment

Corticosteroids are beneficial in pemphigus. A good and skilled nursing is to be given. There are few conditions in medicine which present a greater challenge to the trained and sympathetic nurse. Bath in permanganate 1 in 25,000 and the application of calamine lotion are advised.

ECZEMA

This is a tissue reaction that involves the epidermis and the upper portion of the dermis. The reaction commences with erythema in which pinhead-sized vesicles occur. These vesicles are situated in the epidermis. They rupture and discharge fluid. The oozing surfaces may be secondarily infected and crust formation may take place when the skin thickening takes place producing the lichenified stage.

Causes of Eczema: (*a*) Predisposing and (*b*) Exciting.

Predisposing — Local predisposing causes are:

(*i*) Excessive dryness of skin.
(*ii*) Excessive sweating.
(*iii*) Excessive greasiness.
(*iv*) Chronic Venous congestion seen in the legs due to varicosity or due to chronic venous congestion proper.
(*v*) Contactants.

General predisposing causes

(*i*) Heredity.
(*ii*) Age : In infancy, infantile eczema is a special feature.
(*iii*) General debility.
(*iv*) Indiscretion in diet.

Infantile Eczema is a perplexing and exasperating condition. The eczematoid dermatoses commonly occur in the cheeks, chin and forehead. The central area of the face is spared. The condition is said to be sensitisation to some food as cow's milk. The house dust may aggravate the condition.

Management of infantile eczema

(*a*) Dietetic restriction. It is advisable to cut down cow's milk and to use goat's milk.

(*b*) Prevent house dust.

Local measures depend on the condition being acute, acute exudative, sub-acute or chronic.

During the acute stage, without exudation Aluminium acetate lotion or Burrow's solution is used. During the acute exudative stage calamine lotion or calamine oily liniment is the best. During the sub-acute stage Zinc cream and during the chronic phase Lassar's paste are the best.

For bath, starch bath is the best. No soap is to be used in infantile eczema.

The general measures are:

(*a*) Antihistamines as Amphodryl or Actidyl Elixir.
(*b*) Sedatives as bromides.
(*c*) Partial immobilisation of the patient from scratching.

Eczema in adults: Eczema may occur due to various causes: (1) Physical stimuli *e.g.*, Photosensitivity to light producing Solar Eczema. (2) Chemical Eczema *e.g.*, due to external contact with a chemical *e.g.*, dyes, watch straps, lip sticks. (3) Infective Eczema : This is presumed to be due to sensitisation to streptococcus. The area most commonly affected is the retro-auricular fold. (4) Varicose Eczema occurs in the lower parts of the legs when varicose veins occur.

Dermatitis Autophytica is a condition occurring in hysterical girls and is due to some psychological aberration.

The Management of Eczema

Certain local conditions predispose to eczema, for instance, the xeroderma-

tous skin. This can be kept supple by glycerine and water. The use of soap in a patient prone to eczema should be avoided, since soap by its degreasing and keratolytic effect favours percutaneous penetration of environmental allergens. Varicose veins must receive attention.

Diet: High protein diet with restricted carbohydrates and fats are of value. Avoid diets to which the patient is sensitive *e.g.*, eggs and peas.

Internal: (1) Sedatives and tranquilisers are beneficial in getting over itching.

(2) The antihistamines relieve itching and help the controlling of the capillary reaction.

(3) Prednisolone 5 mgms. thrice daily and gradually tailing off the dosage is helpful in generalised eczema.

PSORIASIS

The lesions commence as a dull red papule covered with waxy white scale. The papules gradually increase in size to form plaques. These scales are silvery white. The silvery white line produced in scraping the lesions is known as *Tache de Bougie*. The sites are Scalp, Back of the elbows, Front of the knees and Small of the back.

Psoriatic patients are usually in the best of health. They are generally constipated. They are generally high strung hypersensitive individuals. The histopathological section shows (1) Elongation of the rete ridges, (2) The stratum corneum is parakeratotic, (3) Infiltrating polymorphs migrate into suprapapillary portion forming Microabscesses of Munroe.

Etiology of Psoriasis

(1) Toxic Theory : Some toxins are held responsible.

(2) Infective : Prof. Kryle believes in a filter passing virus.

(3) Metabolic : There is increase in blood cholesterol and it has possibly something to do in the causation of the disease.

(4) Endocrinal : Psoriasis increase during puberty and menopause. It is possible that there is some endocrinal disorder.

(5) Psychosomatic : Psoriasis may be influenced by emotional upset. There is an interesting case of a student getting psoriasis prior to examination.

Therapy—General : (1) Tranquiliser, (2) Corticosteroids for psoriatic lesions associated with arthritis, (3) Chronic recurrent cases respond to auto-haemo-therapy. T.A.B. Vaccine; collosol mercury sulphide and arsenic.

Local Measures

(1) 0.5% Dithronal or cignolin or chrysarobin.

(2) For Scalp :
R/-Liquor picis carb. Dr. 1.
Acetone Oz. 1. Mix.

(3) U.V.R. exposures.

Pityriasis Rosea or Gilbert's disease: Numerous papular and scaly lesions occur on the trunk and proximal parts of limbs. The lesion commences with a reddish-yellow oval patch with the long axis horizontally. This is called herald patch. In 7 to 14 days generalised eruptions of papules and scaly

lesions are produced. The itching is slight.

Etiology

The herald patch is due to external infection and the secondary lesions are due to the absorption from the primary lesion.

Treatment: 2% Salicylic acid ointment is helpful.

LICHEN PLANUS

Diamond-shaped papules occur in this condition. They have a violet or blue colour. The papules may group together to produce various shapes. Papules may occur on the mucous membrane of the cheeks and lips producing hexagonal white patches.

Lichen Verrucosus: It is a chronic thickened variety of Lichen Planus. Histopathologically there is a perivascular infiltration of the lymphocytes in the papillae which hug the epidermis.

Treatment : Externally antipruritic lotion and systemically cortisone or A.C.T.H. Large doses of Vitamin B_1 have been found beneficial.

Pityriasis rubrapilaris or Lichen rubra acuminatus is a chronic affection characterised by small red conical papules around the hair follicles on the limbs and trunk with redness and scaling.

The etiology is unknown. A deficiency of Vitamin A predisposes to the condition. There is hyperkeratosis of the follicular orifices. Large doses of Vitamin A and C are found beneficial. Locally cooling lotion with lead and glycerine is beneficial.

PITYRIASIS RUBRA OR EXFOLIATIVE DERMATITIS

Causes

(1) Metallic poisoning as arsenic.
(2) Blood diseases as leukemia or Hodgkin's disease.
(3) Mycosis fungoides.
(4) Overtreated skin lesion *e.g.*, in psoriasis.
(5) Pemphigus foliaceus.
(6) Idiopathic.

The measures are :

(1) A.C.T.H. or Cortisone.
(2) B.A.L. for metallic poisoning.

BACTERIAL INFECTIONS OF THE SKIN

Impetigo Contagiosa is caused by streptococcal infection and in the crusts, staphylococci are found. The lesions commence as reddish macules which soon become vesicles. The vesicles are under the stratum corneum and rupture readily. Secondary infection takes place. The serous and purulent fluid become crusted. New satellite lesions appear. The commonest site is the face.

Treatment

(1) Removal of crust with starch boric poultice.
(2) Antibiotics externally for 5 to 7 days.
(3) Systemic administration of antibiotics.

FOLLICULAR IMPETIGO OF BOCKHART

Small dome-shaped pustules occur in relation to hair follicle. The favourite sites are the extremities and scalp. The measures are : antibiotics externally and systemically.

Sycosis Vulgaris is a purulent folliculitis in the beard and moustache area due to staphylococcus aureus.

Treatment : Vioform cream or any antibiotic cream externally.

Internally—A course of antibiotics.

Furunculosis or crops of boils is an infection of the pilosebaceous follicle due to staphylococcus. The various predisposing causes are (*a*) Diabetes, (*b*) Debility, (*c*) Focal sepsis.

Carbuncle is a conglomerate ferun-cle.

Therapy :

(1) Treatment of possible known factors as diabetes.
(2) Penicillin.

DISEASES DUE TO ANIMAL PARASITES.

SCABIES

Cause : Itch mite or sarcoptes scabiei. The parasite is 0.3 m.m. in length. The impregnated female forms a burrow in the epidermis. Each impregnated female lays about 40 to 50 eggs. The eggs pass via larval and nymph stages into adults. This cycle takes about 14 days.

Sites : Webs of the fingers, ulnar border of the hands, anterior axillary folds, buttocks and pubic region. There is intense itching which is worse at night. This may produce secondary infection producing widespread impetiginised lesions.

