pressure[13]. To produce pulmonary hypertension, its vascular network must be grossly reduced[14]. Pulmonary vasconstriction can also occur if the bronchial passages are blocked causing alveolar hypoventilation[15]. If the bronchial passage is restored, ventilation improves and pulmonary vascular pressure is restored to normal.

Symptoms are insidious at onset and the disease takes a long protracted course. Initial manifestations are often overlooked and are considered to be due to pulmonary tuberculosis itself. So, an early diagnosis of tuberculosis and institution of proper, regular and adequate treatment help to stave off the development of cor pulmonale. In many cases, development of cor pulmonale is noticed when features of congestive heart failure become apparent. Patients, observed in hospitals, represent only a preselected group and many of them are from lower economic level of the society. Those who can afford prefer treatment at home or in nursing homes. Thus, quite a number of cor pulmonale cases today go unnoticed and so tuberculosis remains as a relatively uncommon cause of chronic cor pulmonale. We have reasons to believe that cor pulmonale, following tuberculosis, is not uncommon. Moreover, due to potent chemotherapeutic drugs, a significant portion of residual deaths after tuberculosis are due to cor pulmonale, even when tuberculosis itself has been arrested[8].

As tuberculosis is still the most prevalent chronic respiratory malady in India, the incidence of chronic cor pulmonale as an after effect of tuberculosis is not likely to be so low. With high prevalence of lung tuberculosis among poverty striken people, with inadequate medical facilities and incomplete treatment, number of cor pulmonale cases are likely to be more. Hence, a long term study, using a standard parameter of serial lung function test, at least in hospitalised pulmonary tuberculosis cases, may reveal more probable cases of cor pulmonale in future[5]. Further, investigation by clinical examination, x-ray, ECG, blood gas studies and follow-up of these cases may provide us with a more accurate answer of incidence of the disease.

MANISH CH. PRADHAN

[13]DENOLIN, H.—*Verhj Deutsch Ges Kreislaufforsch*, 21:217, 1955, quoted by [10]McMichael, John.

[14]BOLT, W., FORSSMANN, W. AND RINK, H.—Selektive Lungenaugiographie, 1957, Stuttgarh, quoted by [10]McMichael, John.

[15]ROSSIER, P. H., BUHLMANN, A. AND WIESINGER, K.—Physiologie Und pathophysiologie der atmung, 1956, Springer-Verlag, Berlin, quoted by [10]McMichael, John.

CURRENT TOPIC

Tuberculosis in India—A Perspective

D. R. NAGPAUL*

Although tuberculosis is known to have existed in India from time immemorial, not much is known about how it was distributed, its spread, and the disease trend till lately. Much of the information in the Vedas and Ayurvedic Samhitas deals with the prominent clinical features of the disease and the kind of patients who might respond to certain treatment. Neither notification nor health statistics existed then, that could have thrown light on the rise and fall of the disease in the country. The information that has become available over the last few decades, excepting properly conducted epidemiological surveys, also is not very reliable. Therefore, conflicting claims are often made about the disease being on the increase, endemic or declining in the country with no means available to confirm the claim.

TUBERCULOSIS EPIDEMICS :

Like acute infectious diseases, the chronic infectious diseases also spread in communities as epidemics. But, the characteristic secular curve of these epidemics is not easily discernible. Complete information about a tuberculosis epidemic is not available in any country of the world. In some European countries notifications of tuberculosis deaths became a regular practice from late 18th century and the mortality curves drawn on this basis suggest that a tuberculosis epidemic may last from 200 to 400 years (Grigg, 1958). If so, the doubt arises whether ancient countries with thousands years of tuberculosis experience have had one or several tuberculosis epidemics. In view of paucity of information, some degree of conjecturing may be permissible on the basis of the known facts.

For projecting a proper conjecture one must also draw upon the accepted typical features of the disease during the ascending and descending phases of the epidemic. During spread of the disease in "virgn soil", the infection, disease and death rates are all high and there is little difference between them; the dsease is acute, rapidly progressive and often fatal, and is concentrated among the young as well as the city dwellers who live under conditions favouring dissemination. Spread to rural areas occurs much later, but in a very similar manner. During the descending or waning phases, however, the above mentioned rates are not only comparatively low but very different from each other. The disease now is chronic, slow or indolent, fibrotic, not so fatal and concentrated among the elderly. During the endemic phase, all the features of the waning phase prevail but, barring the temporary fluctuations, there is little or no change in the various rates over long periods.

