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Editor:
Dr. P.K. Sen

Co-Editors:
Dr. M.D. Deshmuka
Dr. N.L. Bordia

Associate Editors :
Dr. H.B. Dingley
Dr. S.P. Pamra

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News & Note * Abstracts

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The Indian Journal of Tuberculosis

Vol. XXV

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No. 1

ENTERS ITS SILVER JUBILEE YEAR

The Founding Fathers of the Tuberculosis Association of India envisaged the publication of a Journal to disseminate knowledge about tuberculous diseases and to serve as a communication medium for those engaged in anti-tuberculosis work. In the early years of the Association, this objective was sought to be partially served by the Indian Medical Gazette, the sponsors of which undertook to bring out one issue of the Gazette every year as a Tuberculosis Number. As the activities of the Association developed, particularly after Independence, and the tuberculosis problem began to receive greater attention in official and non-official quarters, the Association felt that it was necessary to have a Journal of its own. The Association started the Indian Journal of Tuberculosis in 1953. Its aim was to serve as the official organ of the Association and to facilitate workers to keep abreast of the fast-changing and revolutionary developments in tuberculosis control work.

It is true that the annual national conferences on tuberculosis and chest diseases do provide opportunities to our workers, who assemble from all parts of India every year in different State capitals, for exchanging notes about their experiences. But it would be conceded that a good number of workers may not be able to attend the national conferences. In recent years some of our State Associations have also been holding state-level conferences and quite a few of them have been organising seminars also. These, however, may not get the benefit of the counsel of seasoned senior workers from other parts of the country and abroad. Moreover, conferences and seminars cannot serve as substitutes for a Journal. The Journal publishes a wide variety of articles including original work by contributors on various aspects of tuberculosis and chest diseases in general. This may not always be possible in conferences where very often it is difficult to accommodate even seasoned workers. The only medium which can bring out what is best in our workers for the benefit of the profession is the Journal. The Indian Journal of Tuberculosis has been endeavouring to fulfill this objective to the best of its ability.

Over the years, the Journal has built up a reputation as a publication of high standard. Its readership covers workers in India and a number of institutions and organisations outside. A vast developing country, with its fast increasing population, regional variations in culture, economic status and mode of life, India has been making gallant efforts to meet the challenge of its colossal tuberculosis problem. Its development programmes can be said to be a purposeful and bold adventure. The work done in some institutions in our country has been recognised internationally as pioneering. Developed countries

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where tuberculosis has been controlled or contained have considerable amount of accumulated knowledge about tuberculosis control work and this can be profitably used in India and other developing countries. Tuberculosis is not an internal problem of any country. It is international. Therefore every country has something to give to and take from others in dealing with this disease. No country can be safe from tuberculosis so long as it exists in any part of the world and therefore no one can rest on the oars in a state of complacency. Developed countries can also profit from the knowledge and experience available in countries like India in matters tuberculosis. The Indian Journal of Tuberculosis can be counted upon to play its part in this regard.

A pragmatic programme to counter any problem can be said to have five different stages, viz, exposition, development, climax, anti-climax and catastrophe or *denouement*. The "actors" at every stage have to plume themselves to fit into the general picture without faltering so as to achieve the ultimate goal, particularly the final one. Catastrophe can be averted if timely steps are taken to achieve the *denouement*. The Association as the pioneer in the tuberculosis movement in our country exposed the magnitude of the problem before the Health Survey and Development Committee in 1944 and after and thanks to the support it has been receiving, contributed its mite for the development of the tuberculosis control programme. The climax came when Government introduced the National Tuberculosis Control Programme in 1962. The Association sensed the tendency towards an anti-climax when the national conference in 1973 and also this journal voiced concern that the control programme was not working satisfactorily and moved the Union Health Minister to have the position examined by an expert committee. The authors of the sixth Plan can be expected to take note of the findings of the expert committee and the suggestions made by this Association to streamline the forthcoming Plan. A popular publication like the Indian Journal of Tuberculosis, which is the only medium in India to voice the views of the specialists and the public, has an important role to play in the future shape of things.

A number of our Tuberculosis Associations have been engaged in constructive work with the object of assisting Government in implementing the control programme. Tuberculosis Centres, Clinics and District Control Units have been doing remarkably good work. The work carried out in the Chemotherapy Centre, Madras has attracted world attention. The studies undertaken by the New Delhi TB Centre of the Association and other prestigious institutions have also won recognition. The Journal serves as the medium to put across for the benefit of our workers what our institutions and Associations are doing.

Today, tuberculosis has ceased to be the problem of only individual patients. It is the problem of the entire community and is being tackled in a most courageous manner in India through the control programme. The social approach through voluntary and effective Care Committees in Delhi has vast inherent humanitarian potentialities which is *a sine qua nonto* promote the anti-tuberculosis movement. *Shibir*s as the means for providing a comprehensive tuberculosis service to the people in the rural areas, initiated by Dr. M. D. Deshmukh in Maharashtra, reminds one of Mahatma Gandhi's *padayatras* to promote mass-contact movement. This programme has been taken up by

Karnataka; it should also be taken up by other States to fill up the gaps in the implementation of the control programme. The Journal has thrown open its pages to popularize the utility and practicability of this programme.

With this issue the Indian Journal of Tuberculosis is in its Silver Jubilee year. The tendency today is to consider tuberculosis as one of the many chest diseases and tuberculosis services are being integrated with the general health services. The nomenclature of our national conferences has been changed of late as conferences on tuberculosis and chest diseases. The Editorial Board considered the question as to whether the Journal also could be named as Indian Journal of Tuberculosis and Chest Diseases. As tuberculosis still continues to be a major public health problem in India demanding primary attention of every one the Board decided that the Journal should continue as Indian Journal of Tuberculosis for the present.

The Editorial Board takes this opportunity to pay its tribute to the past members of the Board, particularly late Dr. P. V. Benjamin who laid the Journal's foundation and nurtured it in its early years. The Board is grateful to all those who have been helping the publication of the Journal and is confident that this help will be available in future also. Its thanks are due in an abundant measure to the contributors of articles and it is hoped that they, particularly the young workers, will continue to help us to make the Journal an influential and authoritative medium for disseminating new knowledge, fostering new thinking and shaping new policies and programmes. It thanks the Tuberculosis Association of India for continuing this publication and the State Associations who have enrolled, at their cost, District TB Officers as subscribers and appeals to all State Associations to do likewise. Last, but most important, the Board thanks the subscribers and advertisers for their cooperation and patronage and hopes to receive from them the same encouragement in future also.

FIFTEEN YEARS PROGRESS OF NATIONAL TUBERCULOSIS PROGRAMME

N.L. BORDIA*

Introduction

Even though the advanced countries of the world have been attempting to control tuberculosis from the beginning of this century the progress was very slow. In the earlier years the developed nations concentrated on treatment of patients in sanatoria/hospitals, isolation and improvement in the standard of living of their population. But with the discovery of anti-TB Chemotherapy and its extensive application, problem of tuberculosis began to get reduced quickly. In India we had no clear idea of the real problem of tuberculosis in the country as a whole. The National Tuberculosis Survey was therefore undertaken through the ICMR (1955-1958).¹ By that time information on prevalence of tuberculous infection had become available through tuberculin testing surveys as a prelude to mass BCG vaccination. The survey showed that tuberculosis was almost equally prevalent in the rural areas as in the urban. 1.4 to 1.7 per cent of the population was found to have lesions in the lungs detected on miniature X-ray consistent with the diagnosis of active tuberculosis and a quarter of these were excreting tubercle bacilli. Tuberculosis was found more frequently in slum dwellers in low-socio-economic groups. In the males, tuberculosis prevalence increased with increasing age while in the females the disease was on the whole less than in the males and the peak of prevalence was reached at about 35 years of age. There was a fall in the prevalence rates in higher age groups in women.

New Thinking on National Tuberculosis Problem

Results of National Tuberculosis Survey lead to the necessity for a new approach to the strategy for tuberculosis control, a strategy concentrating mostly on the problem of the community rather than the individual patient. Available means and techniques were to be applied on a mass scale giving higher priority to quantity than to quality. By then anti-TB drugs were available which were acceptable and which could be taken by all patients themselves in their own homes without much supervision and which the Government could afford to provide free of cost to all the patients.

Background of Existing Organisation of Health Services

Health is a State subject. Responsibility for

*Emeritus Professor of Tuberculosis M.G.M. Medical College Indore M.P.

implementation of control measures rests mainly with the State Governments. The Government of India coordinates the health activities so that uniform development takes place. Even then, resources of various Governments in manpower particularly trained personnel, finances, etc. vary from state to state. The Central Government provides plan assistance for new development *during the plan* period only. Later, all responsibility for running these services is of the State Governments.

Planning for Tuberculosis Control

During these years, "Planning for development" through its five year plans had been accepted as the policy of the Government of India which aimed at evolving a healthier and happier society by rapid socio-economic development as quickly as possible, say in 15 to 20 years. Broadly speaking national health plans covered expansion of rural health services, training of personnel, control of communicable diseases, provision of safe water supply and improvement of environmental sanitation etc. Family welfare programme had to be given the highest priority so that the gains of development plans were not neutralised by disproportionate rise in the population. The basis of the health development plans formulated then, still continues on the same lines. Though development of tuberculosis services found a place in the health plans but it could not receive the same priority as was given to malaria, small pox, water supply, development of rural health services and family welfare (Family Planning) etc. This was mainly because epidemiology of tuberculosis was such that it could not be eradicated or even controlled as quickly as malaria and small pox could be.

Development of the National Tuberculosis Programme, Changes in Methods of National Tuberculosis Programme due to Newer Discoveries.

With large scale use of anti-TB drugs, specially Isoniazid, mortality due to tuberculosis had rapidly come down throughout the world. It became obvious that the slow progress by conventional methods like improvement in the standard of living, isolation and rest cure etc. could be enhanced by large scale use of anti-TB drugs. By that time treatment of patients in the developed countries of the world was being undertaken in hospitals and sanatoria. Many tuberculosis workers in India had experienced.

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that drugs could be equally efficacious under domiciliary conditions. The Government of India, therefore, established the Tuberculosis Chemotherapy Centre, Madras in 1955 for evaluating the efficacy of newly discovered anti-TB drugs on domiciliary basis in comparison to treatment with the same drugs in the hospital. By 1950 BCG vaccination had also been introduced and was found to be easily applicable to the entire country and was acceptable to the people. BCG vaccine laboratory was established in 1949 to produce this vaccine not only to serve the national needs but also to supply the South East Asian countries. A research unit for BCG trial and a study of the time trends of tuberculosis with minimum treatment service had been established at Madanapalle in South India under the ICMR. As these studies were being conducted, the Government of India also established in 1959 the National Tuberculosis Institute at Bangalore to undertake applied research in developing a sound organisation and administrative machinery and to study techniques and simple methods which could be applicable under Indian conditions, specially rural parts of the country which comprises 82 % of the population. Training of the personnel was an essential part of the activity of the Institute. Methods of treatment by modern anti-TB drugs which could be undertaken in the home and which in the long run will reduce the problem of tuberculosis in the country were to be evolved. The W.H.O. promised to give technical assistance and the UNICEF was to provide essential equipments like X-ray equipment, transport, BCG kits,

X-ray films etc. for this purpose. Later the Institute developed the working of district tuberculosis programme as is now being undertaken from 308 centres in the same number of districts. By that time, the first five year plan had been completed. In that plan Mass BCG vaccination campaign was given the utmost priority. During the five year plan period 1951-56, 87 million persons were expected to be tuberculin tested and those found non-reactors were to be vaccinated. Against this target, 71.5 million were tuberculin tested and 24.5 million were vaccinated. This proved the feasibility and acceptability of mass BCG vaccination campaign in the whole country. Though 200 TB clinics were to be newly established, actually only 55 could be developed or upgraded by 1956. These Centres were supplied with 100 MA X-ray machines with odelca cameras and culture laboratory equipment at the cost of the Government of India. The State Governments were only to provide personnel and the buildings. The weakness of the organisation in the States became apparent when culture facility had totally failed to develop at any centre and apart from carrying out microscopic examination of sputum, no other work was undertaken. Even the miniature X-ray units could not be fully utilised due to shortage of trained personnel. Three training Centres viz. at New Delhi, Patna and Trivandrum were established and they were assigned the task of training of personnel and to develop methods for efficient anti-TB service as applicable in their areas. As the available beds were inadequate, 5,000 additional beds were established. (Table I).

Table I

*Components for Development Plans for Tuberculosis
First Five Year Plan*

		Targets	Achievements
1.	Mass BCG vaccination campaign.	87 million tests.	71.5 million tests 24.5 vaccinated.
2.	Establishment of TB clinics.	200	55
3.	Establishment of Training Centres.	3	3
4.	Provision of TB beds.	5000	5000
5.	Research	Madanapalle field research unit.	1951
	Central funds.	TB Chemotherapy Centre, Madras.	1956
		National TB survey.	1955-58
	BCG vaccination and training centres were centrally aided.		

Table II

Second Five Year Plan 1956-1961.

	Targets.	
1. Mass BCG vaccination campaign.	170 million	population coverage.
2. Establishment/ upgrading of TB clinics	180	80
3. Provision of training and demonstration centres.	10	3
4. Provision of TB beds.	4000	4000
5. Research (Central) National Tuberculosis Institute Research & Training.	1959	
6. Rehabilitation Centres.	8	6

In the second five year plan viz. from 1956-61 the basis of the plan remained the same. More district TB centres were established and three more Centres for training and demonstration were established though the target was 10. Research found an important place and was expanded more substantially. Six Rehabilitation Centres at Delhi, Lucknow, Amargarh (Gujarat), Hyderabad, Bangalore etc. were to be established. (Table II).

A major change took place with the beginning of the third five year plan when the pattern of assistance to the States was revised and liberalised. Central assistance on non-recurring expenditure like construction of buildings and supply of equipment was fixed at 75% while the State Government was to bear only 25 % of the expenditure on this account. The State Governments however, had to meet 50 % of the running cost during the plan period of five years.

A total expenditure of 118 million rupees was provided in that plan. At the end of that plan period the responsibility for running all these Centres had to be borne entirely by the State Governments. During the Third Five Year Plan 119 TB clinics or District TB Centres were established, 68 million persons were tuberculin tested and those found eligible were BCG vaccinated. Against the target of the establishment of five TB training and Demonstration Centres, eight were established. Only the States of Assam, and Madhya Pradesh remained without Training Centres. The establishment of TB beds which had been given high priority in the previous plans was given a low priority. Against a target of 5,000 beds only 1571 beds were estab-

lished between 1961 and 1966. (Table III) Attempt was made to provide Mobile X-ray units, but against a target of 25, only five could be established. It had become clear by then that Rehabilitation Centres were not necessary and therefore were taken out of the plan. In 1963 a closer evaluation of the third five year plan achievements revealed certain lacunae for which corrective action was taken. For example, it had been observed that the State Governments who were supposed to provide anti-TB drugs were not able to give drugs to their patients. The result was that inspite of there being microscopes, x-ray units, trained staff and patients, many detected cases went without drugs. From the year 1963 anti-TB drugs were also supplied to TB clinics undertaking domiciliary treatment as free assistance by the Government of India without any matching contribution from the States. This was really in the middle of the Third Five Year Plan. There was no rigidity about utilisation of plan provisions and change could be made in the plan in its middle so that it succeeds.

The Fourth Five Year Plan (Table IV) initiated in 1967 included a few more central schemes. Two Regional Organisations, one in the North with the New Delhi TB Centre as its Headquarters and one in the South at N.T.I. Bangalore had been established even in the Third Five Year Plan period as advance action of the Fourth Plan. Four more of such Centres were to be provided in the Fourth Five Year Plan. Each Centre was to have a senior Medical Officer of the status of a Tuberculosis Control Officer, senior X-ray and Laboratory Technician, a Treatment Organiser and a Statistician with

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Table III

Third Five Year Plan 1961-1966 (Total Amount : Rs. 11.8 crores).

	Targets	Achievements
1. Mass BCG vaccination	100 million tests	68 million
2. TB clinics/Distt. TB Centres.	200	119
3. State Centres or Training Centres.	5	8
4. Beds for TB patients.	5000	1571
5. Mobile X-ray units	25	5
No rehabilitation Centres.		

Pattern assistance 75% Central funds on non-recurring and 50% on recurring in the plan period. Two regional organisations established as advance action though their development was for the fourth plan.

Table IV

*Fourth Plan *1967-1972*

Central schemes.	Targets.	
1. National Tuberculosis Institute (Training & research)	20 lakhs	on expansion.
2. Regional organisations.	Additional four	Nil
3. Upgrading of training centres to the level of the National Institute.	Two	Nil
4. Anti-TB drugs.	Four crore	3 crores
5. Drug supply clinics run by voluntary bodies.	One crore	0.6 crore
6. TB clinics or Distt. TB centres.	200	120
7. Training and demonstration Centres.	4	2
8. Tuberculosis beds.	5000	2000

Note: One Years' Plan holiday.

ancillary staff. Their main job was to supervise and promote the development of District TB Centres and mobilise and brief the personnel and to evaluate the working of the National TB programme in the respective regions. It was also envisaged to upgrade two TB and Demonstration Centres so that they could undertake training of the entire teams for the National

TB programme as was being undertaken at the NTI Bangalore. Provision of anti-TB drugs to TB Clinics or District TB Centres run by voluntary bodies was also provided in the scheme. This has been a very useful scheme as all TB Clinics undertaking domiciliary treatment could be supplied with anti-TB drugs from this grant. In this plan period BCG vaccination was to be

Table V

Fifth Five Year Plan (1972-1977) (Amount: Rs 8.25 crores)

Centrally scheme 100% financed by Central Government.	
I. The supply of anti-TB drugs films and BCG vaccine.	8.25 crores.
State Schemes. No Central Assistance.	
TB Clinics T.B. Centres. TB beds.	9 crores State Sector.

integrated with the District Centres in the States. It was also hoped that all the then existing 336 districts of India would have District TB Centres, at least one in each. All States should have at least one Training and Demonstration Centre. Pattern of Central assistance in the Fourth Plan was 50% on State scheme while the Government of India bore 100% cost on Central Schemes.

Fifth Five-Year Plan (Table V)

When the Fifth Five Year Plan was initiated a serious thinking took place on development of TB services. It was decided that financial responsibility of development of new District TB Centres, Training and Demonstration Centres, supply of anti-TB drugs, films and BCG vaccine etc. should be entirely of the Government of India. In other words the TB programme in the plan period as far as new development was concerned became a centrally sponsored scheme. It was expected that the States would make use of the Central assistance wholeheartedly, since they had only to provide building and personnel to run the services and that too at the expense of the Government of India.

Unfortunately the State Governments were more busy with other health schemes like smallpox eradication, family planning programme, and running general health services etc. Trained personnel were not available and the buildings could not be constructed as expeditiously as needed. The result was that the Central assistance could not be utilised as well as it should have been. Today we do not know how much out of 8.25 crores provisions in the Central Sector and 9.0 crores provided in the State sector of this plan provision has been utilised. Though anti-TB drugs, films and BCG vaccine were made use of fairly well, even then the amount provided in the budget of the Government of India for this purpose could not have been fully utilised. Thus

the Fifth Plan was not such a success as was expected.