The various complications are urticaria, scratch dermatitis, secondary infection, albuminuria and eosinophilia.

Scabies is a contagious disease and hence in the management, every member or contact should be treated.

The first principle is to lay open burrows by warm water and soap and scrub with rubber brush. During the non-infected stage, one of the following :

(1) 25% Benzyl benzoate emulsion.
(2) Sulphur ointment.
(3) Tetmosol.
(4) Eurax.

The application must be thorough and should be applied in all affected areas properly. The dress worn should be sterilised in boiling water. A bath should be taken 3 hours after applying ointment. All the contacts are to be treated. During the infected stage, it is advisable to use plain zinc cream or sulphonamide cream and systemically use antibiotics before switching on to one of the antiscabitic treatments mentioned above.

SCABIES NORVEGICA

This condition occurs in lepers and mental patients. The disease is characterised by extensive crusting composed of dried pus and scales riddled with galleries of innumerable parasites and their ova. Distortion and destruction of the nails is not uncommon. The causative organism is the same itch mite.

PEDICULOSIS OR LICE INFESTATION

Pediculosis Capitis: Head lice (Pediculus capitis) commonly found in the scalp and sometimes seen in the hairs of the beard of the old men. The ova or nits are abundant. There is severe itching producing excoriation and crusting.

Pediculosis Corporis : Body lice are found in the under clothing and are stuck to body hairs. The lice transmit relapsing fever and typhus fever. The skin due to pediculus corporis becomes dry, pigmented with areas of dermatitis. This condition is called Vagabond's disease.

Pediculosis Pubis : The pubic louse or crab louse is shorter and stouter,

than its companions. The lice produce scratching.

The lice produce a peculiar pigmentation of the skin called Maculae cerulae. The colour is slaty. The lesions are said to occur from the pigment in the insect.

Treatment of pediculosis: 3 to 10% D.D.T. is effective against pediculosis.

CLIMEX LECTULARIUS OR COMMON BEDBUG

The symptoms are itching and burning with pea or bean sized elevations on the skin.

Treatment: Tropical application of spirit of camphor. The measures against bedbug is 5% D.D.T. spray.

CATERPILLAR DERMATITIS

The caterpillar produces erythematous macules, urticaria and intense itching.

Treatment: Soothing Lotion externally.

DISEASES DUE TO VEGETABLE PARASITES

The term tinea is used to include the superficial inflammatory skin conditions produced by the Dermatophyte group.

The principles of treatment of Fungus infections: The various antifungal agents used in dermatology:

(1) Whitefield's Ointment.
(2) Unsaturated fatty acids like propionic acid, caprylic acid, and undecylenic acid are especially beneficial in fungus infections of hands and feet.

Tinea Capitis or Fungus infection Scalp

Commonest age between 5 and 10 years. There are two types of Fungi (*a*) small spored and (*b*) large spored Fungi. Multiple lesions are produced by the small spored Fungi. A cow tethered to a pole in a rich meadow• and thus compelled to feed on a circular patch. The appearance of the patch when the cow has finished with it will be that of ringworm. The stumps of hairs are bent and twisted in all directions. The bright green florescence of the infected hairs can be seen with U.V.R. passed through a filter. The large spored type produces a single patch resembling an abscess. This is called Kerion.

Treatment: Shampooing hairs with soap and spirit followed by application of fungicidal ointments. In certain cases epilation by X-Ray exposures is helpful.

Tinea circinata or Ring worm of the glabrous skin or Tinea Corporis

In this, circumscribed circular patches are produced. The ringed edge is composed of papules or vesicles.

Treatment: Antifungal Ointments. Unguentum chrysarobin is used as a fungicide when the patch is small and in adults.

Tinea Barbae (or Fungus infection of the beard or moustache area)

There are 3 clinical varieties. (*a*) The annular type which is mild; there are several reddish rings. (*b*) The inflammatory type is characterised by deep-seated follicular pustules and nodules which are irregular. (*c*) The sycosis type resembles the sycosis vulgaris variety.

Treatment

(1) Warm Boric compress.

(2) Reduce inflammation by applying 0.5% aluminium acetate.
(3) Antifungal ointments.

Fungus infections of hands and feet

Four clinical varieties are described:

(1) Acute vesiculobullous variety.
(2) Chronic intertriginous variety between toes and fingers..
(3) Chronic hyperkeratotic variety and
(4) Type with fissuring.

Treatment: For the first two types, warm 1 in 10,000 permanganate bath and applying Burrow's solution are the initial procedures. Then the treatment is similar in all varieties. The unsaturated fatty acids as undecylonic acid or propionic acid or caprylic acid 5% is helpful.

Tinea versicolor or pityriasis versicolor

It is a special type of fungal infection producing yellowish-brown patches on the skin. The appearance is more hypopigmented than brown. The brown variety is caused by a variant and is known as Erythrasma.

Treatment: Scrub the skin with 20% Hypo.

TUBERCULOSIS OF THE SKIN

Tuberculosis of the skin may be :—

(1) Primary tubercular infection.
(2) Secondary or reinfection tuberculosis.
(3) Tuberculids are lesions produced in persons with high degree of immunity.

Of these lupus vulgaris belonging to the second group is the commonest.

Lupus Vulgaris:—Multiple nodules called apple jelly nodules occur in this condition. The nodules are in the periphery and there is thin scar in the centre. In Lupus Verrucosa cutis, hard greyish white hyperkeratosis develops.

Scrofuloderma occurs in relation to tubercular glands underneath producing collar stud abscess and invading the skin. As an example of tuberculid, erythema induratum scrofulosorum or Bazin's disease can be given. The lesions are purplish red with swelling on the back of the calves. Sooner or later they ulcerate.

Treatment of Tuberculosis of Skin:

(a) Improvement of general health.
(b) Anti-tubercular drugs.

DISORDERS OF THE SKIN APPENDAGES

Sweating of the palms and soles or localised hyperidrosis: The cold clammy hand shake is a key sign. Emotional instability plays an important cause.

The measures are—

(a) Tranquiliser orally.
(b) Measures to reduce sweating by drugs as Banthine and practal 50 mgm. thrice daily.
(c) External—R/

Aluminium chloride	..	3
Boric acid	..	7
Alum	..	10
Salicylic acid	..	3
Amylum	..	5
Talc	..	100

ALOPECIA OR BALDNESS

The predisposing factor is focal sepsis. One or two circumscribed or oval patches may be seen. Spreading areas show short stumps like marks of exclamation.

Treatment : Stimulating hair tonics are useful for falling of hairs and alopecia. They contain spirit Rosemarini and oil of citronella with can tharidine.

Grey hairs or canites : This is due to the disorder of the pigment cells in the hair matrix. The greying produces a salt and pepper look.

Para aminobenzoic acid as Dermachrome or Paba is beneficial

ACNE VULGARIS

It is a disease of the bloom of youth. There is hypersecretion of sebum and hyperkeratosis of hair follicles.

The various measures are :—ACNE is mnemonic.

A Correct *A*naemia and also give Vitamin *A*.

C Correct Constipation and also give Vitamin *C*.

N *N*ever use creams or snows.

E *E*xercise and *E*ndocrines as stilboesterol.

Externally : Pusey's liniment or Lotion Salicylic cum Sulphur.

Internally : Stilboesterol tablets. Dose—In women 0.5 mgm. daily for 10 days after periods and in men for three weeks at a stretch.

LEUCODERMA OR VITILIGO

This is produced due to disorder of pigmentation. The cause is not known. The pigmentation is stimulated by U.V.R. exposures and by a drug called Ammimajus or Hishopweed which contains some active principles that initiate pigmentation. The preparation is meladinine which is applied externally and given orally.

This subject of Common Skin Diseases has not been discussed intoto but an attempt has been made to give a BIRD'S-EYE VIEW OF DERMATOLOGY.

Frightened patients when they want a cure,
Bid any price and any pain endure,
But when the Doctor's remedies appear
The Cure's too easy and the price too dear.

A BRIEF SKETCH OF THE ORIGIN AND ACTIVITIES OF THE MADRAS CITY BRANCH—INDIAN MEDICAL ASSOCIATION

DR. D. V. VENKAPPA,
President, I.M.A. Central.