Grigg (*loc. cit.*) also made several other significant observations about tuberculosis epidemics, based on theoretically drawn epidemic curves supported by many a piecemeal observation from several different countries. Thus, the tuberculosis epidemic curves of different countries or the different regions

National Tuberculosis Institute, Bangalore
*B.Sc., M.B.B.S., T.D.D., Director

of the same country may appear to be different (but are not really different) due to varying socio-economic environment and resultant host-parasite relationship. Similarly, the peaks of tuberculosis infection, morbidity and mortality may occur at different times and to different extents in a country for different age, sex and ethnic groups. In other words, to gauge a tuberculosis epidemic in its true perspective, a broad review spread over as long a period as possible is necessary, because the statistics of a decade or two, collected from different regions and selected consciously or unconsciously for diffrent reasons, may more often mislead than tell the true position.

TUBERCULOSIS IN INDIA :

With regard to India, no direct evidence is available as to the number of epidemics that the country has had so far, nor is the true position on the current tuberculosis epidemic curve known.

The most prominent findings of the National Tuberculosis Survey (ICMR, 1959), Tuberculosis Prevalence Survey in Tumkur (Raj Narain *et al.*, 1963) and Tuberculosis in a Rural Population of South India, a five-year Epidemiological Study (National Tuberculosis Institute, 1974) are that (i) the tuberculosis morbidity in India is largely confined to older age groups, (ii) prevalence in the rural areas is similar to that in cities and (iii) the gap between infection and disease rates (38 per cent and 0.4 per cent respectively) is very larger indeed. All the 3 features satisfy the already mentioned characteistics of the declining and or endemic phases of the epidemic. Being a scientifically collected epidemiological information, it should reliably mean that the epidemic is waning or has become endemic.

The tuberculosis mortality information for India being deficient and not very reliable still apprears to suggest that the disease since the turn of the century has followed the same general trend as in some western countries endowed with the requisite notification data. Rogers (cited by Lankaster, 1920) at the time of World War I had estimated on the basis of post-mortem findings spread over 22 years that 17 per cent of the total deaths in hospitals were due to tuberculosis. The decennial estimate of crude mortality during the 1911-21 census period in India was 47 per mille. And, on that basis, the computed tuberculosis mortality would be 800 per 100,000 persons, if hospital deaths were truly representative of the general deaths. Lankaster (*loc. cit.*), after taking note of Roger's estimate of tuberculosis mortality, also quoted the other available statistics: Calcutta 2.1, Bombay 2.8, Madras 2.5 and Ahmedabad 5.9 per mille, and felt that the actual tuberculosis mortality would be 4 per mille or higher in most of the Indian cities. But, that was just about the position in Czechoslovakia (Radkovsky, 1959) around 1880 i.e., 40 years earlier. In any case, this is the earliest available rough estimate of tuberculosis mortality in India. McDougal's (1949) estimate of the specific mortality around 1949, however, was 200 per 100,000 persons whereas that of Frimodt-Moller for the same year for the Madanapalle town and surrounding area was 253 per 100,000 (Frimodt-Moller, 1960). Frimodt-Moller further reported that the mortality in his study area was reduced to 64.1 between 1951-53 and 21.1 between 1954-55 per 100,000 persons, which he could not explain except due to a natural decline plus the rigorous antituberculosis measures introduced by him in the study area. In the Bangalore district, not far from the Madanapalle study area, in the longitudinal epidemiological study done under conditions with no antituberculosis measures at all being implemented, the estimated tuberculosis mortality during 1961-68 has been 100 per 100,000 persons (National Tuberculosis Institute, *loc. cit.*)

It is realised that the quality of the above given mortality statistics for India is very different and there must be some regional variations as well. Nonetheless, the estimates if plotted along a curve depicting the declining mortality in Czechoslovakia since 1880 will show striking similarity of the general trends (Radkovsky, *loc. cit.*). In other words, the tuberculosis epidemic in India perhaps has been following the same course, some 30 to 40 years behind, as in Czechoslovakia at least till 1950, when the era of potent antituberculosis drugs began. Before coming to such a conclusion it would be wise to guard against the possibility that the fall in tuberculosis mortality was not largely or entirely due to a fall in the general mortality. In a way generally similar to that in Czechoslovakia (Radkovsky, *loc. cit.*), the crude mortality in India also came down from 47 per mille in 1911-21 to 15 per mille in 1971, but the fall in tuberculosis mortality has been steeper i.e., falling from about 17 per cent contribuiton to the general mortality in 1920 to about 5 per cent now, comparing favourably with that of Czechoslovakia namely from 15 per cent contribution in 1900 to around 3 per cent in 1957.