TB Clinics had formed the basis of domiciliary treatment services in the country all these years. Beds were few considering the size of the population. Most of the old existing TB clinics were upgraded into district TB centres or new district TB centres were established, the aim being that there must be at least one centre in each district with an average population of about 1.5 million. Today these centres form the core of the national tuberculosis programme. From these centres case detection and treatment service is organised throughout the district from health care institutions of various denominations. The main objective is that case detection and treatment service should be available to all symptomatics even at the periphery.

By now, 610 TB clinics of different denominations are existing in the country, 308 of these are equipped with miniature x-ray units and are functioning as upgraded district tuberculosis centres. The number of tuberculosis beds stands at 42,500 against 35,000 in 1962. 224 million persons have been vaccinated with BCG since the inception of the campaign in 1950. Sixty nine districts have no district TB Centres out of the 379 districts (Table VI).

Major Difficulties in the Successful Implementation of the National Tuberculosis Programme

I. Tuberculosis could not receive that priority which some other communicable diseases and family welfare (family planning) programmes received. There were no clear cut instructions and compulsion to peripheral health centres for implementation of the tuberculosis programme. The peripheral health centres did not utilise the available facilities for case detection and treatment supervision on treatment was lacking.

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Table VI

Progress in Development of Tuberculosis Clinics/District Centres/T.B. Beds etc.

	1947	1962	1965	1968	1971	1974	1976	1978
1. T.B. Clinics of all denomination.	70	307	414	502		580	600	610
2. District T.B. Centres undertaking District T.B. Programme.	Nil	70	124	194		284	300	308
3. T.D. Centres (Tr. D.C's)	Nil	6	14		15	17	17	17
4. T.B. Beds	7000		35000		38000	41500		42500

(Figures are approximate)

Instead of becoming every body's responsibility it became nobody's responsibility.

II. The District Health Officers and Medical Officers and other district officers did not have TB control on their job chart as one of their important responsibility.

III. The programme was integrated with the general health services at the periphery but there were no tuberculosis workers at the Primary Health Centres, Taluk hospitals and in Municipal towns, with the result that tuberculosis was nobody's responsibility. The multipurpose general health service at the PHC did not take interest in tuberculosis work. Integration there fore failed and programme did not develop at the periphery.

IV. There was lack of supervision from the state health directorate. Even the regional organisations did not develop. Only two such centres were established; one in New Delhi and at the N.T.I. Bangalore. There was not even adequate budget for their travel. Wherever supervision was efficient, work developed very well.

V. Lack of understanding in the patient population and their families was responsible in defaulting in drug collection. Irregularity in taking anti-TB drugs is conducive to treatment failure and development of drug resistance and chronic invalidism in an infectious state.

VI. There should have been a continuing evaluating state and central organisation so that timely corrective action could be taken at the appropriate time. By now a national prevalence survey should have been undertaken to re-evaluate the problem and study the change

that has taken place in the prevalence of tuberculosis.

VII. In the early stage of BCG campaign, health education and publicity was integrated with the tuberculosis programme. Later it was merged with extension education at district and state level. This arrangement totally failed because no publicity for tuberculosis was really done at district level and at the periphery.

It is estimated that there are 9 million tuberculosis patients in the country of whom only 1.5 million are on treatment. Estimate of the number of patients on treatment has been made on the basis of drugs consumed every year in the country. Less than a quarter of this number is under treatment at the District TB centres and their peripheral sub-centres. Of these patients 35 to 40% take drugs regularly while the others either stop treatment after taking drugs for 3 to 4 months or take drugs irregularly for a little longer period. This is a very disquieting aspect of the National Tuberculosis Programme.

Planning for the Sixth Plan

General administrators often feel that tuberculosis too should be eradicated as has been done in the case of small-pox. It should be appreciated that tuberculosis is a different type of disease. A person infected with the tubercle bacillus may develop the disease many years later depending on constitutional, environmental factors etc. Enhancement of resistance against disease by improved standard of living plays an important role in prevention of disease. Population explosion, persistence of slums, ignorance, poor nutritional standards etc. do not allow

achievement of tuberculosis control unlike in the Western countries. Eradication has not been possible even in developed countries of the west. TB control will be a very slow process, do what you may, even though miraculous drugs are available today. Discipline in the people, and understanding of disease among the ignorant masses is even now so low that hardly 30% patients take regular treatment as long as advised, inspite of the fact that the drugs are available within easy reach even in a rural area.

It should be appreciated that India's tuberculosis programme is not much of a control programme but it is a minimum essential service to the people to prevent disease by BCG vaccination and provide minimal effective diagnostic and treatment service within our means. Our people have the right to receive at least this essential service. When this service is fully

developed it will form a sound basis for control of disease. As more effective drugs are applied the impact may be even better.

Inspite of these deficiencies, there is no doubt that there has been a change in the epidemiology of tuberculosis even within the last 15 or 20 years. Bone and joint tuberculosis is indeed less common and caseating lymph nodes are rarely seen. Tuberculosis is developing in the higher ages mostly over 35 years and gradually tuberculosis is becoming a disease of the elderly males. Tuberculous Meningitis and miliary tuberculosis are getting much less common. Tuberculin allergy is less frequently seen in small children and the age at which children get infected is now higher. These observations in themselves are a great hope since they indicate that we are slowly progressing towards the control of tuberculosis.

FIELD SERVICE TRAINING IN TUBERCULOSIS CONTROL

N.K. MENON And A.K. CHAKRABORTY

(From National Tuberculosis Institute, Bangalore)

Introduction

The main activities under the National Tuberculosis Programme are (i) diagnosis and treatment of the sputum positive tuberculosis patients through the existing and expanding peripheral general health institutions (PHI), (ii) obtaining an acceptable level of coverage with BCG vaccination of the susceptible population (0-19 years) and its continued maintenance, (iii) adequate maintenance of records and (iv) minimal but effective reporting of activities by the workers at all levels.

It is envisaged that through the efficient performance of the above activities it will be possible to achieve a systematic reduction in the problem of tuberculosis over a period of time. For this purpose, a large number of personnel need to be trained on specific job performances. The personnel involved in various activities at different level are given in Table 1. In the present paper, it is proposed to discuss, (i) the training needs of the above categories of staff in relation to the activities as given in Table 1, (ii) the training programmes currently in practice to meet the above needs, (iii) achievements made so far with respect to the training and (iv) changes envisaged in training contents and methodology to keep abreast with the structural and functional changes being made in health services.

Training needs and Methods

A. Peripheral (PHI level)

Since majority of tuberculosis patients in the community contact the PHIs for the first time, diagnosis and treatment of tuberculosis carried out from these institutions is the *sine qua non* of any tuberculosis programme. For efficient performance of these activities from the PHIs, the training for the PHI staff in carrying out anti-tuberculosis programme activity is a necessity. There are on an average 50 health institutions in each district. A Medical Officer, Laboratory Technician and Dispenser from each of these Institutions need to be trained to enable them to carry out the work as shown in Table 1. How to train this large army of health workers is the question. It is considered unnecessary to train specialised tuberculosis workers for these Institutions. The problem is sought to be solved by a method of imparting in-service on-the-job training of the existing staff at all the PHIs through the

team of district key personnel² of the District Tuberculosis Programme (DTP). The latter in their turn are trained at the National Tuberculosis Institute (NTI). The key personnel are required to make visits to these PHIs (implementation visit) and train the PHI staff for short durations in the standardised procedures of simplified tasks aimed at case detection and treatment. As for example, the Medical Officer of the PHI is briefed by the District Tuberculosis Officer (DTO) on selection of symptomatics, treatment regimens in DTP, examination of stained sputum slides, maintenance of records and reporting pertaining to anti-tuberculosis work at his centre. The Pharmacist or any other suitable person is trained by the Treatment Organiser (TO) of the District Tuberculosis Centre (DTC) on drug regimens, maintenance of cards, defaulter identification and retrieval system. The Laboratory Technician of the PHI is trained by the Laboratory Technician (LT) of the DTC about Ziehl-Neelsen staining methodology, examination of slides for AFB and recording and reporting. It has been recommended that the training should be of 4 to 7 days duration and the trained personnel are made to repeat the tasks in the live situation of the functioning PHI. This on-the-job training is expected to generate confidence of the staff in the methods and tools to be applied by them in their respective PHIs on day-to-day basis. Replacement training and re-training of the PHI personnel³ are essential features of training of such personnel which ensure the continued availability of trained staff at the PHIs making good the depletion of previously trained staff as a result of transfers, promotions, retirement, death etc.

B. Intermediate level (DTC level)

The principle functions of the DTC are delivery of health care to patients attending DTC either on their own or on referral and implementation and supervision of anti-tuberculous work at all the PHIs in the district (Table 1). An average district with 1.5 million population is equipped with one DTC where a Tuberculosis Control Team is posted.² This team is responsible for performing the key functions mentioned in Table 1 assisted by a number of non-key personnel. The key personnel form the pool of specialised expertise for the entire district and comprise of a Medical Officer (DTO), a Tuberculosis Health Visitor (TO), an X-ray Technician (XT), a Laboratory Technician (LT), a non-medical

Team Leader for BCG (BCG NMTL) and a Statistical Assistant (SA). This team of six key personnel receives training at the NTI for a period of 13 weeks and on their posting back to the respective places of duty perform the functions for which they are trained, the most important of which can be singled out as the extension of programme activities to the periphery (PHI). As already explained this extension is achieved by their visits to the PHIs, when they impart on-the-job training to the vast number of PHI workers. They also supervise the on-going programme and correct the deviations whenever detected, so that continuous training to the PHI workers is maintained.

Since case finding, treatment, training of PHI personnel and maintenance of records and reports are a series of interdependent multi-disciplinary tasks, groups of key personnel are trained as teams at the NTI which enables them to clearly understand the implications of each other's functions. They are trained at the NTI since it is the pivot of the National Tuberculosis Programme (NTP). Also, this is because a body of staff with knowledge and programme expertise is available ready at hand to start with, who could ensure the diffusion of knowledge on standardised techniques and methods developed by them over the years through a series of assiduous operational researches. The training for 13 weeks can be theoretically divided into 3 terms (Table 2). It is largely oriented to work experience for trainees with some didactic lectures to explain the rationale of different activities required to be performed under the DTP. Needless to say that it has a public health bias and the objective is to turn out workers who have to assume more of managerial roles in the programme. Continuous practice of their respective skills, role play, dummy training,² seminars and group discussions are liberally built in to give as much practical experience as possible. The trainees are formed into tuberculosis control teams and a team is given the experience of actual field planning, field implementation and supervision of anti-tuberculosis work at PHIs and in BCG vaccination.

Apart from continuous on-going assessment, each candidate is assessed initially, at mid-term and finally in order to have feed back as to their progress.

C. Top level (Programme supporters)

Since the DTO has to function within the ambit of the district health organisation, the administrative support and encouragement by the District Health Officer (DHO) is a must. Moreover, the DTP is now the direct responsibility of

the head of the district health organisation—*i.e.*, DHO or CMO, and the DTO is the specialised staff officer to assist him in management of DTP. Similarly, the State level TB Officer (STO) is responsible for planning, monitoring and supervision of the programme in the entire state. The Director of the State Tuberculosis Centre of the respective state is to assist the STO in monitoring and supervision of the programme in the entire state. These senior staff need to be made conversant with the programme dynamics so that they can play useful roles. The briefing of these categories of senior officers is achieved through a 10 working day seminar organised at the NTI. During the seminar, the general concept of the programme and problems coming in the way of its efficient functioning and the likely solutions are highlighted. Further, they are given clear idea as to the specific roles they are required to play in the programme. These seminars are open even to the teachers of tuberculosis, general medicine and public health to keep them informed on the current concepts and methods in tuberculosis control. They in turn could impart this knowledge to the undergraduates.

Training achievement and utilisation

So far, no statistics are available for the number of PHI personnel trained all over the country by the NTI trained Tuberculosis Control teams from 1961; but the number must be very large indeed! However, till 1977, a total of 3110 key programme workers have been trained at NTI.⁵ Their statewise and categorywise breakdown is given in Table 3. This large number of persons trained should have sufficed to man more than 384 districts in the country but for the loss of trained persons due to unavoidable contingencies. Whereas information on the proportion of trained persons utilised in the programme in the entire country is not available, it has been shown for the Southern Region, consisting of 4 states of Andhra Pradesh, Karnataka, Kerala and Tamil Nadu, that 68 % of the 66 DTCs had NTI trained DTOs. On the other hand, only 38 % of the total trained DTOs from the entire region had been actually utilised (44 of 115 trained). In other words, all the trained capacity is not utilised resulting in non-availability of trained man power in 32 % of the DTCs.³ Similar situation also exists for all other categories of key staff. It is vital for initial as well as continued training of PHI workers that properly trained key personnel are available constantly at the DTCs. This can no doubt be achieved by removing the existing mal-distribution.

Future of field service training in tuberculosis control

The concept and methodology of health care

Table 2

Time-table of subjects covered in District key personnel training course at NTI and method of communication

Conceptual terms in training	Period	Subjects covered	Method of communication
Initial term	0-4 wks.	<p><i>Medical Officer :</i></p> <ol style="list-style-type: none"> 1. Health set up in the country 2. Problem of tuberculosis, epidemiological and sociological orientation 3. Case finding methods and their applicability 4. Chemotherapy regimens and their suitability for programme 5. District Tuberculosis Programme in concept and outline 6. Use of diagnostic tools 7. Statistical methods <p><i>Paramedical Personnel :</i> Elementary knowledge on the above</p>	<p>Didactic lectures</p> <p>-do- and practicals</p> <p>Common lecture for all categories on subjects 1-6.</p>
Mid-term	4-10 wks.	<ol style="list-style-type: none"> 1. Planning & implementation⁴ of DTP 2. Programme supervision⁴ and monitoring 3. Programme management like maintenance of supplies, solution of specific problems in PHIs 4. Familiarisation with technical contents of job requirements by respective categories like sputum collection and staining for LT, X-raying & X-ray maintenance for XT, preparation of records and reports for SA, BCG, vaccination methods and organisation for NMTL, X-ray reading & sputum examination for MO etc. 	<p>Group training as Tuberculosis control teams :</p> <ol style="list-style-type: none"> (a) Practical training by dummy, field visits to DTCs & PHIs (b) Preparation of field visit reports and problem faced by the team (c) Interpretation of reports. <p>Practical sessions</p>
Final term	11-13 wks	Consolidation and rationalisation of aspects already learnt	Group discussions, seminars, public speeches on allotted subjects, guest lecture on subjects of topical interest.

delivery through village level 'community health workers' supervised by "multi purpose health workers"⁶ is being evolved today in the country to render primary health care facilities more acceptable and pragmatic for the vast rural masses. It is possible that when they go into operation, this scheme will make it incumbent on the primary level workers to collect sputa

from symptomatics while on their routine rounds in the villages under their care and to enquire about drug intake by the patients already on treatment. This will mean that these workers will need necessary skill in symptom questioning and sputum collection and smear preparation as well as in drug defaulter retrieval. For this purpose, training of the above staff should consist

Table 3*

Training achievements and utilisalon

Categories of staff	States (Total No. 22)	Union Territories (Total No. 9)	Miscellaneous**	Total
MO	535	19	7	561
TO	648	20	5	673
XT	466	11	7	484
LT	531	13	9	553
SA	465	13	8	486
BCGTL	343	9	1	353-

* Excludes 120 WHO Fellows of various categories.

** Coal mine labour welfare board, Railways, Bombay corporation, Northern regional tuberculosis centre etc.

of demonstration of simple and standardised techniques with the objective to prepare them to perform the simplified tasks. Though such training will not demand high degree of specialised expertise but the size of the trainees calculated as 5.8 lakhs proposed to be trained in about 2 years⁶ could no doubt pose problems. However, while the community health workers are training in other job responsibilities at their respective PHIs, they may also be briefed on the tasks related to tuberculosis programme. The trainers at PHIs *i.e.*, the Medical Officers of the PHCs in their turn are proposed to be trained at the rural family planning training centre (RFPTC) on the contents and methods of anti-tuberculosis training which they are required to impart to the workers at the peripheral levels. For the guidance of the RFPTCs lesson plans have already been prepared and are being tried out at some of the RFPTCs.

For BCG vaccination also, a new method of giving vaccination through the PHIs utilising the existing staff of general health services is being developed. When implemented this will replace the mass BCG vaccination programme in vogue at present. To carry out this activity, the general health workers require a short training which needs to be organised. It is possible that the services of the existing BCG technicians and the

BCG NTI could be utilised to provide fining either at the district headquarters or at each of the PHCs. Simplified manuals and lesson plans have also been prepared to impart training on BCG vaccination to the general health workers.

Conclusion

In the NTP case finding and treatment of patients of tuberculosis carried out through the PHIs is considered as the basic features of the programme. However, available reports on the efficiency of performance point out that considerable improvement is required.⁷ While most or many of the reasons for this shortfall in performance of PHIs could be traced to administrative and managerial deficiencies, others could be related to the factors of knowledge, attitude and skill² of the PHI workers. The need of training of the PHI workers as a method of bringing about changes in the above fields cannot be over emphasised. It is a painstaking and continuous process and it was envisaged that the DTP key personnel would be the instrument of dissemination of training to the large body of PHI workers. Whereas, the DTP key personnel are by and large functioning effectively in their technical responsibilities, their efficiency in training and re-training the PHI workers leaves much to be desired.⁷ If DTO has to succeed, the

key personnel have to play a more positive role as trainers of field workers and organisers of the programme in the district.

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INTERPRETATION OF TUBERCULIN TEST

S.P. PAMRA*

Body's reaction to primary infection with *Mycobacterium tuberculosis* is different from the reaction to subsequent infections. Following primary infection, the local reaction becomes manifest after about a week or so, is mild and develops slowly. The reaction to subsequent infections, on the other hand, reaches its maximum in 48 to 72 hours and is more severe. This difference in the body's reaction is known as Koch's phenomenon and is due to the fact that primary infection confers allergy (and also immunity). Allergy, though it can be conferred only by living micro-organisms, can be elicited even by dead micro-organisms. Thus when tuberculin (which is merely dead tubercle bacilli) is injected and a reaction appears at the site of injection after 2/3 days, it should indicate that the individual has already been infected. In other words, Koch's phenomenon is the basis of tuberculin reaction.

It also follows from above that if an individual is not infected with *Mycobacterium tuberculosis*, tuberculin should not give any reaction whatsoever. In fact, it is not always so. Many persons do show some reaction to tuberculin even if they are not infected. The original tuberculin prepared by Koch, known as 'Old Tuberculin' (O.T.) contained not only dead bacilli but constituents of the medium also in which the bacilli were grown. Thus some or all the reaction to O.T. could be due to hypersensitivity to the constituents of the medium apart from hypersensitivity to bacillary products. To remove this short coming, use of O.T. is virtually given up and instead, P.P.D. is used which is free from the constituents of the medium and contains only bacillary products.