The Convener of the Sub-Committee for the Souvenir of the 13th Madras State Medical Conference has asked me as the fittest person to contribute an article on the above subject. Whilst feeling grateful and thankful for giving me the opportunity I can only recapitulate the salient facts about its rise, growth and progress for the past 24 years up to the present day. Before the advent of the Indian Medical Association in the year 1928 medical associations existed under different groups, the chief of them being the existence of District Medical Associations manned by the then District Medical Officers under the British regime. Members happened to hail from the rank and file of the Profession without distinction in Government Service, Private practice, Dt. Boards, Local Funds etc. Side by side some organisations existed on independent basis such as Madras Medical Association, Independent Medical Practitioners' Association, the South Indian Medical Union, so on and so forth, most of them merging with the Indian Medical Association. The South Indian Branch of the British Medical Association was one catering to the needs of medical graduates and Officers of the Indian Medical Service. Necessity then arose to represent the entire profession without its castes and creeds when the All India Medical Licentiates' Association was founded in the year 1913 in this State playing its role to protect and safeguard the interests of the Profession and, in particular, medical licentiates. The Conferences of this Association were presided over by the I.M.S. Officers from the top to the District Medical Officer at the bottom. The Indian Medical Association took the lead in the year 1928 having similar objects and has now emerged as the only national medical organisation on an all India basis to represent the voice of the Profession in our country. This, it may be stated, is the back ground before the emergence and birth of the Madras City Branch in the year 1934 which became merged with the Indian Medical Association though it had had its existence as a separate entity on the analogy of the erst-while District Medical Associations.

THE SOUTH INDIAN PROVINCIAL BRANCH

In the year 1940 a federation of five District Medical Associations was formed, *viz.*, Madras, Tiruchirapalli, Madurai, Ramnad and Tirunelveli. Other District Medical Associations subsequently joined one after the other. The South Indian Provincial Branch,

therefore, began with a membership of about 500 at first, the Madras City Branch having on its rolls about 250, the rest being made up by the other branches. The State Branch then came into existence in the same year affiliating other branches under its jurisdiction. There was also a liaison between the Andhra and Madras State Branches by a Joint Committee which had functioned for some time.

The First All India Medical Conference was held in the year 1937 under the auspices of the Madras City Branch when Dr. B. C. Roy presided over its deliberations and Lt.-Col. K. G. Pandalai was the Chairman of the Reception Committee and Dr. U. Krishna Rao, General Secretary. It was a great success having been attended by leading and distinguished members of the Profession from all over India and other high Officers of Government.

The Provincial or State Conferences were held regularly and Madras City Branch had two Sessions to its credit, the one in the year 1948 and the other in 1953 whilst the ensuing Conference in 1958 under its auspices happens to be the 13th and the third Session. The Presidents of the State Branch (formerly S.I.P. Branch) were elected mostly from the City Branch. The first of those was Captain V. D. Nimbkar, thereafter, Drs. T. S. Tirumurti, U. Krishna Rau, K. C. Nambiar, P. Natesan and M. Santosham had their turns. Dr. P. A. S. Raghavan of Tiruchy was elected twice as well as Dr. Y. P. Vasudevan. Dr. D. V. Venkappa was the interim President during the absence of Dr. T. S. Tirumurti at Trivandrum for a short period.

ACTIVITIES OF THE CITY BRANCH

Regular meetings were held and distinguished members of the Profession took part in its deliberations year after year. It had the opportunity of welcoming eminent members of the Profession from abroad, the teams of the World Medical Association and the World Health Organisation and other Specialists. Mention may be made of other social activities such as Tea Parties and At Homes for those of our members who had earned recognition through the Association or the Government. Latterly Annual Dinners have been the special features when entertainments by children of medical men form the chief attraction. The activities have been so great that special mention may be made of those energetic Secretaries who had played an important role and whose names could be recorded in these pages, *viz.*, Drs. P. T. Raghava Chari, K. L. Narayana Rao, R. Sankaran, V. Vijiaraghavan and U. Sripathi Rao. The Presidents whom I had stated earlier figured largely in that capacity on behalf of the State Branch in these activities.

ACHIEVEMENTS

The following reforms were effected during the period :

1. The abolition of Medical Schools and the ushering in a uniform standard of medical education, the M.B.B.S. Degree.
2. The amendment of the Madras Medical Registration Act of 1914 by the abolition of compartments and establishment of a Medical Register in an alphabetical order

FIG. 1
Dermo-Graphism

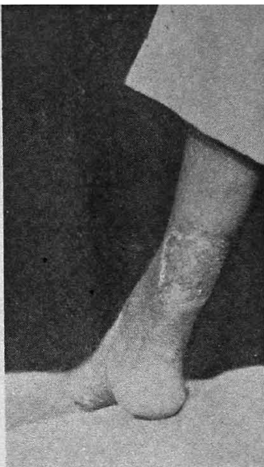


FIG. 2
Eczema

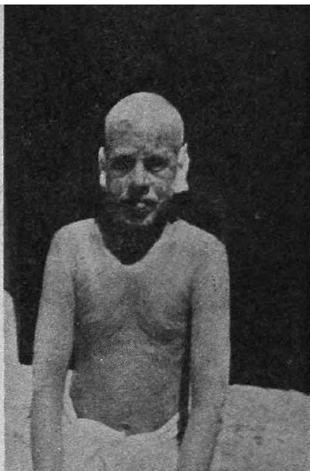


FIG. 3 Exfoliative Dermatitis

FIG. 4 Lichen Planus

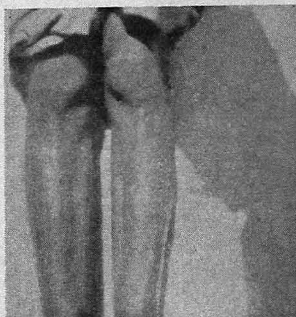


FIG. 5 Bockart's Folliculitis

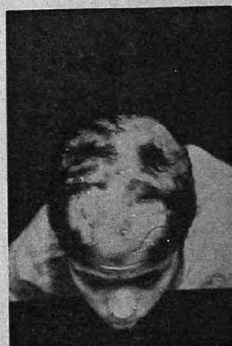


FIG. 8 Alopecia Areata

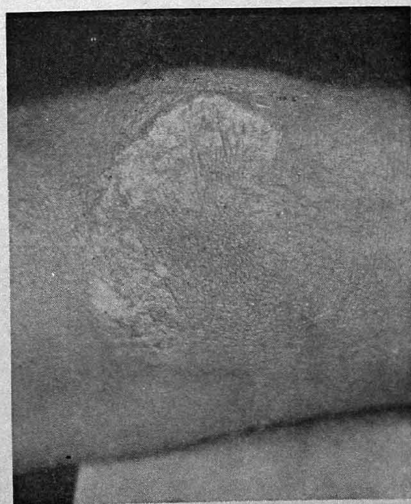


FIG. 6 Lupus Varicosus

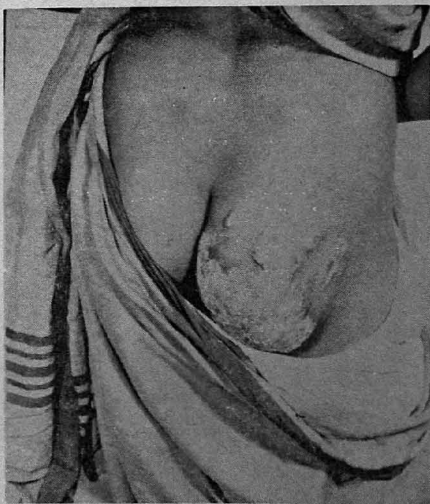


FIG. 7 Lupus Vulgaris

irrespective of qualifications and academic distinctions.

3. Unification of the Civil Medical Services and getting rid of the compartments in the Services.

The above three reforms were mainly due to the activities of the All India Medical Licentiates' Association.

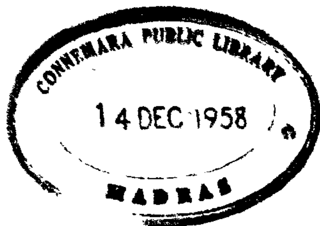
4. Abolition of Sales Tax on Medical Practitioners.
5. Efforts to amend the Rent Control Act and partial success.
6. Exemption from the Madras Shops and Establishment Act.
7. Efforts to democratise the Employees State Insurance Scheme.
8. Representation on Committees by Government and reference to our organisation in matters connected with Medicine & Public Health.

Be it said to the credit of the Madras City Branch, it had rendered meritorious services by its whole hearted cooperation with the Government and sister organisations in achieving the objects of our Association. The membership of the Association has been steadily increasing.

The Profession of Medicine is indivisible and none can come in the way of our progress. The Association makes no difference whether a member belongs to the Services or private practice or any other walk of life. The Association is the parent of us all. It never was the idea of giving publicity to this aspect and it is the only remedy to bring home to our brethren the solidarity and unity of the Profession on which every well wisher lays stress for fulfilling the objects for which the Association stands. As one who is intimately connected with the organisation ever since its inception it is my prayer to the Almighty that-good-will and comradeship should prevail amongst us.