In the opinion of many experienced clinicians in this country tuberculosis has undergone a considerable change in its clinical presentation, especially over the last quarter of a century. Many retrospective studies (Tuberculosis Association of India, 1958, 1968) despite their scientific weakness have clearly brought out the gradual change from a comparatively more acute and extensive disease among the young to a more chronic, less extensive disease among the elderly. The near consensus of these reports has been on a marked decrease of the concomitant complications of pulmonary tuberculosis, e.g., enteritis, laryngitis, amyloid disease, matted lymph glands with discharging sinuses, etc. It is significant that very similar changes were noticed in countries where tuberculosis has definitely declined. The evidence in India, therefore, cannot be brushed aside as unreliable.

DECLINE OF ENDEMICITY:

It has been contended that the available direct epidemiological information merely signifies no change in the prevalence of bacillary tuberculosis in the country, at least for about 2 decades (Table 1). And, that equal prevalence in urban and rural areas merely means that we are truly in the endemic phase of the disease. It has also been argued that such a conclusion would be in keeping with the long chequered history of India where empires rose and fell like nine pins leading to wide and repeated dispersal of the population and a good mixing of rural and urban people (Tuberculosis Association of India, 1968). It could be argued with equal force that poverty, malnutrition and congested living, that have been with us for long, would hardly favour the occurrence of conspicuous epidemiological changes over 2 or 3 decades. And, that long range indirect evidence in a chronic disease like tuberculosis cannot have less validity than the direct but short range epidemiological findings.

Some direct evidence in favour of a declining trend, however, has lately become available from the longitudinal epidemio-

TABLE 1—SHOWING PREVALENCE OF TUBERCULOSIS PER 1000 IN VARIOUS EPIDEMIOLOGICAL SURVEYS IN INDIA

Type of disease	Survey period				
	National survey 1955-58 (ICMR, 1959)	Madanapalle survey 1950-55 (Frimodt-Moller, 1960)	Delhi survey 1962 (Pamra, 1966)	Tumkur survey 1960-61 (Raj Narain *et al.*, 1963)	Bangalore survey 1961-68 (National Tuberculosis Institute, 1974)
Bacillary	4.0	4.1	4.0	4.1	4.1
Active abacillary	18.0		13.2	14.9	—
Inactive abacillary	—	10.6		—	—
Inactive insignificant	—	9.1	27.5	—	—

logical surveys. In Table 2, the Bangalore study (National Tuberculosis Institute, *loc. cit.*) is representative of the natural time-trend of tuberculosis in a rural area whereas the Delhi study (Pamra *et al.*, 1968) gives the position in a slum population served by the New Delhi TB Centre providing treatment to the diagnosed patients of the area. In both the studies migration could have interfered with the findings, but the more conspicuous and impressive effect of drought in the former study suggests that migration may not have interfered much. Of course, the observed decline in the already quoted Madanapalle longitudinal survey (Frimodt-Moller, *loc. cit.*) was ascribed to the applied rigorous community control measures. Significantly, the decline observed in the infection as well as disease rates in the Madanapalle rural TB control area and the Bangalore natural time-trend rural area occurred first in the younger age groups, as was to be expected. It would, therefore, be reasonable to infer that there is a gradual but slow natural

TABLE 2—SHOWING LONGITUDINAL STUDIES OF PREVALENCE OF PULMONARY TUBERCULOSIS IN THE DELHI AND BANGALORE AREAS

Delhi area	Prevalence per mille of		
	Sputum positive cases	Abacillary active cases	Total cases
1962	4.0	13.2	17.2
1964	4.0	8.9	12.9
1967	4.0	9.7	13.7

Bangalore area	Prevalence per mille of sputum positive cases in age group (year)				
	5-14	15-34	35-54	⩾55	All
1961-63	0.94	3.77	6.16	11.46	4.06
1962-64	0.72	3.59	5.25	12.08	3.72
1964-66	0.36	3.04	6.04	10.10	3.37
1966-68	0.37	2.58	7.56	12.19	3.93

decline of tuberculosis in the country. That morbidity g easily disturbed by adverse conditions like severe malnutritic drought, etc., may inadvertently lead to the impression th tuberculosis is on the increase in the country or an area.