This, however, has also not solved the problem altogether because even P.P.D. is not a pure and a specific antigen. There are a large number of atypical mycobacteria which are closely related, antigenically, to the *Mycobacterium tuberculosis*. If an individual is infected with one of these atypical mycobacteria, tuberculin will give some cross reaction because of the partial antigenic similarity. This low grade hypersensitivity—termed non-specific sensitivity—thus complicates the value of tuberculin test in determining whether an individual is infected with *Mycobacterium tuberculosis* or not.

Attempts are being made to isolate that fraction from the P.P.D. which is specific for *Mycobacterium tuberculosis* and is not present in any

other mycobacterium. Once that fraction becomes available, tuberculin test will become highly sensitive and specific. If the individual is not infected with *Mycobacterium tuberculosis*, there will be absolutely no reaction to this fraction and if the individual is infected, there will be some reaction, size of the reaction (which depends on the allergy producing capacity of the individual) being of no consequence to the interpretation.

Tuberculin reaction consists of erythema and induration. Since erythema is sometimes difficult to assess, it is ignored in the interpretation of tuberculin test and induration alone is taken into consideration. It is customary to consider an individual with an induration of 10 or 12 mm to 1 unit of PPD RT 23 as positive, indicating infection with tubercle bacillus. This definition is to a certain extent arbitrary, since the size of tuberculin reaction, even amongst the infected individuals depends, to a very large extent, on the allergy producing capacity of the individual. We see every day that individuals who are definitely infected e.g. sputum positive patients of pulmonary tuberculosis, give various grades of tuberculin reaction. Thus in an individual who shows a small reaction to tuberculin, say 5 to 9 mm induration, two explanations are possible. Firstly, he may have been infected with an atypical micro-organism and thus gives a small cross reaction to tuberculin. Secondly, the individual may be infected with *Mycobacterium tuberculosis* but being a low allergy producing individual, his reaction is low. It is one of the most difficult problems to decide which of the two explanations apply and whatever it is, it is liable to introduce a certain degree of error in interpretation of the tuberculin test. If reactions below 10 mm do not rule out definitely the possibility of infection with *Mycobacterium tuberculosis*, what then is the sanction behind the reaction of 10 or 12 mm to be considered as the point of demarcation between infected and non-infected individuals?

When tuberculin reactions in a large number of individuals in the younger age groups (among whom there are many uninfected individuals as against older age groups where practically every one is infected in high prevalence countries like ours) are charted in the form of a curve, the curve is found to be bi-modal. The left hand side of the curve and the first mode in such cases obviously represent non-specific sensitivity and remaining part of the curve and the second mode represent specific sensitivity to tuberculin. The anti-mode in such curves is therefore taken as representing

* Director, New Delhi Tuberculosis Centre, New Delhi.

the point of demarcation between infected and uninfected individuals. This anti-mode usually lies at 10 or 12 mm reaction. Such a definition obviously does not exclude the probability of some infected individuals being considered uninfected by definition and vice versa. This is not likely to cause any difficulty in epidemiological studies where the problem is how many are infected and not who is infected and who is not infected. Thus, whatever size of reaction is taken as the point of demarcation, the margin of error in epidemiological estimations will be almost similar and will not interfere with or vitiate any conclusions. In clinical practice, on the other hand, it is important to know whether an individual is infected or not and this the tuberculin test in the present state of our knowledge and technology is incapable of telling, more particularly if the reaction is less than 10 or 12 mm.

Some times an attempt is made to resolve this difficulty by testing the individual with a higher strength of tuberculin. This however does not solve the difficulty in any way. The size of reaction, apart from other things referred to already depends on the quantity of antigen that is injected. In other words, increasing the dose but keeping the threshold of positivity as unchanged is obviously wrong. Furthermore, if the specific reaction to tuberculin is increased by increasing the dose, so also will the cross reaction due to non-specific sensitivity be increased. Therefore, testing with a higher strength of tuberculin does not solve any problem.

Some people use BCG as tuberculin for diagnostic purposes. This is even more unscientific and inaccurate. BCG, antigenically, is at least 15 times stronger than 1 TU PPD. More over BCG is a bovine bacillus and almost all infections in our country are caused by *Mycobacterium tuberculosis* which is human in type. Therefore, the reaction to BCG cannot be compared with the reaction to 1 TU PPD nor is it in any way more specific or more helpful in differentiating the infected from the uninfected.

Some times dual testing with PPD derived from *Mycobacterium tuberculosis* and another PPD derived from atypical mycobacteria is advocated. If the size of reaction with the latter is more than with the former and the former is below 10 mm, it is usually taken as an evidence of infection with the atypical mycobacterium and vice versa. This again is not absolutely accurate. The number of atypical mycobacteria which could cause infection is very large indeed. It is virtually impossible to use the tuberculin prepared from all types of mycobacteria for testing, and if PPD derived from one particular atypical strain alone is used, it may not mean much if the infection is

with another atypical mycobacterium. PPD-B (derived from Battey bacillus) is mostly used for dual testing because Battey bacillus is the most common atypical infecting agent in USA where dual testing was evolved. The situation may not be exactly the same in our country. And if the individual is infected with an atypical mycobacterium other than the Battey bacillus, dual testing with both the PPDs *i.e.* one derived from the human type and the other from Battey bacillus may result in a weak tuberculin reaction *i.e.* less than 10 mm.

Does this mean that the tuberculin test is useless at present and unless the fraction of the PPD specific to *Mycobacterium tuberculosis* is identified and isolated, tuberculin test will continue to be equivocal in interpretation? It is not entirely so. It has already been mentioned that for epidemiological purposes if every one with a reaction of 10 or 12 mm (or even higher) to 1 TU PPD is considered as infected it serves the purpose fairly adequately. In clinical practice, it appears that if the reaction is more than 10 or 12 mm the individual can be considered as definitely infected. If the reaction is less than 5 mm, the individual may, for practical purposes, be considered as uninfected though the remote possibility of infection having taken place cannot be ruled out by any means. In the case of those showing a reaction of 5 to 9 mm, it would appear to be expedient to consider them as infected if differential diagnosis is the objective. Since tuberculin test is an indicator only of tuberculous infection and not of disease, not much harm is done by considering such persons with intermediate reaction as infected and subjecting them to other examinations for differential diagnosis. By doing so, a case will not be wrongly excluded from the differential diagnosis of tuberculosis and even if such a reaction is wrongly considered as infected, further diagnostic criteria will help to eliminate the presence of disease. If BCG vaccination is the objective, then also the equivocalness of interpretation of tuberculin reaction will not matter since pre-vaccination test is not done as a matter of routine and secondly even if an infected individual is wrongly considered as uninfected owing to the deficiencies of interpretation of tuberculin test, and vaccinated, no harm will follow the vaccination.

Lastly, it may also be pointed out that tuberculin test only helps to determine whether an individual is infected or not. There is no degree of reaction which may indicate the possibility of active disease. All that can be said is that a strong reaction is indicative of recent infection and/or high allergy producing capacity of the individual. Such high reactors, however, are more prone to develop disease in the course of time.

HEALTH HAZARDS OF AIR POLLUTION AND SMOKING

R. VISWANATHAN

Introduction

Even, though the impact of environment on man's health has been known for a long time, it is only during recent years that the importance of environmental factors like Air Pollution in the development of disease has been given increasing recognition. Increasing urbanisation concomittant with explosive population growth, particularly in our country and rapid industrialisation, have resulted in increased number and variety of pollutants thrown into the air that we breathe.

Sources of Pollution

There are various factors responsible for air pollution. The type of pollution will depend upon the sources from which they arise. There is the natural air pollution which includes wind blown dust, pollens and other aeroallergens, micro organisms, fog, ozone from lightning, negative air pollution like reduction in oxygen content of air and increase in carbondioxide of the atmosphere.

The Central Public Health Engineering Research Institute, Nagpur, conducted pilot air pollution surveys in 4 large cities in India. The results are given in the following table :-

Table 1

	SO ₂ ppm.	NO ₂ ppm.	O ₃ ppm.	Particles ug/m
Bombay	.014	.012	.017	238
Delhi	.03	.012	.013	700
Calcutta	.028	.017	.018	527
Kanpur	.03	.095	.025	488

Other sources of pollution arise from fuel combustion, fly ash, smoke, transportation, nuclear explosion, photochemical smog and disposal of solid waste. The main types of pollutants are particulates, sulphurdioxide, nitrogen-dioxide, carbon monoxide, oxidents, hydro-carbons and radiations.

I would like in the first place to deal with the

possible chances in the constituents of air that we breathe like oxygen and carbondioxide.

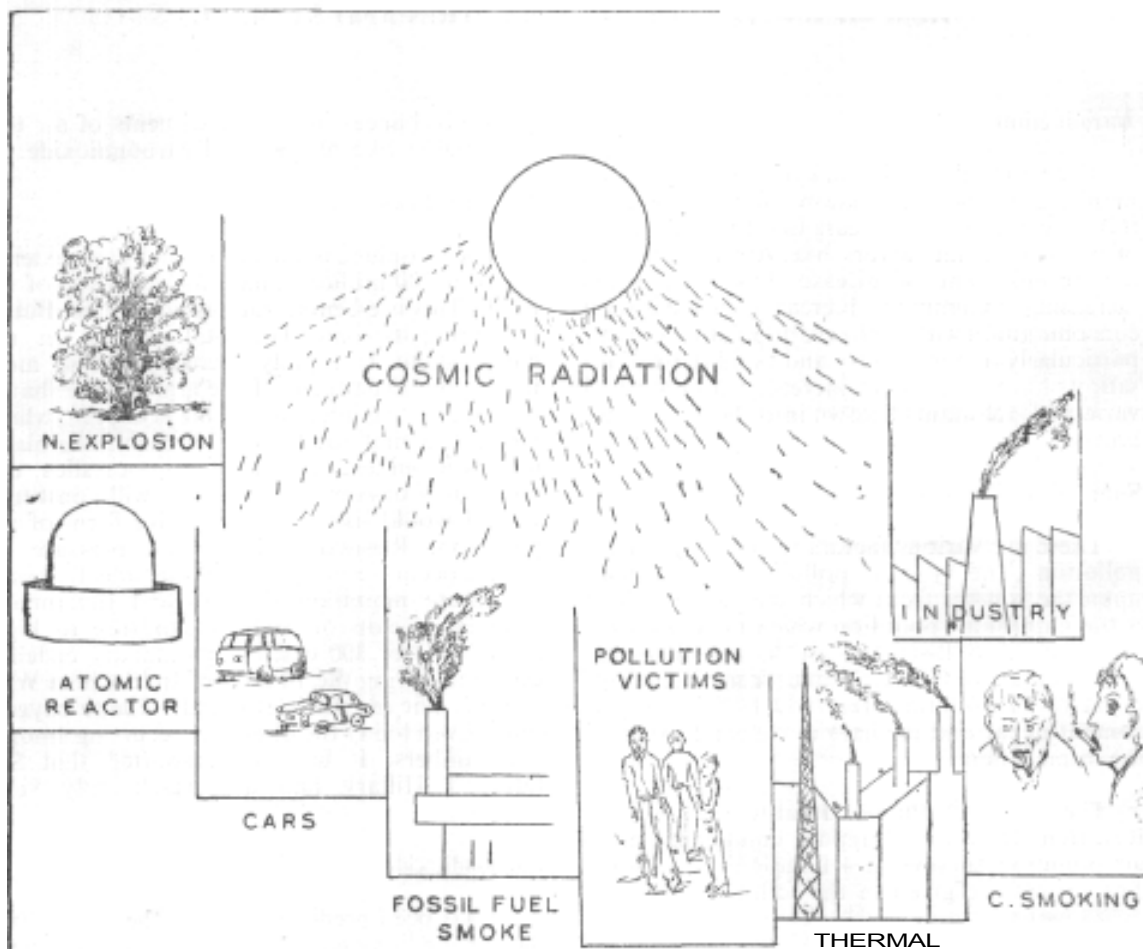
Oxygen Lack

The biosphere is a ten mile thick layer extending over 200 million square miles surface of the earth. This is essential for sustaining life. But it is nearing its limit. The total demand on the environment is rapidly increasing. The most important constituent of the atmosphere that is essential for maintenance of life is oxygen, which means the vital air. If for any reason like black dumps in mines, in hulls of ships or silos, the amount of oxygen is reduced, it will constitute what I would like to call negative form of air pollution. Reduction in partial pressure of oxygen occurs naturally at high altitude. Diseases like acute mountain sickness and pulmonary oedema develop on sudden exposure to high altitude. Over 300 cases of pulmonary oedema occurred during the 14 days of Indo-China War of 1962. The disease is essentially due to oxygen want. Even today the disease is occurring among hill climbers. It has been reported that Sir Edmund Hillary had an attack only very recently.

Carbondioxide

It has been predicted that by the year 2000, the carbondioxide of the air will have increased by 14 to 30%. The balance between oxygen and carbondioxide in the earth's atmosphere is extremely important to all life in the planet. Animals take oxygen and release carbondioxide and water or become a part of lithisphere as a sediment or an organic deposit such as Peat.

If the absorption of carbondioxide in photosynthesis by plants is reduced by extensive deforestation, there is likelihood of the levels of carbondioxide increasing in the atmosphere. Fortunately, nature has provided measures for the oxygen/carbondioxide balance. If this is disturbed as seems to be the case, carbondioxide is likely to increase certain regions and in certain places. The inhalation of high concentration of carbondioxide has deleterious effect on the body. Industrial and biological activities do contribute to variation in atmospheric carbondioxide. Our poor people, the *jhuggi* dwellers in the urban areas and the landless labourers in the rural areas—often sleep on the ground—sometimes even on waterlogged soil beneath which the carbondioxide content may vary from 3.9 to 9.1 per cent in the air spaces. During winter,



North Indian villagers sleep at night—6 to 10 persons in a room with doors and windows closed—with *angithi* to keep themselves warm, and thus are likely to inhale high concentration of carbondioxide and other gases. Patients suffering from chronic obstructive lung diseases are likely to have raised carbondioxide tension in their blood which may lead to serious complications like carbondioxide narcosis. One of the effects observed during our studies is the rise in pulmonary artery pressure.

Entry of pollutants through inhalation

Disease from other forms of extrinsic pollutants can be produced either by inhalation or ingestion or skin contact. Since we inhale 5 litres of air per minute and since 40 square meters of alveolar surface are constantly being exposed to the pollutants of the air that we breathe, one is justified in considering that the lung is perhaps the commonest mode of entry of pollutants into

the human body. There is a certain relationship between the dose of the inhaled pollutant and the kind and degree of response produced in the tissues. The quantitative understanding of the dose response relationship requires that the magnitude of the dose be expressed in terms of the effective dose rate at the critical site within the body. It is not enough if one knows the atmospheric concentration and the minute volume of the air that is inhaled. The product of these two can only give the rate of delivery of pollutants into the respiratory system. It does not indicate, however, how much of these pollutants so inhaled reach the critical site. Much of them is cleaned away by certain physico-physiological processes, and as such, is not available for the production of disease. In order to establish a quantitative dose response relationship at the critical site one must estimate how much of inhaled pollutants is initially deposited and at what sites in the respiratory system, how rapidly and in what degree, the deposited particles are

cleared from the respiratory tract and lungs and finally what fraction of the deposited material reaches the critical site within the lungs or other parts of the body to cause disease.

Deposition

Since the particulate air pollutants are primarily responsible for development of different forms of pneumoconosis, I would like to summarise the factors governing the deposition of air-borne particles in the different parts of the respiratory system and their clearance. Passage of atmospheric particles into the lung parenchyma are governed by certain, physiological principles. Forces of inertia cause deposition within the nasopharyngeal chambers and at such points of branching of the airways wherever the direction of flow changes. Effectiveness of inertial deposition increased with air velocity and consequently decreases with depth in the respiratory system. In the terminal airways, the particles are deposited by gravity settlement based on Stoke's Law. Such settlement of particles will depend upon the settling velocity and the time available for settlement. Microscopic particles suspended in air are subject to Brownian action due to bombardment by gas molecules. 100% of particles above 5 microns get deposited somewhere or the other of the air passages. At half micron it is brought to the order of 25 %. Percentage deposition also increases as particle size decreases below half micron. For particles smaller than 0.1 micron, the percentage deposited out of a total respired air approaches in value the fraction of tidal volume which reaches the pulmonary air space. Hence the percentage deposition of such particles approaches 100%.

Regional deposition

Regional deposition is of importance because the risk of disease production depends upon the site at which a particular type of pollutant is deposited. Particles larger than 10 microns are deposited in the nasal passages. Most of the particles upto 2 microns are deposited in different parts of air passages. The percentage of inhaled particles which penetrate into and deposited in the alveoli have a value between 1 and 2 microns.

Clearance

The particles deposited in the respiratory passages upto the respiratory bronchioles are removed by the mucociliary mechanism. Most of the particles that settle on the alveolar surface are transported to the mucous blanket of the trachio-bronchial lining, membrane.

Mode of passage into lung tissue :

From the point of view of disease develop-

ment the passage of particles into the lung tissue is of fundamental importance. The particles may either directly pass through the air sac wall or may be engulfed by phagocytic cells which pass into the lung tissue.

It will be perhaps worthwhile considering the nature of tissue reaction produced by different pollutants in the first instance and later consider some specific diseases.

Tissue reaction to pollutants sulphurdioxide:

Sulphurdioxide is one of the commonest gaseous pollutant in the atmosphere. It is established that in a city like New York, the monthly mean concentration may go upto 0.3 ppm and 24 hour maximum 1.2 ppm. In the London smog of December 1952, a two day average value of 1.34 ppm. was recorded. Sulphurdioxide even at 1 ppm. can increase airway resistance. There is, however, a constitutional factor in regard to sulphurdioxide irritability of the bronchi. When there is a sudden increase in sulphurdioxide content of the air, as was found in the London smog episode of 1952, people who are suffering from chronic bronchitis and emphysema can get sudden aggravation of symptoms leading even to death. During the London episode over 3500 excess deaths during one week occurred. People of the old age group suffering from chronic cardio-respiratory diseases were the ones mostly affected. There were two other acute episodes of a similar nature which have occurred, namely the Meuse River Valley episode in Belgium in 1930 and Donora episode in U.S.A. in 1948.

Nitrogen-dioxide

The concentration of nitrogendioxide in urban air is usually less than 1.1 ppm. Human volunteers exposed to even 3 ppm. have not shown any definite clinical symptoms. Continuous exposure to increased nitrogendioxide concentration in the atmosphere make them more susceptible to infection.

Carbon-monoxide

As a result of increase of automobiles on roads, concentration of carbonmonoxide of 15 ppm. and sometime as much as 115 ppm. have been reported. In road tunnels, values may go upto 500 ppm. Because of its great affinity to haemoglobin, carboxy haemoglobin is produced with corresponding less amount of oxygen being carried by the haemoglobin circulating in the blood. The result is defective oxygenation of tissues.