Family Planning

" Before retiring take a glass of water and that's all."



"Lofty thoughts for Lonely Moments"
"A thought for a day 'ere the year's away"

COLLECTIONS

DR. V. VIJAYARAGHAVAN
Ophthalmologist
Member, Madras Medical Council & Hony. Presidency Magistrate,
Vice-President, I.M.A. City Branch & Hony. Joint Secy., I.M.A. State Branch

Learning and wisdom are not identical. They are not always on talking terms. Learning looks backwards to the past and wisdom looks forward to the future.

* * *

Every high way of human life dips in the dale now and then. Every man must go through the tunnel of tribulations before he can travel on the elevated road of triumph.

* * *

Studies serve for delight, for ornaments and for ability. Their chief use for delight is in privateness and retiring ; for ornament, it is discourse and for ability, is in the judgement and disposition of business.

* * *

Every hard duty that lies in your path, that you would not rather do, that it will cost you pain and struggle or sore effort to do has a blessing in it. Not to do it, at whatever cost is to miss the blessing.

* * *

No horse gets anywhere until, he is harnessed. No steam or gas drives anything until it is confined. No Niagara is ever turned into light and power until it is tunnelled. No life grows great until it is focussed, dedicated and disciplined.

If we could read the secret history of enemies, we would find in each man's life a sorrow and suffering enough to disarm all hostility.

* * *

Predestination is a great consolation to man. If he believes that whatever comes to his lot, pleasant or painful, acceptable or detestable, is the fruit of his acts or thoughts then he has nothing to grudge or grieve for.

* * *

Many earnest people, in the excess of their zeal, do incalculable harm to those whom they greatly desire to help. People with sore and bruised hearts usually need loving sympathy and strong, kindly friendship much more than they need theology.

* * *

Men are more apt to use spectacles than looking glass, spectacles to behold other man's faults than looking glass to behold their own. A man can see a little speck of dust in his neighbour's eye, while utterly unaware of the great beam in his own.

* * *

The power to please is a great asset. It will do to you what money will not do. It will often give you capital which your financial assets alone would not warrant. People are

governed by their likes and dislikes. We are powerfully influenced by a pleasing, charming personality. A persuasive manner is often irresistible.

* * *

If you want to know how a stranger gets your measure, how he can tell you who you are, you have only to mind your manner and watch your speech. As your speech is so are you, for it is by spoken word that one saw the degree of our culture or betray the depth of our ignorance.

* * *

Man is never so ashamed of his mistakes, misdeeds or misfortunes before any body else in this world, as he is before his own self. The world may not know of his mistakes or misdeeds, and may not even care to know his misfortunes, but he knows them all, and cannot meet eye to eye with his own self.

* * *

Perhaps the most valuable result of all education is the ability to make yourself do the thing you have to do, when it ought to be done, whether you like it or not, it is the first lesson that ought to be learnt; and however early a man's training begins, it is probably the last lesson that he learns thoroughly.

* * *

No man is born with great abilities. Abilities develop with constant and earnest efforts to make the best use of one's capacities, his latent faculties and conditions in life. The worth of a man is the co-efficient of his inherent qualities and external circumstances, or the result of the proper adjustment of the two.

Trials and hard places are needed to press us forward, even the furnace fires in the hold of that mighty ship, give force that moves the piston, drives the engine and propels the great vessel across the sea in the face of the winds and waves.

* * *

Every man considers himself wiser than the rest of mankind, and even sometimes questions the wisdom of his Creator. He knows not, that all his knowledge immensely magnified and his wisdom infinitely intensified, cannot even scan an atom correctly and completely.

* * *

It is always weakness to be fretting and worrying, questioning and mistrusting. Can we gain anything by it ? Do we not unfit ourselves for action, and unhinge our minds for wise decision ? We are sinking by our struggles when we might float by our faith.

* * *

The love which each individual help needs so desperately for its health and balance must come to it from the other selves which surround it. There must be established between self and others a love relationship of give and take on which the lives of all depends.

* * *

Money is meant to be man's servant, and so long as he is its perfect master it may be a blessing to him, and an instrument with which he may do great good. But when he gets down on his knees to it and crawls on the dust for its sake and sells his manhood to get it, it is only a curse to him.

* * *

Life gives trust in place of suspicion, even though trust be misplaced. It is

better a thousand times, that our trust should be misplaced than that our suspicion should be ill formed. Suspicion of the motive of the others is a terrible root of evil and it is not until we make love, the standard of our daily conduct, that we realise that how natural it is to be suspicious.

* * *

It is by being cast down and not destroyed; it is by being shaken to pieces; and the pieces torn to shreds, that men become men of might and that one a host, whereas men that yield to appearances of things and go with the world, have their quick blossoming, their momentary prosperity, and then their end, which is an end for ever.

* * *

Literature is one of the instruments and one of the most powerful instruments, for forming character, for giving us men and men armed with reason, braced by knowledge, clothed with steadfastness and courage inspired by that public virtue of which it has been well said that they are the brightest ornaments of the mind of man.

* * *

Man little knows that calamities are beyond his patience to bear till he tries them; as ascending the heights of a mountain which looks bright from below, every step we rise, shows us some new and gloomy prospect of hidden disappointment; so in our descent from the summit of pleasure, though the vale of misery below may appear at first dark and gloomy, yet the busy mind still attentive to its own amusement, finds something to flatter and to please.

You can adjust your accounts with everybody else in the world except your own self. Your own misappropriation or misuse of means, you never make good. Mistakes in calculation may be mended but mischievous misapplication of resources can never be recompensed.

* * *

The best things of life come out of wounding, wheat is crushed before it becomes bread. Incense must be cast upon the fire before its odours are set free; the ground must be broken with a sharp plough before it is ready to receive the seed. It is the broken heart that pleases god. The sweetest joys in life are the fruits of sorrow. Human nature seems to need suffering to fit for being a blessing to the world.

* * *

Repentance is not merely a little twinge of remorse over some wrong thing. It is not simply a gust of tears at the recollection of some wickedness. It is not a mere shame at being found out in some meanness or uncleanness or dishonesty. It is the revolution of whole life. Sins wept over must be forsaken for ever. Repentance is a change of heart, a turning of the face just the other way. It is well for us to make diligent quest to be sure that we always abandon the wrong doing which we deplore, that we quit the evil course which we regret, that we turn away from the sin to which we confess.

* * *

There are many lives that creak and grate harshly as they live day by day: Nothing goes right with them. They need lubricating with oil of gladness, gentleness, or thoughtfulness. Have your can of oil with you. Be

ready with your oil of helpfulness in early morning to the one nearest you. It may lubricate the whole day for him. The oil of good cheer to the down-hearted one—Oh, how much it may mean! The word of courage to the despairing speak it.

* * *

Always remember that though another may have more money, beauty, brains than you, yet when it comes to the rare spiritual values such as charity, self-sacrifice, honour, nobility of heart, you have an equal chance with every one of the most beloved and honoured of all people.

* * *

Private opinions create public opinions; public opinions overflow eventually into national behaviour; and national behaviour as things are at present can make or mar the world. That is why private opinions and private behaviour and private conversation are so terrifyingly important.

* * *

There is something about one's personality which eludes the photographer, which the painter cannot reproduce, which the sculptor cannot chisel. This subtle something which every one feels, but which no one can describe which no biographer ever put down in a book, has a great deal to do with one's success in life.

* * *

Human friends eagerly want to help and they come to offer sympathy and consolation. But in such hours the most helpful of us are only like men standing on the shore of a dark and stormy sea, while our friends are far out on the wild waves. We cannot go to them to give help or rescue. Our little boats cannot ride in the mad surges. All we can do is to stand on the shore, as it was, and look with pitying eye and heart at the struggling ones in the angry sea. That is the very best that the richest human love can do. Thus it is in all life's deep needs. It is in such hours we realise the blessedness of All Powerful and only He can comfort in any sorrow and give victory in any strife.

* * *

Poverty, hardship and misfortune have pressed many a life to moral heroism and spiritual greatness. Difficulty challenges energy and pursuance. It calls into activity the strongest qualities of soul. Many a headwind has been utilised to make port. God has appointed opposition as an incentive to faith and holy activity.

* * *

Loose the day loitering it will be the same tomorrow and the next more dilatory, for indecision, brings its own delay. And days are lost, lamenting for lost days. Are you in earnest? Seize this very minute. What you can do or think you can, begin it. Boldness has genius, power and magic in it. Only engage and the mind grows heated. Begin it and the work will be completed.