Apart from anything else, the "no change in prevalenc argument favouring endemicity is not helped by the sizeal and rapid turn-over observed in the composition of prevalen cases. Fifteen villages constituting a part of the sample National Tuberculosis Survey, were resurveyed by the Nation Tuberculosis Institute after 5 years of the first survey (R Narain *et al.*, 1962). The prevalence rates of radiologic disease were 1.7 per cent and 1.8 per cent and of sputum positi cases 0.36 per cent and 0.46 per cent respectively in the 2 surve But, of the 26 bacillary cases of the 1st survey, 14 had die 4 could not be contacted, 6 had either become sputum negati or become x-ray normal/inactive and only 2 had maintain the *status quo* at the time of the subsequent survey. The fre cases of the 2nd survey had come mostly from the x-ray norma and some from x-ray abnormals of the 1st survey. Similar w the experience from the longitudinal epidemiological study the Bangalore area. In other words, the considerable inc dence of fresh disease is being marked by the sizeable sel healing and deaths while in the endemic phase one wou expect low prevalence and incidence rates.

The available evidence strongly suggests that India alrea has had more than one tuberculosis epidemics. At present w are somewhere on the descending limb of the latest epidemi Grigg (*loc. cit.*) believes that the latest tuberculosis epidem in England began in the 16th century and in Europe a hunde years later. As in Eastern Europe, the latest tuberculos epidemic in India may also have begun in the 17th centur The very slow or nil rate of decline is perhaps due to the gene rally unfavourable environment with personal privations an or droughts causing temporary fluctuations in the seeming stable prevalence.

ROLE OF TUBERCULOSIS CONTROL PROGRAMMES:

Can such a slow decline in the epidemic be hastened b tuberculosis control programmes?

Environment is a fundamental factor in the ecological tria of tuberculosis: Socio-economic conditions can alter the ep demiological situation powerfully, for good or bad, over decade or two. Since BCG vaccination has no influence o the naturally infected population and chemotherapy merel eliminates some cases but cannot prevent cases from occurring a tuberculosis control programme has a low potential fo influencing the epidemic curve, over a short period. So fa no reported study has successfully demonstrated the prim influence of antituberculosis programmes in controlling th disease, without a concomitant marked improvement in th standard of living of the people. But, control programme certainly help.

Under the National Tuberculosis Programme, infectiou cases of tuberculosis are being diagnosed at a comparatively late stage. They, presumably, would already have done a major part of the damage (spread of infection) that they ar capable of doing. Moreover, a sufficiently large number o infectious cases (especially in the rural areas) has not so fa come under effective chemotherapy, as has happened in some other countries or under study conditions. In the Madan apalle study area (Frimodt-Moller, *loc. cit.*) with contro

TABLE 3—SHOWING ESTIMATED SPUTUM POSITIVE CASES IN AVERAGE INDIAN DISTRICT WITH AND WITHOUT DISTRICT TUBERCULOSIS PROGRAMME AT THE END OF ONE YEAR

	No. of cases at t_0 (prevalence)	Fate of prevalence cases during one year			Cases added (incidence)	No. of cases at t_1 (prevalence)
		Dead (sputum negative)	Cured (sputum negative)	Remaining (sputum positive)		
Without programme (natural time-trend)	5,000	700	1,000	3,300	1,700	5,000
With programme :						
Not diagnosed	4,224	590	845	2,789	1,700	4,761
Diagnosed	776	147	357	272		
Total	5,000	737	1,202	3,061		

measures applied vigorously it was after 2 decades that morbidity was reduced to less than half. The bacillus also possesses the power of mutation and under ineffective chemotherapy develops resistance to drugs quickly. Therefore, 2 crucial factors are needed before chemotherapy under the programme could help reduce tuberculosis: A significant number of infectious patients brought under effective chemotherapy and a couple of decades, if not more, of efficient effort. BCG vaccination to be really useful must (i) be given correctly and (ii) constantly cover a significant proprotion of the susceptibles in the community. These requirements are difficult but not impossible to meet.