Ozone

The toxicity of ozone has become an increasingly important problem with increasing number of supersonic flights and space exploration, since a layer of ozone is present in significant quantities in the troposphere. Ozone is also a dominant constituent of photo-chemical smog. Los Angeles smogs have levels upto 1 ppm. of ozone. Even 0.5 ppm. produces in volunteers substernal soneness cough and laryngitis. We exposed batches of animals for varying periods to 1 ppm. ozone for 3 hours daily. Changes like congestion, mild to moderate interstitial oedema and alveolar oedema were observed. In some animals there were desquamation of the lining cells of the trachia.

Table 2

2 PPM O ₃ on 6 Mice Lungs 3 hrs. daily for 3 months	
Congestion	3
Alveolar Oedema	2
Interstitial Oedema	4
Trachial Lining Desquamation	2

We also made 6 bronchitics and 6 normal men inhale 1 ppm. ozone for 10 minutes. All of them showed changes in their lung function after breathing ozone. They also experienced certain symptoms like substernal soreness, dryness, cough etc.

Table 3 and 4
O₃ on Ventilation in 6 Normals

	Before X ±S.D.	After X ±S.D.	Sig.
VC	4190 ±386	3948 ±439	HS
FEV ₁	3809 ±337	3453 ±410	HS
<i>O₃ on Ventilation in 6 Bronchitics</i>			
	Before X ±S.D.	After X ±S.D.	Sig.
VC	3057 ±378	2727 ±398	HS
FEV ₁	2185 ±372	1780 ±386	HS

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Particulate matter in the air has many adverse effects on one's health. Hence its reduction is of prime importance. The particles suspended in urban atmosphere consist of silica, metallic oxides and salts, carbon particles and oily or tarry droplets. Fly ash is one of the common particulate matter formed as a result of combustion of fuel. As mentioned earlier about 75 tonnes of fly ash is thrown into the atmosphere of Delhi from one thermal station alone.

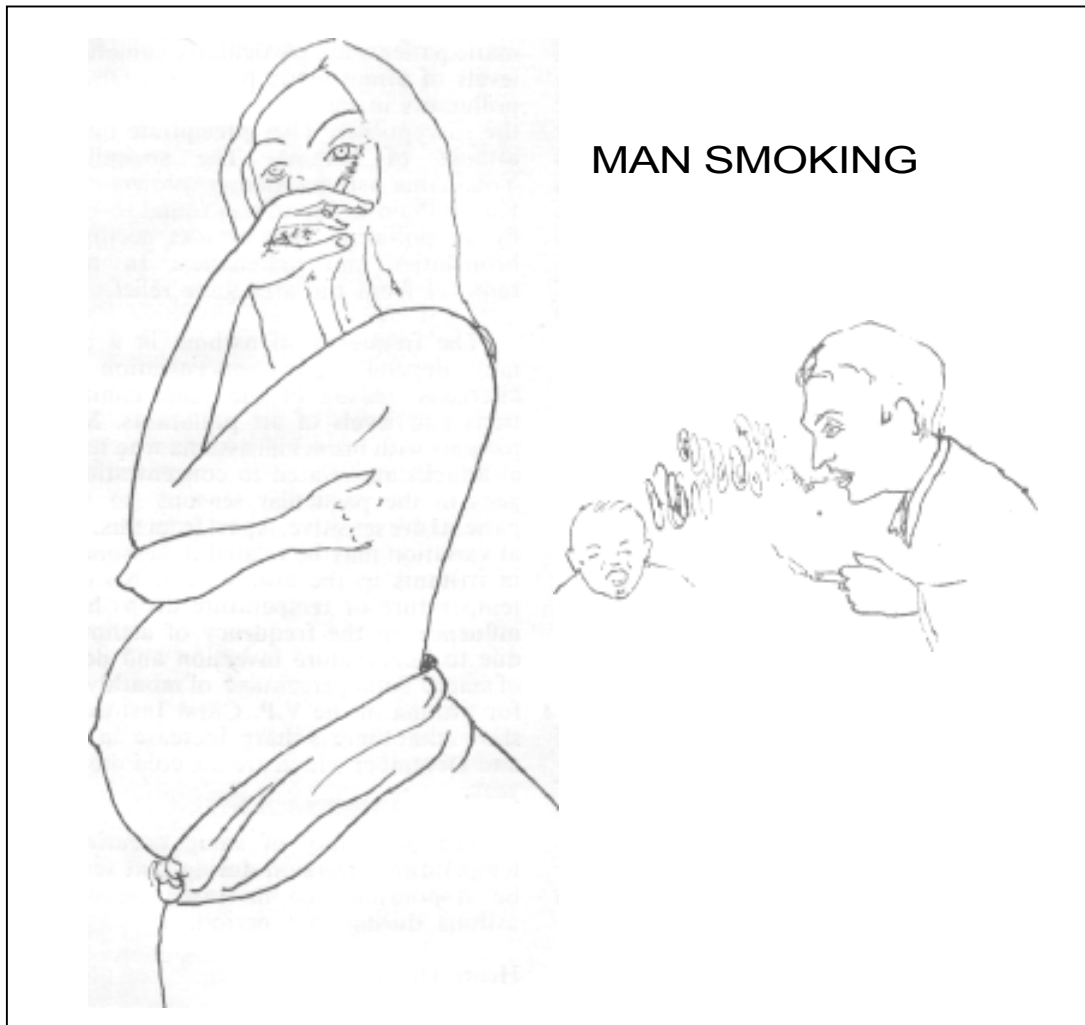
In India, particularly in the rural areas, not only coal but also firewood and cow dung cakes are used as fuel. Hence air in many of the rural dwellings get filled with smoke and fly ash. Irritation of the air passages as a result of inhalation of smoke and fly ash, can lead to chronic bronchitis and emphysema.

Radioactive pollution

Cosmic rays and their products constitute major part of the radiation dose to bone cells and gonads. The major cosmic ray particles in the earth's atmosphere are nucleons, mesons, leptons and photons. The amount of ionising component of cosmic rays on the earth's surface is dependent on latitude and altitude. The average dose for a three hour transatlantic flight would be six millirads. Dose rates for 24 hours on space flights have been measured from 12 millirads at 30 k.m., through 100 millirads at 700 k.m. At 1500 k.m. it is about 2000 millirads.

The main cause of radio activity in the air is radio active materials from explosions of nuclear devices. Other sources are radioactive mining, atomic reactors and fuel reprocessing plants. Of the non-ionising radiation there are man-made as well as naturally occurring pollutants. U.P. lamps, lasers and masers, radio broadcasting and radio installations, microwave communication systems, particles accelerators and nuclear reactors constitute man made pollutants. Natural radiation may either be from outer space or from the earth or from the earth's atmosphere. With increasing use of nuclear reactors and possible increase in nuclear explosions, radiation is becoming a serious hazard to human health. A number of people who survived the atomic explosions at Hiroshima, were nontheless exposed to radiation as a result of which they developed blood diserasis like leukemia.

A discussion on environmental pollution will not be complete without considering the effect of tobacco smoke. Smoking is not only a hazard to the smoker but also to those who inhale the air containing exhaled smoke. It has been shown that young children of parents who smoke are more liable to infection than others.



They often suffer from attacks of bronchitis and bronchiolitic. It has been reported that infants born to women who smoke during pregnancy are more liable to get respiratory infection than other infants. Two of the important conditions which can definitely be attributed to smoking are chronic bronchitis and lung cancer. Numerous reports have been published containing incontrovertible data showing that there is a casual relationship between cigarette smoking and the development of lung cancer as well as chronic bronchitis.

I would like now to consider certain specific diseases that are attributable to air pollution.

Chronic bronchitis and emphysema

There have been several studies both abroad

as well as in India which definitely go to show that cigarette smoking has causal relationship to the prevalence of chronic bronchitis and emphysema. Our studies done in Delhi as well as in Patna go to show that there is significant increase in the number of chronic bronchitics amongst smokers than amongst non-smokers.

A pilot survey conducted among the University employees and families showed significantly more number of cases of chronic bronchitis amongst smokers than nonsmokers. Analysis of data of patients admitted to the Chest Institute suffering from pulmonary heart disease showed also that 71.6 % of them were smokers and the remaining only were non-smokers.

However, how much of the increase in chro-

nic lung diseases may be related to environmental pollution is difficult to determine. Epidemiological data have no doubt helped in assessing air pollution effects. All investigators have agreed about the direct correlation of excess urban death rate from chronic lung disease to air pollution. Recent comprehensive review by Lave and Seakin by multiple regression analysis of large number of epidemiological statistics has shown that the significant variable for all fatal cases of bronchitis was air pollution.

The study conducted by the College of General Practitioners and reported by Fletcher show very clearly that the disabling type of chronic bronchitis is more in larger towns than in the rural areas, whereas the prevalence of simple chronic bronchitis was practically the same in all the two areas.

There are, however, conflicting reports regarding chronic bronchitis prevalence and air pollution. Studies on adult population have been vitiated by the two co-existing factors, namely cigarette smoking and air pollution. Reid's studies on children before the smoking age have, however, shown that the higher the local level of air pollution the greater the incidence of chronic bronchitis.

A few years back we conducted an experimental study to observe the long term effect of cow dung smoke on experimental animals. We were able to produce emphysematous changes in the lungs in majority of the animals exposed for 6 months. Hence household air pollution by fuel smoke can produce respiratory problems.

Lung cancer

In a series of 333 cases of lung cancer in males, about whom we collected data from different hospitals during the last few years, 292, i.e. 89% were smokers. The evidence, however, linking air pollution to lung cancer is not very conclusive. Mills, however, found chronologic parallelism between the increase of automobiles and the increase in prevalence of cancer of lung. Kotin et al found carcinogens like pyrene, 3:4 Benapyrene and other hydrocarbons in the exhaust of petrol engines. Such carcinogens, however, contained in the exhaust fumes of automobiles get diluted to such an extent that their effectiveness in producing cancer is negligible.

Asthma

Bronchial asthma cannot be considered as being caused by non-allergenic air pollutants. It has, however, been observed that asthma is

more common in the winter months. Large and episodic increases in asthma frequency have been observed during autumn in New Orleans. Asthmatic patients are particularly vulnerable to high levels of atmospheric pollution. Obviously, the pollutants in the atmosphere act as irritants to the susceptibles. They precipitate but not cause attacks of asthma. The so called Tokyo-Yokohama asthma among soldiers stationed at Kanto Plain in Japan was found to be triggered by air pollution. The attacks occurred among bronchitics and asthmatics. In most cases removal from the area gave relief.

The frequency of asthma in a community may depend upon concentration of aero-allergens, season of the year, climatic conditions and levels of air pollutants. Majority of patients with bronchial asthma who have seasonal attacks are related to concentration of allergens in the particular seasons to which the patients are sensitive. Apart from this, the seasonal variation may be related to seasonal increase, in irritants in the atmosphere. No doubt cold temperature or temperature drops have strong influence on the frequency of asthma possibly due to temperature inversion and development of smog. Total percentage of monthly admission for asthma in the V.P. Chest Institute for 1975 shows that there is sharp increase in November and December which are the cold months of the year.

The possibility of smog occurring due to temperature inversion during that season might be responsible for increased admissions for asthma during that period.

Heart Disease

Astrep et al have demonstrated the atherosclerosis enhancing effect of carbon monoxide in cholesterol fed rabbits. Cigarette smokers have increasing levels of carboxy haemoglobin in the blood. Doyle et al have shown significant increased risk of sudden death as shown by Kennel. Another air pollutant related to heart disease is a Cadmium which is a naturally occurring mineral. Small amount of Cadmium is found in the atmosphere. A positive correlation has been reported between heart disease and Cadmium in the air by Carrol.

Skin and eye

One would naturally expect skin involvement from air pollutants. No acceptable effects, however, have been reported, though occupational dermatitis is not uncommon.

Eye irritation has been reported from several

cities like Los Angeles and even from Calcutta. Driving once on the roads of Los Angeles even on a day without any visible smog, long before I knew anything about air pollution, I felt irritation in the eyes, which started watering profusely. The doctor friend driving the car assured me that it was only due to exhaust fumes and that all newcomers to the city feel this irritation more than the residents. After I got interested in air pollution in Delhi, and after the Indraprastha Power House started functioning, my ophthalmic friends reported a number of cases of eye trouble from fly ash.

Mode of disease development

I would like to make some generalisations regarding causation, development and prevention of respiratory diseases due to air pollution:

(1) A large number of pollutants that are inhaled can participate in the causation and even more significantly in the aggravation of respiratory diseases.

(2) The pollutants rarely act singly. Automobile exhaust, oxidants, polycyclic hydrocarbons, toxic gases, burning of domestic fuel and smoking, act often jointly with disease causing factors, both extrinsic as well as intrinsic.

(3) While certain toxic agents can produce dramatic effects in high doses, repeated exposures to low concentration act imperceptibly and produce considerable damage in the lungs before the effect is discovered. While the acute toxic manifestations may be due to single agent, chronic conditions like chronic bronchitis, emphysema, fibrosis, intrinsic alveolitis and cancer develop as a result of the action of a number of agents. Only occasionally that a well defined specific chronic disease can be linked to one specific agent, like for instance, silicosis.

The time lag between commencement of exposure and development of frank disease increases the difficulty in incriminating specific agents.

In chronic lung responses like fibrosis, tissue destruction etc. recovery may not be expected even after exposure is stopped. Functional recovery can no doubt occur provided there is sufficient amount of residual pulmonary efficiency.

Prevention

It is the responsibility of the physician to assess the reduction in respiratory functions due

to chronic respiratory diseases. He is no doubt, concerned with alleviation of symptoms and conservation of residual function. Since the probability of cure in chronic disease due to air pollutants is very little, it has become incumbent on the part of the physician to impress upon the authorities to restrict emission of pollutants in the air. For this purpose, legislation is essential. Half-hearted legislative measures are worse than useless. Take for instance the case of cigarette smoking. After many years of representation by learned bodies like the Indian Association for Chest Diseases, Tuberculosis Association of India, etc. a half-hearted legislation has been adopted. Unfortunately, the type of legislation adopted even in western countries do not prevent people from smoking. The only way by which this can be effected is through the drastic measure of prohibiting smoking and/or prohibiting the manufacture of cigarette, beedis etc. That others also are thinking on the same lines is clear from the following statement by Prof. Green :

“A future medical historian may describe as the health conundrum of the twentieth century the phenomenon of an industry permitted to distribute so toxic a product of full knowledge by the public health agencies of the enormous, social and economic costs to the culture.”

This may no doubt be considered as the desperate outpourings of an unrealistic idealist. But then do we not prohibit the indiscriminate manufacture of a number of habit forming drugs. Even if some of them are made, the authorities have introduced the justifiable licensing system. If there are any administrative or other obstacles standing in the way of prohibiting the manufacture, there is no reason why tobacco smoking should not be included in the Government's prohibition programme. Alcohol is no doubt a socio-economic evil and should be prohibited. Except when it is consumed in excess, it does not generally produce serious illness. Smoking on the other hand is definitely a serious health hazard. There is every justification, therefore, for inclusion of tobacco smoking in the prohibition programme of the Government.

Polluting emanations from the industries can no doubt be minimised by compelling them to adopt technological measures for preventing pollutants entering the atmosphere from the factories. This has been done through the Clean Air Act in Great Britain. The time is come for adopting similar legislation in this country also.

Pollutants from automobiles exhaust, fumes are contributing their mite towards the pollution of the air that we breath in ever increasing measure, particularly in the larger cities of India.

My critics may suggest that I should advocate prohibition of automobiles and that we should go back to the bullock cart age. Just as we cannot do away with industries, we have come to a stage when we cannot do away with automobiles. In countries like the United States of America, the automobile engineers are desperately trying

to evolve methods by which the exhaust fumes can be made to undergo complete combustion and let out only innocuous gases. As and when it is perfected, the automobiles industries in India must be compelled to adopt such technological measures in the cars that will be produced in the future.

PROFILE OF CHRONIC NONSPECIFIC BRONCHITIS AS SEEN IN A FIELD SURVEY IN DELHI

DR. (Miss) T.G. RADHA*

(From Vallabhbhai Patel Chest Institute, University of Delhi, Delhi)

Introduction

Almost 30 % of the patients attending the out-patients clinic of V.P. Chest Institute, Delhi, which deals with non-tuberculous chest diseases, are found to suffer from chronic bronchitis. A field survey was therefore carried out to study the magnitude of this problem in the general population. The epidemiological data have already been reported (T.G. Radha, C.K. Gupta, Ajit Singh and N. Mathur M.T.T. 1977). The present report deals with the pattern of the disease as seen in the general population. To the best of the author's knowledge, it is the first report of its kind from India.

Material and Methods

The field survey was carried out in a random sample of the population over the age of 3 years, in a selected area of Delhi, using the household as a sampling unit. The details of the survey have already been published (Radha *at al.* 1977). The MRC-ECCS questionnaire was employed for recording the data. All those patients who complained of having had persistent phlegm for 3 consecutive months for 2 successive years, were considered as cases of chronic bronchitis in the field. To exclude the patients with localised diseases like tuberculosis, bronchiectasis, lung abscess etc., giving rise to this symptom, confirmatory studies were carried out. A detailed clinical history was taken and the signs recorded. Peripheral blood eosinophilia and sputum eosinophilis were counted. FVC, FEV1 and PEFr were estimated in all the patients excepting those who showed acute parenchymal lesions on chest radiograph. Sputum was collected from all the patients of chronic bronchitis between December 1974 and the two middle of February 1975 (Winter in Delhi) in order to confirm that all those who gave positive answers to questionnaire did produce phlegm in winter. The quantity of sputum was measured and the purulence graded.

Results

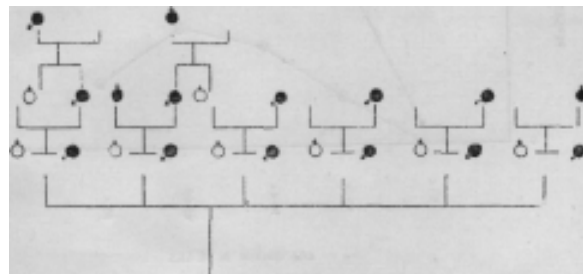
Of the 505 households covered in the survey, 50 had 67 patients with chronic bronchitis. In 11 households more than one member was found

these families the disease affected individuals genetically unrelated, in 6 families a definite genetic pattern could be elicited as shown, in Fig. 1.

Fig. 1

DISTRIBUTION OF PATIENTS IN FAMILIES WITH MORE THAN ONE MEMBER SUFFERING FROM CHRONIC BRONCHITIS

1. Genetically unrelated—□
6 families with 10 individuals.
2. Genetically related—○
6 families with 15 individuals.



The statistical analysis however failed to show that genetic factor had any influence on the occurrence of the disease in the present group.

Pattern of the disease :

The final diagnosis of the 78 symptomatic chronic bronchitics diagnosed in the field is indicated below :-

- 42—Chronic Bronchitis with or without airways obstruction.
- 19—Chronic Bronchitis with Emphysema.
- 3—Chronic Bronchitis with Bronchial Asthma.
- 2—Chronic Bronchitis with Chronic Cor Pulmonale.
- 1—Chronic Bronchitis with Bronchiectasis.
- 5—Pulmonary tuberculosis
- 3—Bronchial Asthma.
- 1—Sinusitis
- 2—Bilateral cystic bronchiectasis.

