ON THE NECESSITY OF TEACHING THE FREQUENCY OF RHEUMATIC INFECTION IN YOUNG INDIANS

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1. *Historical note.*—Only of recent years has the fact of the occurrence of rheumatic infection amongst Indians become widely recognized. Indeed there are still some who do not recognize the existence of rheumatic fever in India at all, whilst perhaps the majority of medical practitioners in India would affirm that rheumatic infection occurs only rarely. It is mainly by the experience and teaching of the professors and lecturers of the medical colleges of India that this obscurity is now being dispelled.

As long ago as 1886, Norman Chevers in his classical *Commentary on the Diseases of India* remarked on the rarity of acute rheumatism in Bengal. 'The experience of a working life time', he writes, 'has taught me that acute rheumatism is rare in Lower Bengal'. He quotes Malcolmsen as having made a similar observation as regards Madras in 1835, 'The common acute rheumatism of Europe is very little known in India', but Morehead of Bombay remarked with greater vision that 'Acute articular rheumatism is not so common in India as in colder climates, yet it is by no means rare'.

Rogers (1910) wrote concerning 'the extreme rarity of rheumatic and scarlet fever in India, and specially in Bengal' and 'the great rarity of true acute rheumatic fever in India . . .'

Megaw (1910) wrote 'on the causation of heart disease in Europeans in India' and investigated two series of heart cases amongst Europeans and Anglo-Indians at the Presidency General Hospital, Calcutta. The first series consisted of 144 cases from 1906 to 1908 and contains 37 cases which were clearly rheumatic, of which 12 contracted their disease in India (4 in the hills and 8 in the plains) whilst 21 were contracted in Europe and 4 were doubtful. One case of chorea was admitted. The second series included 43 cases which Megaw personally investigated in 1909, of which 14 were clearly rheumatic and of which 4 only (one with chorea) were contracted in the plains of India. 'It seems safe to conclude that rheumatism of the type with which endocarditis is associated does definitely occur in the plains of India, but that it is much less common than in colder climates'.

Sir Leonard Rogers (1919) wrote 'Tropical countries differ in a remarkable way from temperate in the almost complete absence of rheumatic fever. Rheumatic fever does occur in hill stations in the Himalayas with European temperatures, but is scarcely ever seen in the hot plains. As a result of the absence of both rheumatic fever and scarlet fever heart diseases differ widely in their incidence. Other affections due mainly to syphilitic atheroma are very common, and organic diseases of the mitral valves proportionately rarer'.

Dr. Basu (1925), Teacher of Medicine, Calcutta Medical School, analysed 446 hospital and private patients examined by him from July 1919 to March 1925 and found (*a*) that 8 per cent of his total 446 cases, (*b*) that 57 per cent of his 28 cases of acute endocarditis, and (*c*) that 47 per cent of his 35 cases of chronic endocarditis were of rheumatic origin. On examining the details of his figures, these percentages certainly do not appear over-estimated. Dr. Basu added that mitral disease is rare, because rheumatic fever is rare in Bengal.

J. T. Clarke (1930), after an exhaustive study of available records, suggested that the disease is practically non-existent in the 'true' tropics. He confines his observations to the strict geographical tropics, and though he quotes the evidence of Calvert and Sutherland to the effect that they had seen rheumatic fever, chorea and nodules in Indian children, which conditions they regarded as rare, he disqualifies this evidence on the grounds of geographical limits. He further quotes Rogers as stating that out of 4,800

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additions in technique, as we feel that owing to the composition of the staff there was no increase in efficiency in 1937 as compared with 1935. We conclude that the 1937 improvement was due to the prophylactic administration of proseptasine. It is worthy of mention that both the medical and nursing staffs were impressed with the smooth clinical course run by many abnormal cases in which our clinical experience led us to expect complications.

Toxicity.—No ill effects have been observed as a result of the administration of proseptasine. We have administered up to 250 tablets (125 grm.) without signs of toxic symptoms.

Dosage.—The manufacturers recommended 2 tablets (1 grm.) three times daily. We were frankly sceptical as to the possibility of this amount being adequate to produce efficient bactericidal action in the blood. Double the dose produced no improvement in results, and we suggest that much larger doses are indicated.