* * *

and I finish with

"Life is full of lessons great,
To mend and make a man more wise;
In virtue path to lead him straight,
And make him higher and higher wise".

"WISE & OTHER WISE"

DR. V. VIJAYARAGHAVAN
Ophthalmologist
Member, Madras Medical Council & Hony. Presidency Magistrate, Vice-President, I.M.A. City Branch & Hony. Joint Secretary, I.M.A. State Branch

Getting out a Joke is no picnic. If we print jokes people say we are silly. If we don't, they say we are glum, if we clip from others, they credit us lazy and if we don't, we are too proud and fond of our own stuff. " Now, some one will say that I have swiped it from others. Yes. I did, but more a mixture mended."

" All in Fun " Each for a day, ere the year's away.

" What is the Function of the Stomach ? "
Asked the Teacher to the boy in the Hygiene class.
"To hold up your pants Sir," he answered.

* * * * *

Tailor, returning from a funeral
" Sure ! Doctor, you must be a happy man."
Doctor : " Why so ? "
Tailor : " Because you never have any of your bad work returned."

* * * * *

" Now frankly," the Surgeon, warned.
" I must inform you that it is a very serious operation ;
Four out of Five Patients die under it ;
Is there any thing I can do before I begin ? "
" Yes " said the Patient ;
" Help me on, with my Shoes and Pants please Sir."

* * * *

" I have no faith of your modern ' Science of Medicine.'
Do you think you can cure me ? "
" Oh don't worry, The Mule hasn't any faith in Veterinary Science but still, the Vet is able to cure him of any illness he may have."

* * * * *

" Is my son getting well ground in Pharmacy ? "
" I would put it even stronger than that," replied the Professor.
" I may say that he is actually standard on it."

* * * * *

" What happened ? " She, asked, as he got out of his automobile to investigate.

" Puncture," he said briefly.

" You ought to have been on the look out for this," was the helpful remark. " You remember the guide warned you there was a fork in the road."

*   *   *   *   *

Mrs. Teawhiffle : "*Did you change the Table napkins as I told You ?* "

New maid : " Yes'm. I shuffled 'em and dealt 'em out so's no one gets the same one as he had at breakfast."

*   *   *   *   *

Pete's Land lady said : " Pete, I am afraid I shall have to charge another 2 Shillings. You're such a big eater."

" For heaven's sake, don't do that " said Pete.

" I'm killin' myself already tryin' to eat what I'm payin' for now."

*   *   *   *   *

The Pharmacist, had to speak vehemently to the man loitering in the Store,

" No Smoking Please ".

" But, I, Just bought the Cigar here."

" *Look " pleaded the Pharmacist*

" We also sell Purgatives here, But,

You can't enjoy them on the premises."

*   *   *   *   *

" Surgeons " commented Dr. Osler " Should have an eagle eye, a Lion's heart and a Lady's hand."

A Surgeon had the first two, but when he was caught holding the third, there was trouble from the Lady's husband.

*   *   *   *   *

" Tell me candidly. Doc, do you think I'll pull through ? " asked the patient.

" Oh, You're bound to get well," *replied the doctor.*

" You can't help yourself. Statistics prove that out of hundred cases like yours 1 percent invariably recovers.

I've treated ninety-nine cases and every one of them died.

Why, man, alive, you can't die if you try."

*   *   *   *   *

" Let me warn you Nurse, the Man in Room 6 Ward 4 " the Doctor admonished the pretty nurse on duty. " He's a dangerous case."

" Why, Doctor ? Because he's so sick ? "

" No—because, he's almost well ".

*   *   *   *   *

Customer : " Is the Manager in ? "

Druggist : " No, he stepped out for lunch."

Customer : " Will he be in after lunch ? "

Druggist : " Why, no, that's what he went out after."

Upon reading the morning's paper Dr. Gopi was appalled to see his own Name erroneously listed in the Death Notice. He charged down to the Newspaper office and vigorously upbraided the Editor for falsely reporting his death.

" I'am awfully sorry ", the Editor told him " but it's too late to mend matters.

As you see, the paper is already out."

" Is that all you can do ? " Shouted the Doctor " Just sit there and tell me the paper is out ? "

" Well," replied the Editor calmly " There is one thing I can do for you Doctor, I can put you in the Birth Notice column tomorrow and give you a fresh start ".

* * * * *

" How did you get along in your action for compensation against the Man whose Dog bit you ? "

" I lost. He had a clever Lawyer who proved that I bit the Dog."

* * * * *

Mrs. Newdriver (to Garage Mechanic) : " They tell me I have a short circuit. Can you lengthen it while I wait ? "

* * * * *

The long suffering Patient was told by an eminent Surgeon, that he might have to submit to a fifth Abdominal Operation.

" But Doctor, why not put in a swing door on my Abdomen when you close it this time for you to open, when you land next on your sixth."

* * * * *

Said the Doctor to his rapidly recovering Patient. " Young Man, you owe your swift recovery to your wife's loving care."

" Very glad to hear it " came the prompt reply : " Since you admit it, you won't mind making the Cheque out to her ".

* * * * *

Ther'd been a lot of talk in a district about the new painless Dentist who'd set up his practice. But, a small boy ended that legend. " He is not painless at all." Said the youngster " I bit his finger when he put it in my mouth ; and he yelled just like any one else."

* * * * *

A physician who would not allow his Patients to enumerate the complaints but only extract answers to his queries was examining an old lady one day and asked,

" Do you have fever ? "—" Yes."

" Do you get head ache ? "—" Yes."

" Do you have shivering's at night ? "—" Yes."

CONNEMARA PUBLIC LIBRARY

"Do your teeth clatter, when you get the Shivering?"—"No!" replied the lady.
"I keep my teeth in a tumbler at night."

* * * * *

A man had swallowed an Artificial eye and was rushed to the Hospital.
The Surgeon who passed a Gastroscope to look into the Stomach remarked "I have stared into many Stomachs but this is the first time that a stomach stares at me."

* * * * *

Doctor, appreciating his son's action said:
"Well son, it was splendid of you to jump off the bridge and rescue your little friend:
What prompted you to do it?"
"Well, Dad—You know, he had my Skates on when he fell in."

* * * * *

The seller of a certain remedy for Nervous disease, has got this unsolicited testimonial.
"I cannot praise your Medicine too much; My wife used to be so nervous, that no one could sleep with her, but now, after taking your drowsy drops, any one can sleep with her."

* * * * *

A Physician picking up his Car at a garage, was amazed at the size of the repair bill.
After he recovered from his surprise, he commented casually to the mechanic.
"You fellows charge more for your work than we of the Medical Profession do."
"Well," said the mechanic, "We deserve it. You fellows have been working on the same old model since time began, but we have to learn a New model every year."

* * * * *

"Doctor, What would you prescribe for my Wife for flat foot?"
"Rubber heels" replied the Doctor,
"Rub-her-heels? With what?"

* * * * *

A member of faculty of a London Medical College was appointed Hony. Physician to His Majesty, King George and he announced that fact, proudly to his students by writing on the black board a notice.
When he returned to the Class in the after noon, he found written underneath his notice this "God save the King."

* * * * *

Doctor: "You may have Whisky, but it should be taken with hot water."

Patient : " How am I to take hot water, Doctor ? My wife won't allow me to take the Whisky."

Doctor : " Doesn't she give you hot water for shave ? "

The next day there was a frantic call on the phone from the wife.

" Doctor, please come at once, My husband is mad,
He wants to shave every few minutes."

* * * * *

Teacher : What is the half of Eight, Frank ? "
Frank : " Which way, Teacher ? "
Teacher : " What do you mean ? "
Frank : " On top or sidewise."
Teacher : " What difference does it make ? "
Frank : " Well, The top half of eight is 0,
But the half of eight side-ways is three."

* * * * *

" Hay, What time is it by your watch ? "
" Quarter to."
" Quarter to what ? "
" I don't know-times got so bad I had to lay off one of the hands."

* * * * *

" Did you mail those two letters I gave you, Son ? "

" Yes ma, at the post office, But I noticed that you'd put the two-cent stamp on the foreign letter, and the five-cent stamp on the City one."

" Oh, dear, what a blunder."

" But I fixed it all right, ma, I just changed the address on the envelopes."

* * * * *

A Medico despite office hours and Operations managed to father eight children. While he was in the Hospital one day his wife answered his phone. A woman patient had a great problem. " Does Doctor practise birth control methods ? "

" Not to my Knowledge " replied the wife.