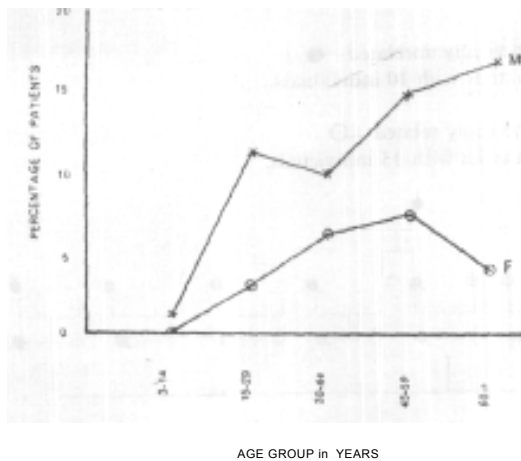
The diagnosis of emphysema and cor pulmonale were based on clinical, radiological and electro-

* Presently working in Sully Hospital, Sully, Wales, U.K.

cardiographic findings. Patients with a 15% reversibility in their airways obstruction to 209 micg. of nebulised Salbutamol and with sputum eosinophilia were labelled as asthmatics. Patients who had a typical history of chronic bronchitis along with sputum eosinophilia and/or reversible airways obstruction were classified under chronic bronchitis. The age and sexwise distribution of those finally diagnosed as chronic bronchitics are shown in Fig. II.

Fig. 2

AGE AND SEX WISE DISTRIBUTION OF CHRONIC BRONCHITIS



The numbers of patients under and over the age of 44 years were equal. There was no evidence of a significant difference ($0.3 > p > 0.5$) in the proportion of males and females, either under or over the age of 44 years. The males however were significantly more in the group as a whole ($p < 0.05$). This male predominance was, however, not seen amongst nonsmokers.

Pattern of symptoms

The mode of onset of chronic bronchitis was sudden in 24 patients (35.8 %) and insidious in 41 patients (61.2%). In 2 patients a definite answer regarding the mode of onset could not be ascertained.

The percentage of patients with persistent cough and sputum for periods under and over 5 years was 67.1 and 32.9 respectively. Of the total number of patients with symptoms over 5 years, only 28.3 % had ever been to a doctor regarding their illness.

In 42 patients (62.7 %), no history of past respiratory illness could be obtained. 25 patients

(37.3 %) complained of having had some respiratory illness prior to the onset of chronic bronchitis. A history of pneumonia was obtained in 20.9 % and non specific acute upper respiratory tract infections in 5.9% of patients. The prevalence of past respiratory illness was significantly more ($p < 0.01$) in families where the affected members were genetically related (66%) as compared to the whole group (37.3%). There was however no statistical difference ($p > 0.05$) between the economic status, ventilation of the house or the type of fuel used for cooking between these families and the rest.

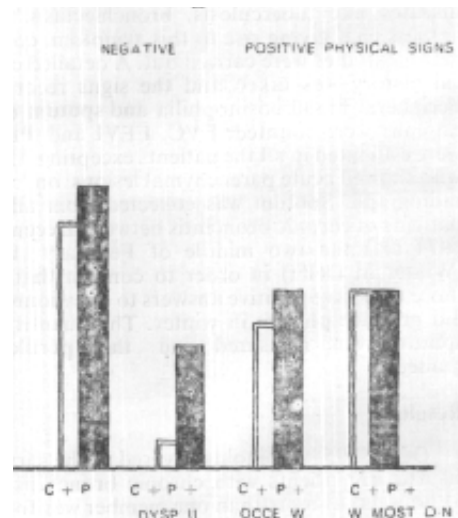
Pattern of Physical Signs

The duration of illness did not affect the presence or absence of physical signs at the time of examination. The proportion of patients having negative and positive auscultatory findings with the duration of symptoms under or over 5 years was not statistically significant ($0.3 > p > 0.5$). Similarly, the mode of onset also did not have any significant effect on the auscultatory findings in these patients.

An attempt was then made to correlate the patients' symptoms with the physical signs and the results are summarised in Fig. III.

Fig. 3

CORRELATION BETWEEN SYMPTOMS AND SIGNS



There was no significant difference between the number of patients with negative and positive

PROFILE OF CHRONIC NONSPECIFIC BRONCHITIS AS SEEN IN A FIELD SURVEY IN DELHI

Table I

Colour of the sputum, sputum cytology and Absolute Eosinophil count

Colour of the sputum	N	SPUTUM		BLOOD
		Polymorphs % X±SE(X)	Eosinophil % X±SE(X)	Eosinophil % X±SE(X)
Mucoid	44	24.1±3.8	7.5±2.3	397±57
Mucopurulent	8	36.5±10.7	6.4±2.0	407±78
Purulent	3	41.0±6.0	6.3±3.3	385±217

Table II

Correlation between Pulmonary function tests and Physical signs

Physical Signs.	Pulmonary function Tests.					
	FEV ₁ %		FRC		RV/TLC %	
	n	m	n	m	n	m
None	27	75.6	15	2280	15	35.2
Rhonchi only	12	65.0*	9	3287**	9	40.7
Rales only	5	67.4	4	2335	4	42.6
Rhonchi + Rales	5	63.9	2	2805	2	63.4*
Hyperinflation only	7	51.3*	5	3802**	5	56.3*
Hyperinflation + Rales + Rhonchi	3	48.8**	2	3954**	2	63.6*
Standard deviation within groups		14.5		702		14.9

* Significantly different from group with no symptoms $p < 0.05$.

** Significantly different from group with no Symptoms $p < 0.01$.

physical signs in any group, except in those with exertional dyspnoea grade II and III, in which the number of patients with positive physical signs was significantly higher than those with negative physical signs. ($0.2 < p < 0.05$).

The correlation between smoking and sputum production was studied. Only 44.7% of the patients were either smokers or ex-smokers. 5 of the 67 patients could not be contacted for various reasons for sputum collection. Of the remaining 51.6% of the patients were found to have more than 5 ml in 24 hours. The quantity of sputum was significantly higher ($p < 0.01$) in smokers and ex-smokers, as compared to those who had never smoked.

Pathogenic organisms could be isolated from only 5 sputa, st. pneumoniae being the organism found in all.

Table I shows the correlation between the colour of the sputum, the cytology and blood eosinophils. There was no significant difference between the mean values of sputum, polymorphs or eosinophils in blood or sputa in mucoid, muco-purulent or purulent sputa. The majority of the patients, however, produced only mucoid sputum.

The correlation between the mean values of FEV₁ %, FRC, RV/TLC, and different clinical signs were tested by analysis of variance and were found to be significantly different in different categories of clinical signs. The least critical difference was worked out for determining the significance of difference in means in each category of clinical signs separately and the results are summarised in Table II.

An attempt was then made to find out if there

was any correlation between, various grades of dyspnoea and hyper-inflation indicated by pulmonary function tests. The RV/TLC was significantly elevated in patients with grade II exertional dyspnoea compared to patients with no dyspnoea. ($F_3 \ 34 = 4.73$). Those with grade III dyspnoea were too small for statistical comparison. No doubt, RV/TLC in grade III was higher than those with dyspnoea.

Table III

Correlation between Dyspnoea and Pulmonary function tests.

Exertional Dyspnoea	Rv/Tlc%		
	N	X	± SE
Grade I	14	46.17	± 4.03
Grade II	5	58.11	± 1.32
Grade III	2	50.53	± 17.07
Nil	16	33.26	± 3.93

It was of interest to note that there was no significant difference ($0.2 > p > 0.3$) between smokers, ex-smokers and non-smokers, with respect to the percentage of patients with or without airways obstruction (FEV 1 % less than 70).

Though FEV1 % values significantly diminished in both males and females with advance in age, neither smoking nor the mode of onset of illness, nor its duration seemed to have any significant effect on the values in each group.

Of the 59 patients who had pulmonary function tests, 37 (64.7 %) had never been to a doctor regarding their illness, while the remaining 22 (35.3%) were already under the care of some physician. When the mean FEV1% values were compared between those who had their symptoms for over and under 5 years in the 2 categories, it was seen that owing to a small sample size, the difference between those with symptoms under 5 years was not significant. In the group with symptoms over 5 years, the mean FEV1% values, in the group that had been to a doctor, were significantly lower than those patients who had not been to a doctor ($p < 0.002$ —Mann—Whitney U test). It was of interest to note that besides the airways obstruction, there was no significant difference between the age, sex, educational and economic level between those who had and had not been to a doctor regarding their illness.

Discussion

N.C. Saha and S.K. Jain 1970 reported the clinical features of 50 patients with an FE V1 % less than 60% attending the out-patients clinic of V.P. Chest Institute, Delhi. Half of these patients were admitted for detailed study. The results were compared to that published by C.M. Fletcher and Burrows (1964). Based on this study they reported a lesser prevalence of smokers, lesser number of patients producing greater than 5 ml phlegm per day, less severe degree of airways obstruction and emphysema, less blood gas disturbances and lesser prevalence of acute chest infection with pyogenic organisms as compared to the group reported by Fletcher and Burrows (1964). The mean age of patients was also lower.

A similar study was carried out by J.S. Guleria, J.N. Pande and R.C. Gupta (1969) in 26 patients, predominantly males with a mean age of 54 years. The patients were divided equally on the basis of diminished diffusion capacity. The authors showed a similar pattern of illness in the 2 groups of patients.

Owing to the very nature of patients selection in these studies, the results cannot be projected on to the overall patients attending the Chest Clinic and certainly not to the general population.

In the present study it was seen that despite the presence of symptoms for over 5 years in 15 (67.1%) of the patients, only 19 (28.3%) had ever been to a doctor regarding their illness. The single factor which was of statistical significance was the much lower FEV1 % values in the latter group. Low FEV1 values were also seen in families where more than one member was affected and more so if they were genetically related. An increased prevalence of past respiratory illness in these individuals may lead one to assume that airways obstruction might well have been due to the damage caused by a past infection. Though smoking correlated well with increased production of sputum, it bore no relation to the airways obstruction. Neither the depth of inhalation of smoke, nor the quantity of tobacco smoked in pack years seemed to have any influence on the routine ventilatory function tests. The lack of correlation may be not only due to the small sample size, as only 29 (99.7%) were either smokers or ex-smokers, but may also be due to the fact that FEV1 or PEFV may not be affected in the initial stages of the illness. The mean FEV1 values of the patients who had been to a doctor, was however, not different from those of chronic bronchitis attending a

Chest Clinic (T.G. Radha and R. Viswanathan 1977).

An interesting finding of this study was that dyspnoea as a symptom was often associated with physical signs and a reduction of FEV1 %. The various grades of exertional dyspnoea also correlated well with the RV/TLC ratios suggesting thereby that a history well taken, combined with physical examination, may give a clue to the severity of airways obstruction.

The number of patients who handed in sputum was 80.1 % (1 hr. after rising) and 81.1 % (in 24 hrs.). Only 12.4% (1 hr after rising), and 11.5% (in 24 hrs.) had returned the bottle with no sputum. Despite the small sample size, this yield is much higher than that quoted by W.W. Holland and D.D. Reid (1965), C.M. Fletcher et al (1965) R. Vander Lender (1969) and W.W. Holland and Stone, R.W. (1965). The difference observed may be due to the fact that the sputa were collected personally by the author. While Holland and Reid (1965) posted the sputum bottles in envelopes, the author handed over glass bottles with cover. Contrary to what Saha and Jain (1970) reported, 51.6% of the patients were found to have more than 5 ml of sputum on rising and 73.8 % to have more than 5 ml in 24 hrs. in the present study. It was of interest to note that only 44.7 % of our patients were either smokers or ex-smokers.

Studies by earlier observers, J. Mulder, J.R.C. Goslings, M.D. Van Der Plaso and P. Lopuz Cordozo (1958), Stuart Harris et al (1953); Elmes et al (1953), (May 1953 and 1959) Knox et al 1955; Edwards et al (1957) have all emphasised the importance of *H. influenzae* and *St. pneumoniae* in chronic bronchitis. Persistence of these organisms in the bronchial system, being treated for any length of time by antibiotics has been shown by Cooper et al (1961); May (1965); Elmes et al (1965); Jenne et al (1970). In the present study, however, despite the fact that sputa were collected in winter, most of them were mucoid and only a small fraction of them grew *St. pneumoniae*. Of the 5 sputa from which the organism was obtained, 3 belonged to patients who were under care of a doctor and 2 to patients who had never been to a doctor. The absence of infection in our patients to the extent quoted by the above workers, might be one of the factors responsible for a higher mean FEV1 values in our patients.

A long term comparative study of these two groups of patients who visit a doctor regularly and those who do not, may answer the questions as to the role of smoking, environmental pollution

and infection in causing and perpetuating airways obstruction.

Acknowledgements

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PNEUMOCONIOSIS

S.K.CHATTERJEE

(From National Institute of Occupational Health, Ahmedabad)

Introduction

The Pneumoconiosis, has come from Greek word *Tivev'mwv Lung; novu dust*. History records diseases arising from dust exposure from very early times. As early as 2,300 years ago Hippocrates and Pliny described breathing difficulty amongst miners. Agricola, in 1558 mentioned about the importance of ventilation in the mines for he knew that dust caused "diseases associated with dyspnoea ultimately leading to consumption." In 1672, Isbrand in Holland, described in his book called "anatomy-corpris-humani", how several stone-cutters died of asthma and he found at post mortem that cutting their lungs was like 'cutting a mass of sand'. Ramazzini (1713) described the breathing difficulty of stone-cutters and how they turned asthmatic and consumptive.

In 1867 Zenker coined the word pneumoconiosis. Pneumoconiosis is defined as retention of any dust particle in the lymphatic depot of the lungs. The term carried no implication of fibrosis or any other reaction nor does it imply disturbed function of the lungs.

Dust—The causative factor

Pneumoconiosis is a disease which is caused by dust in a physical environment created by industrial processes. Much of this is often released in the immediate area where men and women are working. Dust is formed by reducing solid material to small sizes. Processes like grinding, crushing, blasting, drilling etc. produce dust particles of sizes from the microscopic to the visible; their composition being the same as that of the parent material if not altered chemically during the subdivision. Frequently the percentage of the hard mineral such as quartz may be less in the fine dust than in the parent material. Common examples are the minerals or inorganic dusts derived from the disintegration of rock and the vegetable dust like wheat and flour. Smoke from burning carbonaceous fuel like coal, oil, wood etc. all contain droplets as well as dry particles. Tobacco, for instance produces a wet smoke compound of minute tarry droplets. The particle size of tobacco smoke is about 0.25 micron.

When a solid is broken into finely divided particles and released into air, one of the important changes that take place is that the space occupied by the broken material and also the

surface areas are increased many times from that of the original mass. For example, when one CC (0.61 cu in) of quartz is crushed into particles of one cubic micron in size, there will be 10^{12} (1000,000,000,000 one trillion) particles with total surface area of six square meters (9300 sq. in) as compared to six square centimeters (0.930 sq in) for the original block. If we assume a dust concentration of 100 million particles to each cubic foot of air, then one C C of material will occupy on air space of 10,000 cubic feet.

The dust particles, except in the case of asbestos, must be smaller than 10 micron (one micron equal to one twentyfifth thousands of an inch) in their largest dimension in order to enter the inner recesses of the lungs where damage is caused. Hay fever and other allergic types of diseases arise from the larger particles of organic dust which cannot reach the lung at all. The chemical and mineralogical make up of the dust are the deciding factors in determining its injurious properties. It has been shown that silicon dioxide (free silica) in the form of quartz may produce disabling fibrosis whereas little or no fibrosis is produced by the equally hard and sharp cornered aluminium oxide (emery).

The 'organic dust' is divided into (a) Non-living organic dust (b) Living organic dust. The Non-living organic dust may be further subdivided in (1) Toxic or irritant dusts like those producing local irritant symptoms or producing general toxic symptoms or the dust may be both toxic and irritant (2) Allergens—A wide variety of substances may be responsible for allergic reactions in various individuals who are susceptible to these but may have no effect on others. A single substance may in one case cause intestinal disturbances, but in another cause asthmatic attack or in third case may cause skin eruption, and still another person may be affected in all three ways.

Living organic dust includes bacteria like anthrax bacillus found in the dust from skin, fur wools or diphtheria; tubercle, typhus and other bacilli. Bacterial sensitized dust can also be the cause of allergic diseases such as asthma, hay fever, eczema. Dust containing parasitic fungi like moulds on straw, hay, grass may cause some annoyance and discomfort.

The inorganic dust is mostly mineral in origin. The dust may be toxic and/or irritant, fibrosis-

producing or non-fibrosis producing. Toxic and/or irritant inorganic dust comprises heavy metals like lead, mercury, arsenic, cadmium, zinc etc and their compounds. In the fibrosis producing dusts, the most outstanding example is quartz,

Asbestosis	:	Silicate—3 MgO.2 SiO ₂ —2H ₂ O
Talcosis	:	Silicate—3MgO.4 SiO ₂ —H ₂ O
Coal Miner's Pneumoconiosis:	:	Coal dust-carbon.

Pathology of Pneumoconiosis

It has been found that dust behaves in one of the three ways in the body tissues. Dust particles may be absorbed, cause cellular proliferation or remain inert in the tissues. Accumulation of some amount of inert dust particles usually takes place in the lungs due to inhalation from atmospheric air. Strictly speaking, therefore, all adults living in industrialized city have pneumoconiosis, which means "dust retained in the lungs." Where the atmosphere has much coal smoke and soot in the air, the storage of black soot particles in the lungs results. There is generally no functional disability in this type of pneumoconiosis.

Storage of insoluble dust in the lungs is in direct proportion to the concentration and duration of dust inhalation and inverse proportion to the size of the dust particles. When one is exposed to abnormal amount of small sized, (0 to 10 micron size) dust particle over a long period of time, the self cleaning mechanism of the lungs becomes overloaded. The surface of the alveoli becomes covered by dust particles. The phagocytes become dust laden and collect in the perivascular and peribronchial sheaths. The lymphatic vessels, lymph glands and subpleural space become distended and clogged by the dust-laden phagocytes and give the appearance of a lace work of linear dust deposits. This condition is called 'Pneumoconiosis'. Pulmonary function is not impaired in most cases. If, however, the dust produces proliferative fibrosis and damages the bronchioles, alveoli and the pulmonary blood vessels, severe emphysema may result with, pulmonary disability. A useful classification of pneumoconiosis according to the lung pathology can be made under three headings' Major, Minor and Benign as suggested by Johnstone and Miller.¹

MAJOR PNEUMOCONIOSIS Marked tissue reaction

Entity	:	Dust
Nodular	:	Free Crystalline SiO ₂
Non Nodular Silicosis	:	Ultra microscopic-crystalline SiO ₂
Silica pneumonitis	:	Calcined diatomite-crystalline SiO ₂
Diatomite Pneumoconiosis	:	Crystalline SiO ₂
Shaver's Disease	:	SiO as a fume.