It is only about a decade since our National Tuberculosis Programme has been in operation, but not so effectively. With the data available on the natural time-trend of tuberculosis and the operational study (Baily, 1972) of the average achievements under the programme, a simple estimate of the expected contribution to control of tuberculosis has been prepared (Table 3).

Of the average 776 truly sputum positive cases, including drug sensitive and resitant cases, diagnosed and put on treatment in one year in an average district under the programme, 147 would probably be dead (in spite of treatment), 357 would become sputum negative—after applying differential cure rates for the sensitive and resistant cases, and 272 would continue to be sputum postiive after one year of chemotherapy. With regard to the 4,224 undiagnosed cases in the community, the death, cure and *status quo* rates would be the same as in the uppermost row, as if there was no programme. This would mean that 239 cases would be less in the community after one year under the present efficiency of the programme. This rough calculation without the other epidemiological "flows" means a 4.8 per cent annual decrease, over and above the natural decline, which need not be scoffed at. A corollary would be that dividends would be more if case-findings were to be improved in the programme, rather than treatment results from the present 46 per cent sputum conversion at the end of one year to say 80 or 90 per cent.

SUMMARY

There are reasons to believe that India has had more than one epidemic of tuberculosis since the time of yore. The present epidemic might have started in the 17th century. There is evidence that the present epidemic has been declining since the turn of the 20th century. The natural decline at present is very slow, probably because of the prevailing poverty, malnutrition and over crowding. The District Tuberculosis Programme, even at the present level of efficiency, has a potential of accelerating the natural decline. Improved programme efficiency, especially under case-finding, is likely to produce a quicker decline. Rapid socio-economic development and improved standard of living could lead to a more spectacular decline in tuberculosis, but that effort would not strictly fall within the purview of a specific control programme.

REFERENCES

BAILY, G. V. J.—An Operational Model of District Tuberculosis Programmes, Paper Presented at the ICMR Symposium on Tuberculosis, Madras, 1972.

FRIMODT-MOLLER, J.—*Bull. WHO*, 22: 61, 1960.

GRIGG, E. N. R.—*Amer. Rev. Tuberc.*, 78: 151, 1968.

ICMR—Tuberculosis in India : A National Sample Survey, 1959, Indian Council of Medical Research, Special Report Series, 34.

LANKASTER, A.—Tuberculosis in India, 1920, Butterworths, Calcutta.

MCDOUGAL, J. B.—Tuberculosis Global Study in Social Pathology, 1949, Williams & Wilkins, Baltimore.

National Tuberculosis Institute—*Bull.WHO*, 51: 473, 1974.

PAMRA, S. P., GOYAL, S. S. AND MATHUR, G. P.—Proceedings of the XXIII Tuberculosis and Chest Diseases Workers' Conference, Bombay, 1968, Tuberculosis Association of India, New Delhi, p. 61.

PAMRA, S. P.—Proceedings of the XXI Tuberculosis and Chest Diseases Workers' Conference, Calcutta, 1966, Tuberculosis Association of India, New Delhi, p. 91.

RADKOVSKY, J.—The Share of Tuberculosis in Total Mortality in Czechoslovakia, 1888-1957, Working Paper, International Population Conference, 1959, Vienna.

RAJ NARAIN, GESER, A., JAMBUNATHAN, M. V. AND SUBRAMANIAM, M.—*Indian J. Tuberc.*, 10: 85, 1963.

RAJ NARAIN, JAMBUNATHAN, M. V. AND SUBRAMANIAM, M.—Proceedings of XVIII Tuberculosis and Chest Diseases Workers' Conference, Bangalore, 1962, Tuberculosis Association of India, New Delhi, p. 34.

Tuberculosis Association of India—Changes in the Clinical Manifestation of Pulmonary Tuberculosis, 1958, Proceedings of the XIV Tuberculosis and Chest Diseases Workers' Conference, Madras.

Idem—Symposium on Changing Trends in Tuberculosis, 1968, Proceedings of the 23rd National Conference on TB and Chest Diseases, Bombay.

NOTES AND NEWS

V Asian and Australasian Congress of Anaesthesiology

Under the auspices of the Indian Society of Anaesthesiology the 5th Asian and Australasian Congress of Anaesthesiology will be held at New Delhi on September 23—27, 1978. Over 2000 delegates from different countries are expected to join this first joint Congress to be held in India for the first time. Further details may be had from Dr. G. R. Gode, Department of Anaesthesiology, A.I.I.M.S., Ansari Nagar, New Delhi-110 016.