* * * * *

Patient, to an Ophthalmologist,
" Doctor, I suffer a great deal with my eyes ;"
" Oh, how sad madam ; won't you suffer more without them."

* * * * *

" Doctor," said the Patient " my son has scarlet fever and he admits he kissed the pretty young cook ;

" Be calm " advised the Doctor, " you know how boys will be boys."

" But you don't understand, Doctor. To be frank with you, I've kissed that girl myself ;

" Well now, that is complicated, isn't it : " agreed the Doctor.

" Worse than that, I kissed my wife both that morning and that night."

The Doctor was now excited. " Dammit " he admitted, " I now'll get it too "—and I finish

" Jokingly I Joke
To Joke is no Joke,
To know a joke is no joke,
Not to know a joke is a joke
To coin a joke is no joke,
To copy a joke is no joke
To stand a joke is no joke,
And jokes quite not caught are best gulped gently."

If you think you are crazy, you are not ;
If you think everyone else is crazy, you are.

PART III

ASSOCIATION SUPPLEMENT

MADRAS CITY BRANCH

I. M. A.

Silver Jubilee Year 1959

CELEBRATIONS IN OCTOBER

SPORTS SYMPOSIA SOUVENIR

GALA ENTERTAINMENT & DINNER

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179. Dr. T. Nandagopal, M.B., M.S.,
53-B, Lloyds Lane,
Madras-14.