MINOR PNEUMOCONIOSIS Little Tissue Reaction

Anthracosis	:	Soot and Carbon
Diatomaceous Earth Pneumoconiosis	:	Amorphous SiO ₂
Silicatosiis	:	More or less complex silicates with silica bound in molecule.
Micas (pure)	:	
Clays	:	
Fledspars	:	
Vegetable Dust Pneumoconiosis	:	Various organic dust
Mill fever	:	Cotton
Byssinosis	:	Mouldy suger cane
Bagassosis	:	Mouldy hay, straw
Farmer's lung	:	Grain
Grain asthma	:	Tamarind seed
Tamarind asthma	:	Mouldy cotton yard.
Weaver's cough	:	

BENIGN PNEUMOCONIOSIS No tissue reaction

Baritosis	:	Barium sulphate—BaSO ₄ (pure)*
	:	Barium oxide—BaO (Pure)*
Siderosis	:	Ferric oxide—Fe ₂ O ₃ (pure)*
Stannosis	:	Stannic oxide—SnO ₂ (pure)*
Titanosis	:	Titanium-dioxide TiO ₂ (pure)*
Graphosis Chalcosiis	:	Graphite—carbon (pure)*
Limestone marble, cement	:	Calcium salts (pure)*

*The word 'pure' is used to denote absence of silica.

Pneumoconiosis in India

India has a glorious tradition in architecture, sculpture and stone carving which have flourished right from the times of Ashoka. The temples of Chola and Pandya dynasty and the tombs, palaces and forts of the Moghul period are other examples. Many masterpieces of architecture were built by Rajputs. These wonders reflect the price that must have been paid by stone cutters, sculptors and quarrymen. Ajanta & Ellora which had to be burrowed through the solid

face of a mountain, chiselling, inch by inch, making vast halls, dormitories, Chaitas and Vihars, carving of grand statues, stupas etc. all these titanic masterpieces in granite and other rock, must have left behind a vast trail of dust disease.

Silicosis in Mines

The report of Capalanl² as early as 1940 showed presence of silicosis in Kolar gold mines. Silicosis is caused by free silica dust and is within the perview of Pneumoconiosis. From 1940 to 1970, 3,61,210 cases have been diagnosed as cases of silicons to whom compensation has been paid in Kolar gold mines.

In a survey carried out by the Chief Advisor of Factories (CAF)³ in mica mines in 1953 in Bihar, it was found that 34.1 % had nodular Conglomerate silicosis amongst 329 mica miners examined. The prevalence of pulmonary tuberculosis was found to be 18.6% among them.

The coal workers' Pneumoconiosis

The existence of Pneumoconiosis amongst coal workers of India was first reported by Roy in 1956.⁴ The CAF organisation carried out a survey in the coal field of Jharia and Raniganj in 1960-61 and found prevalence of pneumoconiosis to be 18.8% in a population of 950 miners examined. In another study carried out later by Central Mining Research Station involving 1000 coal miners, 3.7% definite cases of Pneumoconiosis and 11.4% doubtful cases were found. In 1964 a second study⁷ by CMRS in a new coal mine was carried out to get a picture of pneumoconiosis among fresh recruits. This study showed an incidence of 8.5% of Pneumoconiosis.

Pneumoconiosis in other types of mines

X-ray study⁸ of 158 mines in Chromium and magnisite mines which was carried out by CAF in the year 1952 did not reveal the presence of silicosis in mines. The CAF⁹ carried out a study in the year 1961 in collaboration with Chief Inspector of Mines, Dhanbad in lead and zinc mines in Rajasthan. The study showed 52 cases of silicosis out of 171 miners examined radiologically. This is really a very high incidence. In a very recent study in an iron ore mine in collaboration with Chief Inspector of Mines, Dhanbad found 42 radiologically abnormal cases (23.7 %) among 177 miners. The lung function tests which were included in the study showed certain deterioration, but it was within the range of findings in miners having normal lung picture. The radiological abnormality in

Iron ore miners (Siderosis) is usually due to the high refransibility of iron dust though its effect on the lung tissue is not very toxic. However, if silica is associated with iron ore, then it may complicate the picture by producing the disability of Pneumoconiosis, known as Sidero-silicosis.

Pneumoconiosis in Factories

First report on the occurrence of silicosis among surface workers was brought out in a paper by Sikand and Pamra⁽²³⁾ at the seventh Tuberculosis conference in November 1949. They reported that 28 (66.7%) out of 42 stone cutters and 3 (12.5%) out of 24 stone breakers showed x-ray evidence of silicosis.

A study was carried out by CAF¹¹ in close collaboration with Chief Inspector of Factories in Bihar in refractory industries. It was found that out of 338 workers examined, 16.7% showed silicosis in silica brick making and 22.8% in fireclay brick making. Sabnis et al¹² found 5.3% cases of Pneumoconiosis amongst 604 workers working in six foundries. CMRS^{13, 14} carried out a study very recently in 14 factories manufacturing refractory materials in Bihar. The total number of workers examined was 1032 of whom 100 were female workers. The male workers showed 1.3% radiological abnormality, though not a single case of silicosis was found among female workers.

Pneumoconiosis due to cotton dust

In India the average daily number of cotton textile workers has risen from 826,00 in 1961 to 1,018,000 in 1973, being the largest in any single industry.

The Indian Council of Medical Research (ICMR) has supported many research projects on Pneumoconiosis commonly known as Byssinosis related to cotton dust. The results of ICMR and other studies are shown in the Table below :

It would be seen that cases of Byssinosis vary from 0.01 to 27.0 %. The difference may be due to differences in assessment of chest symptoms in various studies. But, it shows beyond doubt that Byssinosis, which is also a kind of Pneumoconiosis, is a big problem in the cotton textile industry.

To conclude, Pneumoconiosis is a major problem in mines, factories etc. and requires a careful assessment through longitudinal studies in depth for working out a programme for its prevention.

Table I

Incidence of Byssinosis in Textile Industry in India

	ICMR studies					Other studies			
	Madurai (15)	Ahmeda- bad(16)	Delhi (17)	Madras (18)	Bombay (19)	Bombay (20)	Bombay (21)	Kanpur (22)	
No. of Mills studied	1	13	1	1	3	1	1	2	
No. of workers examined	900	253	1241		D.E. 899	Con. 342	786	485	656
No. of cases of Byssinosis	1	16	80	66	102	7	58	37	21
Percentage of cases of Byssinosis	0.01	6.3	8.4	4.2	27	5	7.57	7.6	3.2

* Presently working in Sully Hospital, Sully, Wales, U.K.

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CURRENT STATUS OF SURGERY IN PULMONARY TUBERCULOSIS

G.M. MASCARENHAS, H.N. ANANDA AND V. ANANDA RAO
(From St. John's Medical College Hospital, Bangalore.)

Surgery still has a definite place in the treatment of pulmonary tuberculosis in our country in contradistinction to other developed countries in Europe, US and Japan where tuberculosis has been controlled with modern chemotherapy and surgery has very little place in the management of pulmonary tuberculosis. It has been provided by experience that the control of tuberculosis, by and large, follows the economic prosperity of a country. But in our country we are still faced with the problem of economic development and as such, surgery, though necessary in a very small percentage of patients, has a place in our tuberculosis control programme.

It is generally agreed that any patient who is a suitable candidate for surgery should have had sufficient medical treatment before being subjected to surgery. The period varies from 6 months to a year. We have generally preferred to accept at least 9 months of medical treatment with suitable drugs and in accordance with the criteria or indications which are fairly standard. We accept patients for surgery when there is a status quo, that is, when no further improvement with drugs is possible and successive X-rays of the chest show that the lesion is persistent and constant.

Indications

The following indications have been adopted by us over the past 21 years:

- (a) A destroyed lung provided the opposite lung is healthy or has a minimal lesion.
- (b) Collapsed or atelectatic lobes provided the rest of the lungs are healthy.
- (c) Bronchial stenosis due to tubercular endobronchitis.
- (d) Residual cavities, provided there is sufficient healthy lung left behind for adequate respiratory function. Under this heading can also be included Bilateral Surgery.
- (e) Bronchiectasis as a result of tuberculosis provided the patient has symptoms of bronchiectasis *i.e.*, cough and purulent sputum. Mere bronchial distortion on bronchogram is not a criterion for surgery.

- (f) Tuberculoma—any solid focus which is more than 2 cm. in diameter.
- (g) Haemoptysis—which cannot be controlled by coagulants, pneumoperitoneum etc.
- (h) Tubercular Empyema—with or without a bronchopleural fistula.

The Pre-operative criteria for accepting a patient as a suitable candidate for surgery

- (a) The patient should have adequate respiratory function. This may be assessed by simple tests like making the patient walk up a flight of stairs and noting the degree of dyspnoea following the manouvere. Should the patient have the accessory muscles of respiration contracting vigorously or excessive intercostal indrawing, he is a poor surgical risk.
- (b) The presence of emphysema which can be assessed radiologically on X-rays and flouroscopy and by timed vital capacity.
- (c) The general condition of the patient—the chronically ill-patient who is emaciated, is a poor surgical risk.
- (d) Presence of bronchial asthma increases the risk of surgery, unless it can be controlled with drugs.
- (e) In long standing cases of tuberculosis, especially when the lung is fibrosed and contracted and the pleura is very thick, problems may arise during surgery in the form of shock and excessive bleeding. However, this can only be assessed during the time of surgical intervention.

Pre-operative Investigations

1. Plain and lateral X-ray of the chest are usually sufficient. Only rarely is tomography required.
2. Sputum examination—Direct smear and concentration method for AFB is done. Even if the sputum is positive this has not affected our decision regarding surgery.

3. Sputum culture and sensitivity for AFB may be useful. But we have not used this since the advent of more modern drugs. However, sputum examination and culture and sensitivity tests for non-tubercular organisms should be carried out. This includes culture and sensitivity of Pus aspirated from an empyema.
4. Bronchoscopy is done in all cases. We don't perform bronchograms for tuberculosis cases, unless it is to demonstrate a bronchial stenosis.
5. Simple respiratory function tests, such as vital capacity, timed vital capacity and as mentioned before in some cases assessing the degree of dyspnoea by making the patient walk up a flight of stairs may be done.
6. In older patients it is preferable to assess the cardiological status with an ECG etc.
7. The presence or absence of diabetes is to be noted in all cases.
8. The haematological status should be assessed.

The Pre-Operative Treatment

Prior to 1976 an operative cover of Streptomycin, INAH Ethambutol and sometimes Cycloserine and Pyrazinamide were used. These drugs were started at the time of admission for surgery which was on average a week before surgery and were continued until the patient was returned to the referring physician. For the past 2 years we have used Streptomycin, INAH Ethambutol and Rifampicin in calculated doses. This is an excellent operative cover and post-operative complications have been minimal.

(b) Breathing exercises to improve pulmonary functions are very important. From our experience, we have found that a woman when admitted has a vital capacity of about 800 and after a few days of physiotherapy the vital capacity has increased to 1,400.

(c) Correction of blood deficiency especially Hb are done by means of blood transfusion and haematenics.

(d) Control of diabetes is important. We normally start these patients on plain insulin three days before operation and examine the urine every 6 hours. This is carried out throughout the post-operative period of 10 days.

Surgical Techniques

Early Surgery is employed in only 2 instances.

1. Haemoptysis.
2. Tuberculoma.

The standard procedures viz. pneumonectomy and lobectomy are done. In addition, we have also performed segmental resections wherever feasible. Wedge resection has been employed in cases of tuberculoma. Bronchial Anastomosis after excision of the stricture has been done in some cases provided the lung distal to the stenosis is healthy. In those cases requiring bilateral surgery, we have done surgery at an interval of one month.

Tubercular Empyema is managed as follows:

In the acute stage, daily aspiration with instillation of antitubercular drugs such as streptomycin or INAH solution, is carried out; but in our experience most tubercular Empyemata are accompanied by a bronchopleural fistula. Repeated aspirations do not help these patients. Therefore, closed intercostal drainage in the dependent position with the aid of pleurogram is done. The subsequent management depends on the status of the underlying and the opposite lung.

In those cases with extensive bilateral tuberculosis closed intercostal drainage may have to be carried out for several months and when lung expansion has occurred, open drainage has been carried out. The treatment for these cases may extend upto 1 year, and healing of the empyema is usually accompanied by healing of tuberculosis. In those cases where the opposite lung is healthy of tuberculosis. In those cases where the opposite lung is healthy and the Empyema has become chronic as evinced by thickening of the pleura the procedure performed is decortication with or without resection of the underlying lung. This may sometimes be a lobectomy or a pneumonectomy. Some of these necessitate resection of the parietal pleura in addition to the empyema wall.

Problems During Surgery

1. Whenever we come across tubercular disease densely adherent to the parietal pleura we employ extrapleural dissection. This is especially so in the region of the apex. Extrapleural dissection may have to be carried out extensively. This results in excessive bleeding or oozing. A simple way of controlling small bleeders is to pack the area with swabs soaked in warm saline and using pressure. The bleeders which usually require to be caught are the intercostals, branches of the Azygos vein, some branches of the peri-

cardiacophrenic vessels and the internal mammary artery. This technique saves a lot of operating time instead of meticulously cauterizing all oozing points.

2. Excessive air leak from cavities may be prevented by dissecting out the main bronchus as a preliminary measure and clamping it especially when a pneumonectomy is planned.
3. Bleeding from the branch of pulmonary artery can sometimes be attended to by catching the bleeding point with a haemostat and ligature. But sometimes there may be a tear in the pulmonary artery. It is not necessary to remove the lung. While pressure is exerted on the tear, the main pulmonary artery is dissected out and a ligature passed around it and the artery compressed. The tear can then be identified and most often can be closed with 5 zero atraumatic arterial silk. Tears in difficult dissections of main pulmonary artery and pulmonary veins that cannot be controlled directly may necessitate opening of the pericardium and intrapericardial ligature of these vessels.
4. To prevent leaking from the bronchial stump we do not strip the bronchus of surrounding tissues. The bronchus is clamped distally and divided. Even the subcarinal glands are not dissected. The main bronchial stump is closed by 5 interrupted stitches and is covered by surrounding tissues which may be pleura or sometimes an intercostal muscle. The cautery is never used to arrest bleeding from the bronchial vessels. If necessary, ligature is used.

Post-Operative Complications

1. Bleeding is always treated by replacement of blood. Major bleeding may require reopening of the chest. This has happened only on three occasions out of a total of 6321 thoracotomies. In two instances, bleeding was found to be from an intercostal artery and on one occasion from a small artery on the auricular appendage after a Mitral Valvotomy.
2. Effusion is treated by aspiration but most often by insertion of Malecots Catheter connected to an underwater seal.
3. Bronchopleural fistula—showing an air fluid level on X-ray. After correct localisation, it is treated with intercostal tube drainage. After

lobectomy, basal bronchopleural fistulae are treated in this manner in addition to pneumoperitoneum if necessary. Apical bronchopleural fistula treated with intercostal tube drainage rarely require a small thoracoplasty. In fact, with prolonged drainage, these fistulae normally close within a period of 8 weeks.

The major problem in lung surgery is the infected pneumonectomy space with or without a bronchopleural fistula. When a pneumonectomy space is infected, aspiration followed by instillation of an appropriate antibiotic after culture sensitivity test of the aspirated Pus usually helps. More recently we have drained the pneumonectomy space and daily instillation of broad spectrum antibiotics has helped to clear the infection. A real major problem after pneumonectomy is a bronchopleural fistula from the main bronchial stump. Patient normally coughs out the contents of the pneumonectomy space and after radiological confirmation the pneumonectomy space is drained with closed drainage. We have had good results with a continuous instillation of broad spectrum antibiotics such as Garamycin instilled twice a day into the pneumonectomy space with clamping of the IC tube for 1-2 hours daily. The worst problem a surgeon is faced with is the large bronchopleural fistula due to giving way almost completely of the bronchial stump. This is fortunately a rare occurrence and is invariably fatal in spite of reopening the chest and redoing the bronchial stump and covering the bronchial stump with an Intercostal muscle graft. In some cases where the space is large in spite of drainage we have succeeded in obliterating the pneumonectomy space with a 1-7 rib thoracoplasty done at one stage. This is usually done after a period of 6-8 weeks when the mediastinum is fixed and there are no chances of paradoxical movement of the chest wall on the affected side.

In the later post-operative period after discharge, patient is referred back to the consulting physician and we are happy if antitubercular drugs are continued for a further period of 1-2 years.

Needless to say the one dread facing the surgeon in the surgery of PTB is the recurrence of the disease. This we have found in those cases which are lost to follow up or who failed to continue chemotherapy during the above period. In only a few cases we have been able to re-operate with success. The rest have to be managed medically.

CHRONIC APICAL PNEUMONIA

B.K. KHANNA

(From K.G's Medical College, Lucknow)

In an earlier article on the subject (Khanna 1969), the author presented 8 cases of chronic apical pneumonia. Out of these, in 3 cases, the pneumonia complicated pre-existing pulmonary lesions in the upper zones, *e.g.*, pulmonary tuberculosis, tropical pulmonary eosinophilia and apical bronchiectasis.

Since then the author has collected another 20 cases of chronic unresolved pneumonia in the upper zones of the lungs, which in many of them, was mistaken and treated for pulmonary tuberculosis.

Material and Methods

The records of 25 cases of chronic apical pneumonia admitted to this department from 1961 to 1976 have been scrutinised. This includes 5 uncomplicated cases of apical pneumonia already reported in 1969 (Khanna, 1969). Cases were labelled as suffering from chronic apical pneumonia only when

- (a) the sputum was found to be repeatedly negative for AFB on smear examination;
- (b) The chest x-ray revealed evidences of segmental and/or lobar consolidation in one or both the upper lobes;
- (c) Bronchoscopy failed to reveal any obstructing pathology in the related lobes or segments;
- (d) The illness had been present for over 6 weeks.

Out of these cases 22 (88 %) were males and 3 (12%) were females. The mean age was 33 years, though the youngest case was 18 years old and the oldest 80 years of age. The mean duration of illness was 14 weeks (range 8 to 32 weeks). All of them had received chemotherapy with antituberculosis drugs (21 cases), injections and capsules (4 cases).

Following the hospitalisation, the doubt, regarding the presence of chronic unresolving apical pneumonia, was raised by the clinical features of the cases, which have already been described in detail (Khanna, 1969). Culture of the sputum for secondary pathogens revealed KI pneumonia in 7 cases (28 %), Staph. Aureus in 3 cases (12%) and pseudomonas pyocynous in

2 (8 %). In the remaining cases the culture was reported sterile. The chest x-ray revealed evidences of pneumonia in upper zones of lungs in all the cases. Cavities within the consolidated area were seen in 6 cases (24%).

Four patients received antituberculosis drugs comprising streptomycin 1G/M1 once a day, Isoniazid 300 mgms. per day and thioacetazone 150 mgms. per day in the hospital owing to the mistaken diagnosis of pulmonary tuberculosis. The mistake was soon rectified and the patients were switched over to antibiotic therapy. The antibiotic therapy consisted of oxytetracyclin (11 cases), ampicillin (4 cases), crystalline penicillin (5 cases), sulphamethoprim (3 cases) and penicillin and streptomycin administered together (2 cases). 9 cases had a change of therapy due to unsatisfactory radiographic clearance. The change of therapy was done according to the culture and the sensitivity pattern of the organisms cultured from the sputum.