Stress on Rural Health in the Sixth Plan

The Planning Commission has recommended an outlay of Rs. 1,319 crores for health and Rs. 765 crores for Family Welfare Programmes during the Sixth Plan period. For the year 1978-79 alone out of Rs. 146.48 crores agreed to by the Planning Commission for centrally sponsored and central scheme in the health sector, a major portion of Rs. 115.90 crores would be spent in rural areas. This information was given by the Union Minister of State for Health and Family Welfare, Mr Jagdambi Prasad Yadav to the Consultative Committee of Parliament attached to the Health Ministry which met recently at New Delhi.

Members noted that the proposed allocation for the Sixth Plan was more than double the Fifth Plan provision. However, there was a strong feeling that even the new outlays, which amount to hardly 3 per cent of the total plan provision, were far below the requirements for effectively meeting the health needs of the country.

International Congress on Prevention of Heart Diseases and Cardiac Rehabilitation

The above Congress will be held on September 11—12, 1978 at Bombay. Details may be had from Dr. C. V. Shah, India House, No. 2, Kemp's Corner, Bombay-400 036.

International Congress of Hormonal Steroids

The above Congress will be held at New Delhi on October 29—Novermber 4, 1978. Detailed information may be had from Prof. K. R. Laumas, Chairman and Coordinator, Department of Reproductive Biology, A.I.I.M.S., Post Box No. 4503, New Delhi-110 016.

Indian Association of Sports Medicine

The 8th Annual Conference of the Association will be held on November 4-5, 1978 at Ludhiana. Detailed information may be had from Dr. S. Mukherjee, Department of Physiology, Christian Medical College, Ludhiana-141 008.

II International Migraine Symposium

The 2nd International Migraine Symposium will be held London on September 28—29, 1978. Detailed informa may be had from the Director, The Migraine Trust, 45 G Osmond Street, London WCIN 3 HD.

XI International Congress of Leprosy

The 11th International Congress of Leprosy will be h on November 11—18, 1978 at Mexico City, Mexico. Deta information about scientific programme may be had f Dr. Stanley G. Browne, Secretary General, Internati Leprosy Association, 57A Wimpole Street, London WIM, 7 England and that about registration, hotel reservation, from Prof. Fernande Latapi, XI International Congress Leprosy, Association Mexicana de Accion Contra la Le A. C. or Vertiz 464, Mexico 7, D. F. Mexico.

XIII Session of General Assembly of Confederation of Medi Associations in Asia and Oceania

The 13th (mid-term) Council Meeting and Special Meet of the General Assembly of Confederation of Medical A ciations in Asia and Oceania (CMAAO) will be held on Nov ber 21—22, 1978 in Bali, Indonesia. Further details may had from the Hony. General Secretary, IMA, IMA He quarters, Indraprastha Marg, New Delhi-110 002.

CORRESPONDENCE

The Editor is not responsible for the views expressed by correspondents

Hospital Infections and Human Hair

SIR,—The role of human hair in the causation of staph coccal hospital infection has been underestimated—particul so with the advent of the tonsorial fashions—the hirsute c Stress has always been on nasal and skin carriers and fom etc., references to the human hair being only a few (Sunn *et al.*, 1965; Noble, 1966)

The hospital staff were investigated as a possible sourc dissemination of staphylococci during a recent outbreak staphylococcal infection in a hospital located in a trop coastal town. From the properly "attired" operation the staff, surgeons and attendants, staff from intensive ther wards and postoperative wards, hairs that protruded out of hood and mask were plucked with sterile forceps and drop directly into glucose broth Nasal swabs were also collec on serum coated swabs from the same individuals. Fifty-th per cent of hairs and only 32 per cent of nasal swabs w positive for staphylococci, 15 per cent being sterile.

Isolations from longer hairs (more than 10 cm.) were dou that from shorter hairs.

These isolates were phage typed at the ICMR Staphyloc Phage Typing Centre, Delhi; only 22.9 per cent belonged phage type I, II and III and remaining 77.1 per cent were typable. The latter, as compared to 30 per cent of untypa isolates of Summers *et al.* (*loc. cit.*) from human patholog