180. Dr. R. Nanjunda Rao, M.S.,
141, Brodies Road,
Madras-4.

181. Dr. P. Nanjundiah, L.M.P.,
11-A, Thorpe Road,
Madras-23.

182. Dr. N. S. Narasimhan, F.R.C.S.,
221, Thambu Chetty Street,
Madras-1.

183. Dr. S. T. Narasimhan,
8-F, Landons Road,
Madras-10.

184. Dr. K. V. Narayana Iyer,
21/A, Harris Road,
Pudupet, Madras-2.

185. Dr. K. Narayanamurthy, M.D.,
52, Mundakanniamman Koil Street,
Madras-4.

186. Dr. R. Narayanan, M.B.B.S.,
3/14, Ormes Road,
Madras-10.

187. Dr. S. Narayanaswamy Iyer,
10, Thirumalai Pillay Road,
Madras-17.

188. Dr. G. J. Narayanaswami Naidu, L.O.,
17, Hensman Road,
Madras-17.

189. Dr. D. Narayana Rao,
New Colony,
Chromepet,
Madras.

190. Dr. H. L. Narayana Rao, L.M.P.,
292, Thambu Chetty Street,
Madras-1.

191. Dr. K. L. Narayana Rao, M.B.B.S.,
389, Mint Street,
Madras-1.

192. Dr. S. Narayana Rao, M.B.B.S.,
Jagadambal Colony,
29/3, Lloyds Road,
Madras-14.

193. Dr. U. L. Narayana Rao,
494, Mint Street,
Madras-3.

194. Dr. P. Natesan, M.B.B.S.,
22/23, Nattu Pilliar Koil Street,
Madras-1.

195. Dr. P. S. Natesan, M.B.B.S.,
163-V, Palm Grove Colony,
Mylapore,
Madras-4.

196. Dr. K. Natarajan,
124, Mannarswamy Koil Street,
Royapuram,
Madras-13.

197. Dr. M. Natarajan, F.R.C.S.,
21-B, Nowroji Street,
Madras-10.

198. Dr. M. Natarajan, M.B.B.S.,
28, Third Main Road,
Madras-20.

199. Dr. S. Natarajan, L.M.P.,
26, Officers Lines,
Pallavaram P.O.

200. Dr. T. H. B. Nedungadi,
15, West Cott Road,
Madras-14.

201. Dr. N. Nemiraja, M.B.B.S., D.G.O.,
258, Linghi Chetty Street,
Madras-1.

202. Dr. Mrs. Padma Edwards, L.M.P.,
35, Jones Street,
Madras-1.

203. Dr. V. Padmanabhan, M.B.B.S.,
'Govardhan' Calvivaru Street,
Madras-4.

204. Dr. P. K. Padmanabha Rao, L.M.P.,
3, Bandi Venkatesa Naick Street,
Triplicane,
Madras-5.

205. Dr. P. M. Palani, M.D.,
300, Linghi Chetty Street,
Madras-1.

206. Dr. Lt. Col. K. G. Pandalay,
'Binfield', Poonamallee High Road,
Madras-10.

207. Dr. M. Parankusam,
46, Bazullullah Road,
T. Nagar,
Madras-17.

208. Dr. L. R. Parthasarthy, M.B.B.S., D.M.R.,
Medical Officer,
Southern Railway Hospital,
Perambur
Madras-11.

209. Dr. Mrs. Parukutty Raman, M.R.C.P.,
Rajasekhara Mudaliar Road,
Off Edward Elliots Road,
Madras-4.

210. Dr. A. Pattabhi, D. M. & S.,
63, Swami Naicken Street,
Chintadripet,
Madras-2.

211. Dr. P. G. Paul, M.B.B.S.,
3-A, Avenue Road,
Madras-6.

212. Dr. Paul Doraiswamy, M.B.B.S.,
13-A, Clemens Road,
Vepery,
Madras-7.

213. Dr. Mrs. Paul E. Doraiswami, M.B.B.S.,
13-A, Clemens Road,
Vepery,
Madras-7.

214. Dr. Miss. M. Pavana Bai,
19, 4th Main Road,
Gandhinagar,
Madras-20.

215. Dr. G. G. Prabhu, M.D.,
23, Main Road,
Royapuram,
Madras-13.

216. Dr. M. B. Prabhu, M.D., M.R.C.P.,
4, Gengu Reddy Road,
Egmore,
Madras-8.

217. Dr. M. G. Prabhu, M.D.,
4, Gengu Reddy Road,
Egmore,
Madras-8.

218. Dr. M. C. Pilliah Chetty,
113, Audiappa Naicken Street,
Madras-1.

219. Dr. S. Purushotham,
86, Govindappa Naicken Street,
Madras-1.

220. Dr. T. Radhakrishnan,
32, Bazullullah Road,
T. Nagar,
Madras-17.

221. Dr. K. Radhakrishnan, M.B.B.S., D.M.R.,
27, Hunters Road,
Madras-7.

222. Dr. P. Radhakrishna Rao, M.B.B.S.,
17, Muker Nallamuthu Street,
Madras-1.

223. Dr. G. Raghavachary,
15, Neelakanta Mehta Street,
T. Nagar, Madras-17.

224. Dr. K. Raghavachary, M.B.B.S.,
56, Nallathamby Mudaly Street,
Triplicane, Madras-5.

225. Dr. P. T. Raghavachary, M.B.B.S.,
100, T.P. Koil Street,
Triplicane, Madras-5.

226. Dr. T. Raghavan, M.B.B.S.,
15, Barnaby Road,
Kilpauk, Madras-10.

227. Dr. P. K. K. Raja, M.B.B.S.,
5, Egmore High Road,
Madras-8.

228. Dr. P. K. M. Raja, M.B.B.S.,
4, Purasawalkam High Road,
Madras-7.

229. Dr. K. Rajagopal, B.SC., M.B.B.S.,
4, Venkatakrishnier Road,
Madras-28.

230. Dr. M. S. Rajagopalan, L.M. & S.,
11, Raghaviah Road,
T. Nagar, Madras-17.

231. Dr. S. Rajagopalan, L.M. & S.,
Masilamani Street,
T. Nagar, Madras-17.

232. Dr. V. Rajagopalan, M.B.B.S.,
Parlakimidi Colony Lay out,
Madras-10.

233. Dr. R. V. Rajan, M.S., M.R.C.P.,
66, Gopathy Narayanaswamy Road,
T. Nagar, Madras-17.

234. Dr. S. Rajan,
Srinivasa Clinical Laboratory,
Khaleel Mansions,
Madras-2.

235. Dr. P. N. Rajappan, B.SC., M.B.B.S.,
2/27, Kattur Sadayappan Street,
Periamet, Madras-3.

236. Dr. N. Rajasekharan,
M.B.B.S., D.G.O.,
50, Pantheon Road,
Egmore, Madras-8.

237. Dr. Rajiah D'Paul, M.B.B.S.,
12-B, Ormes Road,
Kilpauk,
Madras-10.

238. Dr. V. Raju, B.SC., M.B.B.S.,
36, Kuppiah Chetty Street,
West Mambalam,
Madras-17.

239. Dr. Mrs. K. L. Ramachandran,
M.D., D.G.O.,
32, Vepery High Road,
Madras-7.

240. Dr. R. S. Ramachandra, M.B.B.S.,
94, Mowbrays Road,
Madras-18.

241. Dr. G. Ramachandra Murthy,
L.M.P.,
40/41, Pidariar Koil Street,
Madras-1.

242. Dr. M. Ramachandran, L.M.P.,
83, Royapettah High Road,
Near Luz,
Madras-4.

243. Dr. T. Ramachandra Pai, M.B.B.S.,
23, Harris Road,
Mount Road,
Madras-2.

244. Dr. C. R. Ramachandra Pillay,
M.D., M.R.C.P.,
3, West Cott Road,
Royapettah,
Madras-14.

245. Dr. A. Ramachandra Rao,
M.B.B.S., B.S.SC.,
24/1, V.P. Koil Street,
Tondiarpet,
Madras-21.

246. Dr. A. Rama Dass, M.S.,
D.M.O., Railway Hospital,
Perambur,
Madras-23.

247. Dr. T. S. Rama Dorai,
B.SC., M.B.B.S.,
10, Ramaswamy Street,
T. Nagar, Madras-17.

248. Dr. V. Rama Iyengar, L.M. & S.,
100, Santhome High Road,
Madras.

249. Dr. G. Ramakrishna, M.B.B.S.,
6, Harleys Road,
Madras-10.

250. Dr. P. Ramakrishna Mudaliar,
M.D., T.D.D.,
35, Srinivasanagar,
Chetput,
Madras-31.

251. Dr. A. S. Ramakrishnan, M.B.M.S.,
12, Marshalls Road,
Egmore, Madras-8.

252. Dr. C. V. Ramakrishnan,
M.B.B.S., T.D.D.,
Tuberculosis Chemo-Therapy
Centre,
Govt. T.B. Institute,
Chetput,
Madras-31.