The final results revealed that 3 patients had left the hospital against medical advice and the remaining had had a complete clearance of their lesions. One case had the destroyed upper lobe which had to be resected out.

Discussion

Posterior segment of right upper lobe (and corresponding apico-posterior segment of left upper lobe) and apical segment of lower lobes are the common sites for the localisation of the tuberculous lesions. These sites are also the favoured sites for aspiration pneumonia (Brock, 1954) especially when the person is lying in supine position. This complication is seen very frequently after a bout of alcoholism followed by "passing out", when the person is vomiting and retching and may inhale his own vomitus during the act. The pneumonia produced as a consequence may be sterile due to chemical agents in the gastric secretions or be superinfected. None of our patients admitted to have "passed out" after an attack of alcoholism, though 8 of them had had a history of having taken alcohol at one time or the other. Thus, alcoholism does not appear to have contributed to the development of apical pneumonia in this series.

Sputum failed to reveal any pathogenic organisms in 14 cases. This could be as a consequence of antibacterial drugs received by these cases

prior to their admission to this hospital. The commonest pathogen isolated was Kl pneumonia. Klebsiella (friedlander's bacillus) pneumoniae can cause chronic infection of the upper lobe of lungs though rarely (May, 1972 and Narang et al, 1975). Staph. aureus may, sometimes, cause pneumonia; though these may sometimes, cause pneumonia; though these may be present as a non-pathogen in chronic bronchitis during remission. The same holds true for the pseudomonas species (May, 1972).

Cavities in the area of pneumonia were seen in 6 cases, 5 of them had a history of bad breath which may signify anaerobic infection (Lorber, 1975). 3 of these cases had recurrent haemoptysis, with chemotherapy cavities disappeared in 3 cases. In the other 3 cases, thin-walled ring shadows were still present at the time of discharge. Surgery, to resect out the unresolved pneumonic lobe was done in one case. The histology confirmed our clinical diagnosis.

The response to chemotherapy on the whole was extremely satisfactory, leading to almost complete resolution of the pneumonic lobes or segments. Residual fibrotic strands or infiltrates left after chemotherapy continued to resolve and/or fibrose under the influence of chemotherapy advised to be taken by the patients in the follow-up period.

Summary

25 cases of chronic apical pneumonia have been presented. Klebsiella appeared to be the most common pathogen encountered. Chemotherapy alone led to complete resolution in 21 cases. In one, chemotherapy followed by resection of the diseased lobe was done. 3 cases left the treatment and the hospital against medical advice. Alcoholism did not appear to predispose to this malady atleast amongst our cases.

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CHOLESTEROL PLEURAL EFFUSION

S.D. PUROHIT, G.S. SHARMA and K. BANERJEE
(From R.N.T. Medical College, Udaipur)

Introduction

Churten (1882) was the first to describe cholesterol pleural effusion. Number of workers reflected diverge etiological factors for the causation of this rare entity. Curran (1946-48) stated that less than half of the cases of cholesterol pleural effusion had tubercular etiology. Weems (1918) considered deposition of cholesterol in pleural effusion is due to faulty cholesterol metabolism. Desbordes and Levy (1938) raised the importance of albumin/globulin ratio as a factor operating as cholesterolytic or precipitating effect on cholesterol.

As far as our knowledge goes patient of cholesterol pleural effusion has been reported from our country.

Case Report

Mr. K. Singh 24 years young engineer came on 14.7.76 with the complaints of left sided chest pain for the last two years. The pain was dull and localised to the lower part of the left chest. The patient had a jeep accident in June 1973 as a result he had bruises on the legs. Two months after the accident he felt mild pain in the left side of the chest. After about six months he started getting exertional dyspnoea, but there were no constitutional symptoms. He was diagnosed as a case of left sided pleural effusion. 300 c.c. of pleural fluid was aspirated from left pleural cavity and he was advised on inj. streptomycin and INH 300 mg/day with supportive treatment. He continued the therapy for 30 days and stopped the treatment without consulting the treating physician.

After two years pain in the left chest re-appeared. He consulted his family physician at Sirohi, who diagnosed him as a case of pleural effusion of tubercular origin and 500 c.c. of the fluid was aspirated from the left pleural cavity. The fluid was golden yellow in colour. He was advised streptomycin one gm. daily + INH 300 mg with ethambutol 25 mg/kg body weight for two months followed by INH + ethambutol for the next four months. The treatment was stopped by the family physician.

The dull ache in left side of chest still persisted for which he came to our hospital. He was well built; not anaemic and there was no lymphadenopathy. Pulse was 82/mt. Resp. 18/mt.

B.P. 128/78. Examination of chest revealed findings of left sided pleural effusion. C.V.S., G.I.T. and neurological examinations were normal. X-ray chest of the patient revealed findings of moderate pleural effusion on left side. Under local anaesthesia he was aspirated twice in one week and 900 c.c. (750 c.c. + 150 c.c.) of pleural fluid was aspirated from the left pleural cavity.

Laboratory investigations

Blood Hb. 12.5 mg%, R.B.C. 3.8 million/cumm., TLC 7600, p. 67 %, L. 30%, Mono 0%, E. 1 % fasting blood sugar 82 mg %, blood cholesterol 132 mg%, urine and stool examinations were normal.

Pleural fluid

The fluid was opalescent with its characteristic shimmering satin. On Chemical examination the specific gravity of the fluid was 1.020, proteins 4 mg%, cholesterol 512 mg%, sugar 67 mg%. On microscopic examination, characteristic cholesterol crystals were clearly seen. There was no evidence of any cells in the pleural fluid. The opalescence of the fluid did not clear when it was shaken with ether and alkali. Staining of the fluid with sudan III was negative for fat. Culture of the pleural fluid was sterile. Pleural biopsy did not reveal any pathology.

He was given an injection procaine penicilline 4 lac. O.D. for 10 days, analgesics and B complex and was advised active exercises, roentagenogram repeated after one month showed obliteration of the left costophrenic angle.

Discussion

Patient was aspirated four times in two and half years duration and had anti-tubercular drugs for a period of six months. Curran et al reported tuberculosis as a possible cause of cholesterol pleural effusion but this was unlikely in our case as patient neither had constitutional symptoms nor there was any response with anti-tubercular therapy. Moreover the colour of the tubercular pleural fluid is straw coloured but in cholesterol pleural effusion opalescent colour of the aspirate itself is suggestive. The characteristic silvery flashes evident by shaking the pleural fluid, in a test tube indicates the probability of cholesterol as dominant constituent of pleural fluid.

The possibility of the chyle in this case is ruled out as the staining with sudan III was negative. The retention of the opalescence of the pleural fluid after adding ether and alkali, rules out the possibility of fat globules.

In few cases these effusions do not recur after the initial aspiration but not infrequently repeated aspirations are necessary. Repeated aspiration may lead to bacterial contamination of pleural fluid, or formation of broncho pleural fistula. Henceforth decortication usually leads to resorption of the pleural fluid. Surgery was planned in our case too but patient left the hospital against medical advise. Most often quiescent nature of the long standing pleural effusion favours the non-acceptance of surgical intervention.

Early diagnosis is of great help. It will avoid prolonged anti-tubercular therapy or with other anti-biotics. This case indicates that all the pleural effusions must be aspirated and should not be taken as tubercular just on roentegenography

only. Surgical treatment for cases of cholesterol pleural effusion is ideal with prospects of our result as majority of cases are between the age of 20 to 40 years.

The normal convention for the failure to response by the patients indicates change of therapy. This condition may be of borne in mind in patient of pleural effusion who fail to respond to treatment, thus avoiding delay in diagnosis and institution of proper treatment including surgical.

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BOOK REVIEW

PROFILES OF CLINICAL PRACTICE BY K.S. SANJIVI; PUBLISHED BY ORIENT LONGMAN LTD., MADRAS; 1976 PRICE: RS. 17.00.

Before independence medical students, both under-graduates and post-graduates, general practitioners and specialists had to depend on standard text books written by western authors, based by and large on their experience in those countries. During the last few years a number of books written by Indian authors and based primarily on Indian conditions have been published in practically all disciplines of medicine. These books are gradually replacing many of the earlier text books. Whenever a new book is brought out, one naturally asks "why another book on a subject on which many other books have been written by Indian authors"? The author himself has posed this question and given an answer which is, by and large, correct.

This book is entirely different from the usual books on medicine. It is a distinct departure from the conventional and orthodox style. Neither it covers all diseases nor everything known about the diseases that have been covered. Thus, while it may not meet the requirements of an under-graduate who has to pass an examination, it should fulfill admirably the requirements of a general practitioner whose main aim is to suspect, diagnose and treat the common diseases met with. "A general practitioner's priorities lie less in pursuit of knowledge about rare conditions but more in making full use of existing knowledge of common conditions for the benefit of the greatest number", says the author. The topics chosen are no doubt common illnesses which a basic doctor has to deal with practically every day but all such diseases have not been covered by the author. For example, the author states "it is not proposed to discuss chronic bronchitis in this book for the simple reason that the condition is extremely rare in South India." Even if one accepts that chronic bronchitis is more frequent in North than in South India, exclusion of chronic bronchitis from the book is exceptionable since it is not written *only* for the practitioners of South India.

The author is a very popular teacher of long standing and a well known clinician and consultant. Naturally he knows the needs not only of students but general practitioners also. The style of the book is easy, interesting and in fact inimitable. Some of the salient points have been highlighted by brief summaries of illustrative cases which are bound to leave a more lasting impression. Many punches have been pulled and many pertinent and important remarks have been

clothed in pun and wit. For example when dealing with the physical signs in pulmonary tuberculosis the author says "physical signs do not tell lies when they are present, although they do not tell the truth when they are absent." While discussing the surgical treatment of peptic ulcer, it is said "the most important indication for surgery was the training needs of the doctor." All this makes the reading as delightful as it is instructive.

Another very welcome feature of the book are the chapters on community medicine and the general practitioners opportunity and problems vis-a-vis the health care delivery. The author has spelled out the details of the 'mini health centre project' which he started in and about Madras some years ago and is reported to be working very successfully. The chapter also provides an opportunity to the author for expounding his own philosophy of health care for the community—a philosophy which is realistic, sensible, efficient and yet feasible.

Every one has his fads and fancies, so also authors. Dr. Sanjivi under-rates the importance of statistical analysis in the chapter on introduction. According to him, observations of the physician are more important than statistical analysis. Many would not agree with this view. Impressions, since these are based more on the rare and unusual occurrences rather than the more frequent and every day experience, are not likely to be correct always. It is only a statistical analysis that presents the problem in a correct perspective. Obviously it is because of the author's faith in observations that some statements have been made during the course of the book with which most people may not agree. For example, the author advocates essential hospitalization at start of treatment for every case of pulmonary tuberculosis; artificial pneumothorax merely for maintaining contact with the patient; hepatotoxicity following pyrazinamide; unlikeliness of achieving reduction in the duration of treatment of pulmonary tuberculosis; effectivity of pyrazinamide as compared to ethambutol role of frequency of repeated infections in the development of clinical pulmonary tuberculosis etc. These few disagreements, however, do not detract from the merit of the book in any way, for nothing any one says is likely to be acceptable to every one else.

Lastly, it is difficult to imagine that a book like this with a fairly good get up, hardly any

printing mistakes, and a number of fairly well reproduced representative skiagrams could be made available at an exceptionally low price of Rs. 17.00 only. No general practitioner and no medical library should be without this book.

All in all, it is a very valuable addition to the existing ones on this subject and the author is to be congratulated on this achievement.

S.P. PAMRA

NEWS AND NOTES

CHAIRMAN

Dr. B. Sankaran who took over as Director-General of Health Services on 4th January, 1978 succeeds Dr. P.P. Goel as the ex-officio Chairman of the Tuberculosis Association of India.

32ND NATIONAL CONFERENCE

The 32nd National Conference on Tuberculosis and Chest Diseases was held in Trivandrum from 23rd to 27th November, 1977. Dr. K.V. Krishnaswami of Madras was the President. The Conference was inaugurated by Smt. Jothi Veneatachellum, Governor of Kerala. Over 350 delegates including a few specialists from outside attended it. Important papers presented at the Conference as also summaries of other papers will be published in the April 1978 issue of the Indian Journal of Tuberculosis which will be brought out as the 'Conference Number'.

33RD NATIONAL CONFERENCE

The Association has accepted the invitation of the Madhya Pradesh Tuberculosis Association to hold the 33rd National Conference on Tuberculosis and Chest Diseases in Bhopal. Subjects selected for the Conference are : (1) National Tuberculosis Control Programme with particular reference to the 6th Five Year Plan, (2) Chemotherapy, (3) Air Pollution, (4) Tuberculosis in Children, (5) Community participation in Tuberculosis Programme, (6) Man power requirements and the training of personnel for Tuberculosis Control Programme, (7) Laboratory support for Tuberculosis Control Programme, (8) Non-Pulmonary TB., (9) Bronchial Asthma, and (10) Tuberculosis of the Central Nervous system including TB meningitis. Those who wish to present papers may inform the Secretary-General, Tuberculosis Association of India, 3, Red Cross Road, New Delhi together with an abstract of the paper latest by 15-3-1978.

HEALTH VISITORS' COURSE

The 1978-79 TB Health Visitors' Course will commence in July, 1978. The course will be of nine months duration, of which five months will be spent in New Delhi TB Centre, two weeks in Rural Health Centre, two weeks for examination and three months internship (including two weeks in a rural centre in Pataudi, District Gurgaon). The minimum qualification for admission to this course is Higher Secondary/Pre-University with Science of Hygiene and

Physiology in matriculation. Applications for admission to this course should reach Tuberculosis Association of India, 3, Red Cross Road, New Delhi-110001 by 30th April, 1978.

CHANCHAL SINGH MEMORIAL AWARD—1978.

The Tuberculosis Association of India will award a Cash Prize of Rs. 500/- to a TB worker preferably below 45 years of age, for an original article not exceeding 30 double spaced foolscap typed pages (approximately 6,000 words) excluding charts and diagrams on a subject relating to tuberculosis. Papers may be sent in quadruplicate, to reach the office of the Tuberculosis Association of India before 31st August, 1978.

TB SEAL CAMPAIGN

Details of the inaugural functions of the 28th TB Seal Campaign held in the States of Delhi, Andhra Pradesh, Maharashtra and Goa, Daman & Diu were given in the October 1977 issue of the Journal. Details of reports received from other State are given below.

In Bihar, Shri Jagannath Kaushal, Governor of Bihar and Patron of the State TB Association inaugurated the campaign and Shri Jabir Hussain, Minister for Health and Family Welfare and President of the State Association presided over the function. The Campaign was inaugurated in Jammu & Kashmir by Shri L.K. Jha, the State Governor, at a function held at Raj Bhavan, Srinagar. In Karnataka the Campaign was inaugurated by Shri Govind Narain, the State Governor at a function held at Raj Bhavan, Bangalore. In Kerala the Campaign was inaugurated in Trichur by Shri K.K. Balakrishnan, State Minister for Irrigation. In Madhya Pradesh Shri S.N. Sinha the State Governor inaugurated the Campaign. In Pondicherry, Thiru D. Ramachandran, Home Minister of Pondicherry inaugurated the Campaign on the occasion of the opening ceremony of the Government Dispensary at Ariyur on 2nd October, 1977. In Punjab Shri J.L. Hathi, the State Governor inaugurated the Campaign.

REFRESHER COURSE

A Refresher Course in Tuberculosis was sponsored by the Tamil Nadu Tuberculosis Association at Madras Medical College on 5th January. Shri P. Murari, I.A.S., Commissioner and Secretary to the Government Health and Family Welfare, Madras, inaugurated the

course. Brig. Prof. B. Ramamurthi, Professor and Head of the Department of Neurosurgery and Principal, Madras Medical College, presided.

COMMUNITY WELFARE PROGRAMME

The Government Chest Institute and TB Demonstration and Training Centre, Madras, participated in the community welfare programme to extend Mass Miniature X-ray facilities to those who had suspicious shadows in the Miniature X-rays taken earlier. The programme included (1) 'Lung Screening Programme' sponsored by the Lions Club of Ennore-Thiruvottiyur covering the villages of Ennore and Factory workers of Ennore Foundries, (2) 'Mass Screening' of students and inmates of the Indian Institute of Technology in September 1976 sponsored by the authorities of the Indian Institute of Technology, Madras, (3) 'Community Health Check up including Lung Screening' sponsored by the Lions Club of Madras East, (4) Participation in the Exhibition conducted at Thiruvannamalai, (5) 'Community Health Care Camp' at Koyambedu Village sponsored by the Lions Club of Thirumangalam, (6) 'Mass Chest Screening Programme' for the employees of the Ennore Thermal Power Station in compliance with the request of the authorities thereof, (7) 'Lung Screening Campaign' sponsored by the Lions Club of Minjur, (8) 'Lung Screening Campaign' sponsored by the Lions Club of Central of Madras for the inhabitants of Kotturpuram, (9) Participation in the All-India Tourist Trade Fair 1977 by extending Mass X-ray facility at the Medical Pavilion, as part of Health check up, and (10) Health Check-up including Lung Screening programme for the hand cart pullers and cycle rickshaw drivers sponsored and conducted with the cooperation of the police authorities.

SHIBIR/CONFERENCES

Maharashtra State Anti-TB Association organised a Shibir at Chirner in Kolaba District in cooperation with the Rotary Club of South Bombay. The Association also participated in Twin Anti-TB Shibirs at Chopda in cooperation with Chopda branch of National Integrated Medical Association and at Deep Nagar Power Station, Bhusawal arranged by Maharashtra State Electricity Board on 17th and 18th November, 1977 respectively. The Shibirs were specially undertaken to coincide with the 17th Maharashtra State Indian Medical Association Conference held at Jalgaon from 18th to 20th November and to demonstrate to the delegates the working of Shibirs so that they could take similar work in their area. During the Shibirs held at Chopda

and Deep Nagar Power Station, Bhusawal (Dist. Jalgaon) the team of specialists and technicians lead by Dr. M.D. Deshmukh examined a total of 1,410 persons, screened 392, x-rayed 71 positive cases and vaccinated 7,665 persons.

The 7th Karnataka State TB Conference was held on 29th and 30th October, 1977 at Madikeri (Coorg), Karnataka. Shri H.M. Channabasappa, the then State Health Minister, inaugurated the Conference. Shri R. Gundu Rao, the then Minister for Housing & Youth Services, Karnataka, presided over the function. Subjects discussed at the Scientific Sessions included "Childhood TB", "Bronchial Carcinoma", "Coin Diseases of the Lungs". Shri B.M. Cariappa, Secretary-General, Tuberculosis Association of India, presided over the Conference of the Secretaries and the District TB Officers. Shri Cariappa was felicitated on behalf of the Karnataka Association and Shri Channabasappa the then Health Minister of Karnataka presented an Address to him. Dr. H.B. Dingley, Medical Superintendent, L.R.S. TB Hospital, New Delhi, opened the Health Exhibition organised on the occasion.