253. Dr. G. V. Ramakrishnan,
B.SC., M.B.B.S.,
3, South Street,
Sriramnagar,
Madras-18.

254. Dr. M. S. Ramakrishnan, M.S.,
152, Brodies Road,
Mylapore, Madras-4.

255. Dr. S. Ramakrishnan, L.M.P.,
44, Gangathareeswarar Koil Street,
Purasawalkam,
Madras-7.

256. Dr. V. N. Ramakrishnan, B.SC., M.B.B.S.,
Muktha Gardens,
41, Spur Tank Road,
Madras-31.

257. Dr. K. U. Ramakrishna Rao,
4/3, Iyah Mudaly Street,
Chintadripet,
Madras-2.

258. Dr. T. T. Ramalingam, M.S.,
Chelford,
Police Commissioner Office Road,
Madras-8.

259. Dr. A. S. Ramamurthy,
30, Venkatanarayana Road,
T. Nagar, Madras-17.

260. Dr. B. Ramamurthi, M.S., F.R.C.S.,
2nd Main Road, C.I.T. Colony,
Mowbrays Road,
Madras-18.

261. Dr. V. Ramamurthi, M.B.B.S.,
1, South Mada Street,
Mylapore, Madras-4.

262. Dr. V. Ramanamma,
House Surgeon Quarters,
Women & Children Hospital,
Madras-8.

263. Dr. Ramani Sivaraman, M.B.B.S.,
Madras Medical College,
Madras-3.

264. Dr. C. R. Ramanathan, B.SC., M.B.B.S.,
4, Kalyan Mahal,
Off Elliots Road,
Madras-4.

265. Dr. T. R. Ramanathan, M.B.B.S.,
Superintendent,
Monegar Choultry,
Madras-1.

266. Dr. K. Ramanujam, M.B.B.S.,
8, Neelakanta Mehta Street,
Madras-17.

267. Dr. V. P. Rama Pandia Raja, M.B.B.S.,
90, Barbers Bridge Road,
Mylapore, Madras-4.

268. Dr. B. Rama Rau, B.SC., M.B.B.S.,
155/157, Poonamallee High Road,
Madras-10.

269. Dr. C. Rama Rao, L.M.P.,
325, Mint Street,
Madras-1.

270. Dr. C. E. Rama Rao, M.B.B.S.,
29, Govindappa Naick Street,
Madras-1.

271. Dr. K. Rama Rao, L.M.P.,
216, Govindappa Naick Street,
Madras-1.

272. Dr. S. Ramaswamy,
169, Poonamallee High Road,
Madras-10.

273. Dr. S. R. Ramaswamy,
207, Poonamallee High Road,
Madras-7.

274. Dr. S. S. Ramaswamy,
139, Govindappa Naick Street.
Madras-1.

275. Dr. S. J. Ranga, L.M.P.,
Kelyvaram Doraiswami Pillai Street,
Tambaram West.

276. Dr. Lt. Col. V. Rangachary, M.S., D.O.M.S.,
155/1, Lloyds Road,
Royapettah,
Madras-14.

277. Dr. A. Ranganathan, L.M. & S., B.S.SC.,
Health Officer,
Public Health Unit,
Poonamallee.

278. Dr. C. Ranganathan, M.B.B.S.,
53, Dr. Alagappa Chettiar Road,
Madras-7.

279. Dr. K. V. Ranganathan, M.B.B.S.,
Sivasankara Mudaliar Street,
Kilpauk,
Madras-10.

280. Dr. D. V. Rangaswami, L.M.P.,
Kelyvaram Doraiswamy Pillay Street,
Tambaram West.

281. Dr. P. N. Rangiah, M.D.,
2, Thirumurthi Street,
T. Nagar, Madras-17.

282. Dr. K. Rathnakar Baliga, M.B.B.S.,
16, Parish Venkatachala Iyer Street,
Madras-1.

283. Dr. R. Rathnaswamy, M.B.B.S., T.D.D., 16, Police Commissioner Office Road, Egmore, Madras-8.

284. Dr. M. Rathnavelu, L.M. & S., 43, North Mada Street, Madras-4.

285. Dr. P. D. Reddy, 52, Poonamallee High Road, Madras-7.

286. Dr. T. K. Rithuparnam, M.B.B.S., D.M.R., D.M.R.T. (Lond.), 1-A, Park Side Road, Chetput, Madras-31.

287. Dr. Mrs. T. Rodrigues, L.M.P., 102, East Mada Street, Royapuram, Madras-13.

288. Dr. A. Rukmaniamma, L.M.P., 26, Ramakrishnan Street, Madras-1.

289. Dr. C. S. Sadasivan, M.S., 12, Bishop Waller Avenue, Mylapore, Madras-4.

290. Dr. F. A. Saldhana, 10, Victoria Crescent Road, Egmore, Madras-8.

291. Dr. Miss M. Samanthakam, M.B.B.S., 135, Govindappa Naick Street, Madras-1.

292. Dr. K. H. Sambandan, L.R.C.P., 14, Barnaby Road, Madras-10.

293. Dr. P. S. Sambandam, L.R.C.P., M.R.C.S., 'Ashok Vihar,' Madras-3.

294. Dr. K. S. Sampath, M.B.B.S., 9, Venkataroyan Lane, Madras-3.

295. Dr. S. K. Sampath, 123, Lloyds Road, Madras-6.

296. Dr. V. Samuel, L.M.P., 40, Tana Street, Madras-7.

297. Dr. K. S. Sanjivi, M.D., 56, St. Marys Road. Teynampet, Madras-18.

298. Dr. C. Sankaran, M.B.B.S., 'Karpaka Vilas', Nadu Street, Madras-4.

299. Dr. K. S. Sankaran, M.B.B.S., 20, Ramanujam Street, T. Nagar, Madras-17.

300. Dr. M. N. Sankaran, M.B.B.S., 23, Spur Tank Road, Madras-31.

301. Dr. R. Sankaran, L.M.P., 6, Rajabathar Mudaliar Road, T. Nagar, Madras-17.

302. Dr. R. Sankaran, L.M. & S., C.I.T. Colony, 7, Mowbrays Road, Madras-4.

303. Dr. V. Sankaran, M.S., 19, Justice Sundaram Iyer Road, Mylapore, Madras-4.

304. Dr. P. Sankaranarayana, 60, Madhavaram High Road, Madras-11.

305. Dr. M. Santhosham, M.B.B.S., 66, Egmore High Road, Egmore, Madras-8.

306. Dr. Mrs. T. Saraswathy, M.B.B.S., 16, Thackers Street, Vepery, Madras-7.

307. Dr. Miss R. Sarguna, 233, Pycrofts Road, Madras-14.

308. Dr. Miss A. Saroja, M.B.B.S., 45, Mowbrays Road, Madras-18.

309. Dr. D. Sarojini, M.B.B.S., 40, Hunters Road, Vepery, Madras-7.

310. Dr. U. Sarvothama Rao, M.B.B.S., 10, Purasawalkam High Road, Vepery, Madras-7.

311. Dr. M. A. Sayairam, 9, 1st Street, Zackriah Colony, Kodambakkam, Madras-24.

312. Dr. A. B. Scott, M.B.B.S., 10, Casa Major Road, Egmore, Madras-8.

313. Dr. V. S. Selvapathy, D.M.S., T.D.D., 74, Venkatesapuram, Madras-23.

314. Dr. T. Selvasekharan, L.M.P.,
338, Mint Street,
Madras-1.

315. Dr. M. S. Seshadri, M.B.B.S., D.G.O.,
20, Ganesh St.,
Gopalapuram, Madras-6.

316. Dr. U. M. Shah, M.B.B.S.,
187, China Bazaar Road,
Madras-1.

317. Dr. T. C. Shanker, M.B.B.S.,
45, 3rd Main Road,
Gandhinagar, Madras-20.

318. Dr. Shantha Arogyadoss,
M.D., D.G.O.,
17, West Mada Church Street,
Royapuram, Madras-13.

319. Dr. Miss P. Shantha, M.B.B.S.,
35, Thandavaroya Mudaly Street,
Madras-21.

320. Dr. V. Shantha, M.D., D.G.O.,
Cancer Institute,
Canal Road,
Madras-20.

321. Dr. Mrs. E. Sharada, B.SC., M.B.B.S.,
13/6, Jeremiah Road,
Vepery, Madras-7.

322. Dr. Mrs. H. M. Sharma,
M.B.B.S., B.S.SC., H.P.H.,
Professor of Preventive and Social Medicine,
Madras Medical College,
Madras-3.

323. Dr. Lt. Col. T. S. Shastry, M.B.C.M.,
37, Venkatanarayana Road,
T' Nagar, Madras-17.

324. Dr. N. B. Shetty, L.M.P.,
199, Poonamallee High Road,
Madras-7.

325. Dr. P. Shiva Rao, M.B.B.S.,
494, Mint Street,
Madras-3.

326. Dr. P. Shanmugam, M.B.B.S., L.O.,
33, Pidariar Koil Street,
Madras-1.

327. Dr. R. Shanmugam,
M.B.B.S., D.T.M.,
Retired Civil Surgeon,
18, Dewan Rama Iyengar Road,
Vepery, Madras-7.

328. Dr. B. Sitharam, M.B.B.S.,
153, Lloyds Road,
Royapettah,
Madras-14.

329. Dr. K. Sivaramakrishnayya,
1, Lodi Khan Street,
Madras-17.

330. Dr. K. Sivarama Mudaliar, M.B.B.S.,
Madras Medical College,
Madras-3.

331. Dr. A. M. Sivaraman, L.M.P.,
105, Seven Wells Street,
Madras-1.

332. Dr. Sogra Bi, M.B.B.S.,
1/235, Angappa Naicken Street,
Madras-1.

333. Dr. E. Somasekhar, F.R.C.S.,
Khader Nawaz Khan Road,
Madras-6.

334. Dr. T. S. Somaskanthan, L.M. & S.,
2/78, Broadway,
Madras-1.

335. Dr. Miss Soona J. Irani,
Roshin,
Off Cathedral Road,
Madras-6.

336. Dr. K. Sourirajan, M.B.B.S.,
13, Rangier Street,
T. Nagar, Madras-17.

337. Dr. U. Sridhar Rao, M.B.B.S.,
290, Thambu Chetty Street,
Madras-1.

338. Dr. S. Srinivasachari, M.B.B.S.,
8, Usman Road,
T. Nagar, Madras-17.

339. Dr. E. C. Srinivasan, D.O.M.S.,
32, Broadway, Madras-1.

340. Dr. E. V. Srinivasan, M.B.C.M.,
32, Broadway, Madras-1.

341. Dr. L. V. Srinivasan, M.B.B.S.,
16, Usman Road,
T. Nagar, Madras-17.

342. Dr. M. Srinivasan, M.B.B.S.,
127, Lloyds Road,
Madras-6.

343. Dr. N. Srinivasan, D.O., D.O.M.S.,
84, Pondy Bazaar,
Madras-17.

344. Dr. P. T. Srinivasan, M.B.B.S.,
41, A. V. N. Dass Road,
Mount Road,
Madras-2.

345. Dr. T. M. Srinivasan, M.B.B.S., D.L.O.,
1, Nageswara Rao Road,
T. Nagar, Madras-17.

346. Dr. V. Srinivasan, M.S.,
5A, Ponnu Somasundara Mudaly Street,
T. Nagar, Madras-17.

347. Dr. B. Srinivasa Rao,
476, Thiruvothiyur High Road,
Madras-21.

348. Dr. U. Srinivasa Rau, M.B.B.S.,
290, Thambu Chetty Street,
Madras-1.

349. Dr. A. Srinivasalu Naidu, M.D.
54, Harrington Road,
Chetput,
Madras-31.

350. Dr. U. Sripathi Rau, M.B.B.S.,
494, Mint Street,
Madras-3.

351. Dr. Capt. G. Sriramulu, L.C.P.S.,
92, Armenian Street,
Madras-1.

352. Dr. K. Subba Rao, M.B.B.S.,
65, North Tank Square,
Madras-5.

353. Dr. S. Subbulakshmi, M.B.B.S.,
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T. Nagar, Madras-17.

354. Dr. A. Subhadra, B.A., M.B.B.S.,
56, Venkatesapuram,
Ayanavaram, Madras-23.

355. Dr. Subramania Gopalakrishnan, M.B.B.S.,
12/A, Visweswarapuram,
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356. Dr. B. R. Subramaniam, M.B.B.S.,
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Madras-1.

357. Dr. C. K. Subramanian, M.B.B.S.,
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358. Dr. N. Subramaniam,
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Madras-10.

359. Dr. P. S. Subramaniam, B.SC., M.B.B.S.,
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360. Dr. R. Subramaniam, M.D., M.R.C.P.,
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361. Dr. S. Subramaniam, D.M. & S.,
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362. Dr. T. S. Subramaniam, F.R.F.P.S.,
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Madras-1.

363. Dr. V. S. Subramaniam, M.S.,
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Madras-8.

364. Dr. V. C. Sudarsanam, M.B.B.S., D.O.
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Madras-1.

365. Dr. B. V. Sundara Babu, M.B.B.S., D.P.H. (Cal.),
54, Shenoynagar,
Madras-30.

366. Dr. C. S. Sundaram, M.B.B.S.,
P.A. to Chief Medical Officer,
Southern Railway Office,
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367. Dr. V. Sundaram, M.B.B.S.,
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368. Dr. D. Sundarraj, M.B.B.S.,
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Madras-19.

369 Dr. C. R. Sundararajan, M.B.B.S.,
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370. Dr. R. Sundararajan, M.B.B.S.,
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Madras-8.

371. Dr. S. Sundararajan, M.B.B.S.,
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Madras-1.

372. Dr. S. Sundararaman, M.B.B.S.,
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373. Dr. B. M. Sundaravadanam, B.A., M.B.B.S., F.R.C.S., L.R.C.P.,
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374. Dr. Lt. Col. D. K. Sundaresan, M.D., D.T.M. & H., L.D.P.H. (Dub.), 24, Jambulinga Naicken Street, Madras-6.

375. Dr. (Major) M. R. Sundaresan, L.M.P., 7, Raja Street, Madras-17.

376. Dr. L. Sundara Doss, M.B.B.S., 442, Mint Street, Madras-3.

377. Dr. R. Surya Rao, L.M.P., 20, Arunachala Achari Street, Chepauk, Madras-5.

378. Dr. Susan Jacob, M.B.B.S., 37, Gollavar Agraharam Road, Madras-21.

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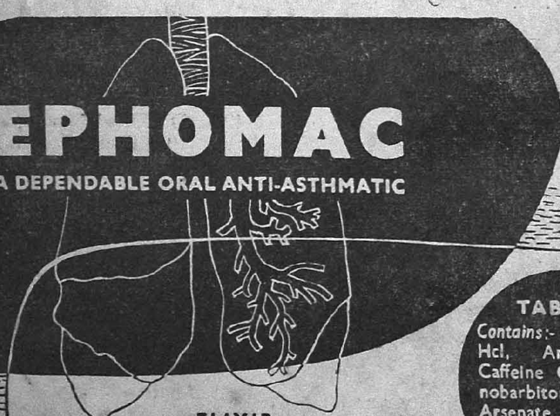

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