The Sixth Annual Conference of Gujarat State TB Association was held in collaboration with TB Research Institute of K.J. Mehta TB Hospital, Amargadh on 8th January, 1978. Smt. Hemaben Acharya, Health Minister of Gujarat inaugurated the Conference. Dr. B.N.M. Barua, Adviser in TB to the Government of India, was the Chief Guest. Dr. Barua also inaugurated the Scientific Session. There were two Panel Discussions, on "Why we cannot control TB" and "Treatment of TB".

ITALIANA HONOUR

Shri B.M. Cariappa, Secretary-General of the Tuberculosis Association of India, has been awarded the Honorary Diploma of Federazione Italiana Contro La Tuberculosis E Le Malattie Polmonari Sociali, by the Tuberculosis Association of Italy. While presenting the award at a function held in New Delhi Prof. G. Daddi, former Director of Forlanini Institute, Rome recalled the devoted and selfless services rendered by Shri Cariappa in the voluntary field and said that this has been appreciated all over the world. He wished Shri Cariappa many more years of useful services for this humanitarian cause. The award citation mentioned the "high degree of efficiency" to which Shri Cariappa had taken the Tuberculosis Association of India himself "well-deserving not only towards his great country but also towards fight against TB in the world."

S.M.S. COLLEGE CELEBRATIONS

“Three Decades Celebrations of S.M.S. Medical College, Jaipur and Old Students Silver Jubilee Meet, 1978” will be held on 17th, 18th and 19th of February, 1978, at S.M.S. Medical College Campus, Jaipur to mark the educational academic, research and the cultural activities achievements of the Institute.

MEMBERSHIP ON THE I.U.A.T, PARIS

The Tuberculosis Association of India is enrolling Ordinary Members on the International Union Against Tuberculosis, Paris, for the year 1978. The annual subscription is F.F. 125.00 equivalent to Indian rupees 230/-. By virtue of this membership the members will receive from the International Union, free of cost, copies of

its Bulletin and publications of the World Health Organisation concerning subjects of concern to the Union and Union's circular letters and scientific and general information documents. Those interested may send their subscriptions to the Secretary-General, Tuberculosis Association of India, 3-Red Cross Road, New Delhi-110 001, latest by the 1st March, 1978.

OBITUARY

Dr. E.P. Jesudasen, Honorary Secretary, of the Pondicherry TB Association died on 6th December, 1977, of a sudden heart attack at Pondicherry. Dr. Jesudasen was an active worker in the tuberculosis field. The Tuberculosis Association of India conveys its deepest condolences to the members of the bereaved family.

NOTICE TO SUBSCRIBERS

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The Indian Journal of Tuberculosis

ABSTRACTS

Vol. XXV

January 1978

Abst. No.

Pulmonary Aspergilloma. A report of twenty-five patients.

G.C. Bower et al. Amer. Rev. Resp. Dis.; 1977, 115(Suppl. April), 90.

Twenty-five patients with pulmonary aspergilloma were seen in a period of seven years. Cavitory tuberculosis was the underlying lung disease in 15 (60%). Ten had coexisting severe chronic non-tuberculous lung disease and 7 had alcoholic liver disease. Haemoptysis occurred in 18 (72%) and the classic radiographic manifestation of mycetoma was present in 18. The agar gel double diffusion test for precipitating antibodies against *Aspergillus* was valuable in the identification of the species causing the colonization and in assessing the course of the disease, whether followed medically or treated surgically. *Aspergillus* precipitins were present in 17 of 18 (94%) and the sputum culture grew *Aspergillus* in 15 of 23 (65%). Fibroptic bronchoscopic examination helped to localize the site of intrapulmonary bleeding when it occurred. Of 14 patients who underwent surgery, two (14%) died within two months of surgery and in 7 patients serious post-operative complications occurred including empyema, chest wall abscess and pneumonia. During long-term post-surgical follow up new bronchiectasis, new mycetoma and hemoptysis occurred. Of 11 non-surgical patients, one died during follow up from hemoptysis and another from respiratory failure. Eight are doing well during follow up and have had no haemoptysis; one has had episodes of bleeding. It is believed that surgical resection for complex Aspergilloma should be reserved for patients with significant, recurrent haemoptysis.

S.P.P.

Radiographic features of pleural effusions in pulmonary embolism

L.J. Bynum and J.E. Wilson, Amer. Rev. Resp. Dis.; 1977, 115 (Suppl. April), 94.

Prospective study of 120 cases of pulmonary embolism (PE) revealed radiographically evident pleural effusion in 60 (50%). Excluding patients

with other possible causes for effusions left 45 patients whose effusions were confirmed by lateral decubitus chest roentgenogram within 3 days of admission, were re-evaluated radiographically every 1-4 days and followed for 10 days or until resolution of the effusion. Decubitus skiagrams were repeated when necessary to delineate the course. All effusions were present at admission and none increased after the second skiagram taken within 1-3 days. All were unilateral, on the same side as chest pain, and on the side where emboli were seen by scan or angiogram. Although 28 patients (62 %) had bilateral emboli, pain was bilateral in only one and effusions were never bilateral. Infiltrates accompanied the effusions in 22 patients (49 %). All effusion occupied less than 33% of the hemithorax; 35 (78%) were less than 20%; mean was 15%. Of 22 effusions associated with infiltrates, 19 (86 %) were larger than 15% of the hemithorax; only 4 of 23 (17%) without infiltrates exceeded 15% ($P < 0.001$). All effusions associated with infiltrates persisted beyond 7 days; 20 of 23 effusions (87 %) without infiltrates cleared within 7 days ($P < 0.001$) and 5 were no longer visible after 3 days. Effusions appeared more than 2 days after PE in 2 of the original 120 patients; both had clinical and scan evidence of recurrent PE at the time. Thus PE causes pleural effusion in half the cases, and half of these have radiographic evidence of infarction. The effusions are unilateral and small; those not associated with infarction are smaller and clear more rapidly. Uncomplicated PE is an unlikely cause of pleural effusion which is massive, bilateral, or increases in size after the initial event. Increase in size or re-appearance of an effusion several days after PE suggests recurrent PE. The mechanism of effusion in PE is unknown but is probably the same as pain, since both are almost invariably unilateral and ipsilateral even with bilateral PE.

S.P.P.

Vanishing lung: Clinical and radiographic features.

J.F. Simeone et al., Amer. Rev. Resp. Dis.; 1977, 115 (Suppl. April), 163.

Six patients (5 males, 1 female, ages 31-60

years) with giant compressive bullous emphysema (Vanishing Lung Syndrome) were studied during the past seven years. Although only 2 of 6 had respiratory symptoms, all had pulmonary function abnormalities. Arterial hypoxemia was present in each case (PaO_2 62 to 76 mmHg). The mean one-second forced expiratory volume (FEV_1) was 58% of predicted, range 44-79% and $\text{FEV}_1/\text{forced vital capacity}$ 67%, range 62-77% indicating an obstructive ventilatory defect. Lung volumes revealed a normal total lung capacity in all five patients studied and an increased residual volume in two suggesting air trapping. Diffusing capacity for carbon monoxide (single breath method) was 19-70% of predicted, average 45%. Chest radiographs in all cases demonstrated bilateral giant bullous disease, primarily in the upper lung zones with marked compression, of remaining lung parenchyma. Ventilation/perfusion lung scans were frequently misleading, appearing to show perfusion of areas shown by chest radiographs, conventional tomography, pulmonary angiography, and computerized tomography to contain little lung parenchyma. This observation supports the previously reported impression that ventilation/perfusion lung scans may be misleading in these patients. Computerized tomographic images provide a new, precise method of evaluating the distribution of bullae and of compressed but otherwise normal lung parenchyma. In the four patients in whom follow up data are available, there has been progression of radiographic abnormalities and deterioration of pulmonary function tests. One of these patients required bullectomy for recurrent pneumothoraces. Patients with giant bullous emphysema frequently are asymptomatic in spite of an obstructive ventilatory defect and far advanced radiographic abnormalities and computerized tomography provides an accurate, non-invasive method for determining the extent of bullous disease. In most patients this disease appears to be progressive.

S.P.P.

Three years prospective study of respiratory disease in rubber workers

L.J. Fine et al. Amer. Rev. Resp. Dis.; 1977, 115 (Suppl. April), 214.

In 1972 and 1975, two hundred and five (205) workers in the rubber industry were surveyed to determine the effect of their occupation respiratory symptoms and pulmonary function. In 1972, pulmonary function testing included one second forced expiratory volume (FEV_1 , forced vital capacity (FVC) expiratory flow rates at 50%, 25% and 12.5% of the vital capacity and closing

volume (CV) determinations. In 1975, simple pulmonary function testing (FEV_1 and FVC) was conducted. The rubber workers were divided into three groups: 31 talc workers exposed to a non-fibrous talc, 73 curing workers exposed to fumes emitted during the vulcanizing of tyres, and 101 workers who were not exposed to any dust or fume. During the three year study period the yearly average decline in the FEV_1 was more than twice that of the non-smokers, 64 ml versus 21 ml for the curing room workers and 82 ml versus 21 ml for the talc workers. Since the rate of decline in FEV_1 in healthy populations containing similar proportions of cigarette smokers is approximately 30 ml a year, we believe that the curing and talc areas are examples of occupational exposures which, in combination with cigarette smoking, produce loss in ventilatory capacity sufficient to result in the development of clinical chronic obstructive lung disease in those who work in these areas for prolonged periods.

S.P.P.

Congenital diaphragmatic hernia: Studies of lung composition and structure.

W.R. Blackburn, P. Logsdon and J.A. Alexander, Amer. Rev. Resp. Dis., 1977, 115 (Suppl. April), 275.

The composition of lungs from infants dying of pulmonary insufficiency associated with persistent left pleuroperitoneal canal and the herniation of abdominal organs into the thorax was examined. The ipsilateral, hypoplastic lung (HL) and the contra lateral, control lung (CL) were studied by biochemical and morphological methods. The infants had not undergone surgical correction. Age matched non-hernia lungs served as a second control (CCL). Total lung wet weight (HL+CL) was significantly ($P < .001$) reduced in hernia infants as compared to CCL. H-Lungs were severely reduced in wet weight and contained fewer cells ($P < .02$) as calculated from DNA analyses (23.54 mg/HL vs 103.33 mg/CL). H-Lungs contained less total lipid (1.12 mg/mg DNA) than C-Lungs (1.56 mg/mg DNA). The phospholipid fraction was only slightly depressed (0.46 mg/mg DNA vs 0.55 mg/mg DNA) Surface active lecithin, "surfactant" was significantly ($P < .02$) decreased in H-Lungs (0.22 mg/mg DNA vs 0.37 mg/mg DNA). Morphologic studies further supported the biochemical data, demonstrating decreased branching, immature epithelial surfaces, reduced numbers of lamellar granules in type II pneumocytes but no significant change in the ratio of type II cells to other pneumocytes. Our data indicate that the H-Lungs is hypocellular, biochemically immature, and surfactant deficient.

Morphologic studies indicate that growth retardation is symmetrical rather than "dysplastic". Collectively these findings offer an explanation for the observed susceptibility of the H-Lung to hyaline membrane disease and odema following surgical correction.

S.P.P.

The effect of acetylation status on isoniazid (INH) hepatitis

D.S. Dickinson, W.C. Bailey, B.I. Hirschowitz, Amer. Rev. Resp. Dis.; 1977, 115 (Suppl. April) 395.

The postulate that rapid acetylation is a risk factor in INH hepatitis has been examined in a prospective study of 66 patients receiving preventive INH (300 mg daily) for upto nine months. A careful history (including other drug therapy and previous hepatic dysfunction), baseline liver function tests were obtained prior to INH therapy. Patients were deemed to have evidence of liver damage from INH if the following conditions were met : normal baseline SGOT, abnormal SGOT < 60 KU on two or more occasions (at least 2 weeks apart), and SGOT greater than 4 x mean baseline value on one occasion. Seven patients (10.6%) had evidence of liver damage. Furthermore, acetylation status was repeated after three months therapy in half of the patients and showed no induction of the acetylation enzyme system. Chi square analysis for incidence of hepatitis disproves the postulate that rapid acetylation is a risk factor for INH hepatotoxicity ($P < .05$).

S.P.P.

Treatment of pulmonary tuberculosis with short-course, intermittent chemotherapy using Rifampicin-Isoniazid.

A.K. Dutt, L. Jones and W. W. Stead. Amer. Rev. Resp. Dis.; 1977, 115 (Suppl. April), 396.

Since December 1975, 113 bacteriologically positive patients with pulmonary tuberculosis in 64 countries have been treated according to a protocol using Rifampicin (RIF) 600 mg & INH 300 mg daily for 1 month followed by twice-weekly supervised medication (RIF 600 mg and INH 900 mg) for 8 months. Special care is taken in monitoring for bacteriological response, drug resistance and drug toxicity. Of the 113 patients, 62 were white, 50 black and 1 oriental. Only 10 dropped out due to toxicity, death or initial INH resistance. Of the remaining 103 patients in various stages of therapy, sputum conversion by culture occurred within 3 months in 77 of 87 patients (88 %) and within 6 months in all but one of 82 patients (98.8%). RIF toxicity (fever, jaundice, nausea, rash) occurred during the daily

phase in 3, thereafter in 2. No hematological or anaphylactic toxicity from RIF has been observed to date. In 2 patients the twice-weekly INH dosage was reduced to 600 mg because of troublesome symptoms. RIF-INH as used in this study is highly effective against bacteriologically proven tuberculosis. The reduced total quantity of drug necessary for a complete course of therapy makes this regimen economically feasible. The danger of administering RIF twice weekly appears not to be as great as it has been stated to be.

S.P.P.

The booster phenomenon in serial tuberculin testing.

J.L. Glassroth and N.J. Thompson, Amer. Rev. Resp. Dis.; 1977, 115 (Suppl. April), 398.

Previous reports suggest that a "booster effect" may create difficulties in interpreting serial tuberculin skin tests. In order to study this phenomenon, healthy volunteers were given a series of skin tests over a period of one week. Tests were read 48 hours after administration. All participants initially received 5 TU PPD-T. Seventy percent also received a companion test containing 5 units PPD-Gause. Repeat PPD-T tests were given either at 48 hours or one week after initial testing. A total of 1521 persons completed all assigned tests. The mean size of repeat tuberculin tests was significantly larger than the mean for initial tuberculin tests. If an increase of PPD-T reaction of > 6 mm of induration from < 10 mm to < 10 mm is considered boosting, then 54 persons (3.6%) boosted.

Initial PPD-T and PPD-Gause reactions were compared for boosters receiving a Gause test. No significant difference in frequency of boosting was found when persons with initial PPD-T < PPD-Gause were compared with those having reactions in a reverse relationship. It is concluded that boosting does occur in all age groups studied but is more common in persons < 35 years and boosting can be produced by sensitization with *M. tuberculosis* or other mycobacteria. Because it is more prevalent, non-tuberculous mycobacterial sensitivity may be the most common cause of boosting.

S.P.P.

Comparison of 12 versus 18 months of chemotherapy after sputum conversion among tuberculosis patients initially treated with three Rifampicin-Isoniazid.

Mary W. Long et al. Amer. Rev. Resp. Dis.; 1977, 115 (Suppl. April), 403.

A total of 822 patients with newly diagnosed

pulmonary tuberculosis were randomly assigned to one of three daily rifampicin-isoniazid (RIF-INH) regimens : RIF 450 mg, 600 mg or 750 mg in combination with INH 300 mg. Following initial 20 weeks of RIF-INH therapy, patients received INH 300 mg and ethambutol (EMB) 15 mg/kg for either 12 or 18 months after their sputum cultures became negative. The rate of bacteriological conversion of sputum among the three RIF-INH regimen was compared in 552 patients who completed 20 weeks therapy. About 60 % of these patients also completed their assigned INH-EMB therapy and were examined for relapse for at least 1 year after therapy was stopped. There was no significant difference in the rate of sputum conversion or rate of relapse between the patients receiving RIF 600 and those receiving RIF 750 mg. However, the RIF 450 mg regimen was significantly less effective than the other two regimens as manifest by a lower rate of sputum conversion and a higher rate of relapse. Further analysis showed that RIF dosages of less than 9 mg/kg/day are inadequate for the treatment of pulmonary tuberculosis. There was no significant difference in the rate of relapse between the patients receiving 12 months of chemotherapy and those receiving 18 months of chemotherapy after bacteriologic conversion. The risk of relapse was highest among patients who failed to complete the initial RIF-INH phase of therapy.

S.P.P.

Toxicity to Isoniazid and Rifampicin in active tuberculosis patients.

K.P. Ramakrishnan et al., Amer. Resp. Dis.; 1977, 115 (Suppl. April), 405.

Isoniazid and Rifampicin have been found to be very effective in treating active pulmonary tuberculosis. One hundred twenty-nine patients were treated with Isoniazid and Rifampicin in 1975. Nine of these patients (6.9%) had significant drug toxicity and the drugs had to be discontinued. Seven patients had hepatotoxicity, two had significant renal toxicity; two patients also had thrombocytopenia. These patients all had far advanced active pulmonary tuberculosis and six of them were heavy alcoholics. In contrast, in a review of 266 patients treated with a combination of Isoniazid, Ethambutol and Streptomycin clinically significant drug toxicity was found only in nine (3.4%) patients.

Seven patients had hepatotoxicity, one had serum sickness; one had severe vertigo. The incidence of significant drug toxicity seems to have increased since the use of the combination of Isoniazid and Rifampicin in treating active pulmonary tuberculosis. Both Isoniazid and Rifampicin are hepatotoxic in varying severity. In addition, Rifampicin is also found to cause acute renal failure and thrombocytopenia. The combination of these two drugs seems to enhance the development of drug toxicity especially in alcoholics.

S.P.P.

Effect of repeated testing of same site with PPD-T in non-reactors

W.W. Stead et al., Amer. Rev. Resp. Dis.; 1977, 115 (Suppl. April), 408.

Because of evidence of tuberculosis within a state prison, it was necessary to conduct several mass testings in 1974-76 to identify cases and to institute prophylactic INH for converters. A question was raised as to the possibility that some of the "conversions" might be due to repeated use of the same testing site rather than to new infection. The matter was studied at a regularly scheduled mass retesting of 328 inmates who were non-reactors 2 months earlier and 23 whose previous reaction had been less than 13 mm. Identical antigen (5 TU PPD-T) was injected into the skin of the repeatedly used volar surface of the right fore-arm and into a corresponding site on the left which we had never used before. The inmates had all been tested 1-5 times before within the past year or two including once within the previous 2 months. Reactions were read 48 hours later. In 301 there was no reaction on either arm and in 7 the reaction responded within 1 mm for a concordance of 9 %. In 21 the reaction on the right was an average of 3.4 mm greater than on the left, while in 21 the left exceeded the right by an average of 3.5 mm. Of the 23 whose previous reaction had been doubtful, 4 showed no reaction in either arm, 7 again showed only a small reaction with little difference between the arms and 12 showed an increase in the reaction to 14 mm or more with little difference between the right and left. It seems likely that these increases were due to a "booster" effect in persons possibly infected with "atypical" mycobacteria. It is concluded that repeated testing at the same site with PPD-T does not produce false positive results.