Although a reduction of 5 per cent is not very striking, we feel that it is encouraging enough to advocate further investigation with very much increased dosage.

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post mortems in Calcutta only one showed rheumatic carditis.

Sir Leonard Rogers wrote (Rogers and Megaw, 1930):

> Deaths from circulatory diseases in the Indian series are just one half of those in the London one. The essential cause of this great difference is the remarkable absence of rheumatic fever and its sequelæ amongst Bengalis. In 4,800 Calcutta post mortems over 37 years only one death was returned as due to rheumatic endocarditis, and that was in an Anglo-Indian who had probably visited a cold climate; there was also only one case of possible rheumatic pericarditis. Moreover, among several thousand specimens in the Calcutta pathological museum accumulated during eighty years, there is only one described as rheumatic endocarditis in an Indian subject and that may well have been of pneumococcal or septic origin, as during twenty years' post-mortem experience the writer found all his endocarditis cases to be due to that class of organism. It may therefore be held that reliable evidence of the occurrence of rheumatic endocarditis in Bengal is still lacking. My collaborator, J. W. D. Megaw, agrees that rheumatic endocarditis is much less common than in Europe, but is by no means rare in Lucknow and not infrequent even in some places where the winter season is not really cold.

In 1923, I became interested in this question, soon after being appointed as physician to King George's Medical College Hospital. During the following years a large number of cases of mitral stenosis amongst young Indians, mostly with congestive heart failure, were admitted to the medical wards. It was the *cause* of this not uncommon mitral stenosis which first aroused my interest. In personal discussion with responsible teachers from medical colleges of all provinces of India and with Indian practitioners and civil surgeons of long experience, I found uniform agreement that undoubtedly definite clinical cases of acute and subacute rheumatic infection did occur in Indian children and young adults, and that the cause of mitral stenosis in India, as in England, was preceding rheumatic infection. Lieut.-Colonel Sandes, I.M.S., Professor of Medicine and Physician to Medical College Hospitals, Calcutta, told me he frequently recognized acute and subacute rheumatic infection in young Indians in his wards in Calcutta.

In 1930 one series of observations was published in this journal under the title of 'On rheumatic infection as a cause of mitral stenosis amongst young Indians'. In that article, I concluded, from my personal experience, in the following words :—

(1) Congestive heart failure following mitral stenosis is quite common in young Indians between 12 and 18 years of age in the United Provinces.
(2) Acute articular rheumatic fever does affect young Indian adults.
(3) Some acute rheumatic fever cases are observed to progress to peri- and endocarditis.
(4) Subacute rheumatic infection (carditis) was more difficult to recognize in Indian children even than in European children, for such children were not often brought to doctors and doctors have not been so far fully taught of its occurrence.
(5) Irreparable advanced mitral disease and congestive failure in young Indian adults is an index in India, as in England, of preceding unrecognized subacute rheumatic infection.
(6) Other possible causes of the common mitral stenosis in young Indian adults could be eliminated, *e.g.*, (1) congenital abnormality, (2) degenerative diseases, and (3) other infections (streptococcal, pneumococcal, scarlet fever, influenzal, syphilis, etc.).
(7) The impression amongst those who had considered the typical world distribution of rheumatic fever (*i.e.*, in cold damp climates) that rheumatic infection would not exist in warm climates such as India cannot be upheld.

Professor Hughes and Yusuf (1930) of the K. E. Medical College, Lahore, wrote 'Heart affections are met with much less often in tropical and in sub-tropical countries than in temperate regions, the difference being largely accounted for by the rarity of rheumatic fever in the former'. (Both these remarks may, in 1937, be considered open to revision.—H. S.) 'This fever probably varies in incidence and type in different parts of the tropics. In the textbooks of tropical medicine it receives little or no attention'. Hughes then describes 35 heart cases, 31 of which were treated in the Medical College Hospital, Lahore, between 1st October, 1929 and 30th April, 1930, being 5.6 *per cent of the total admissions* to those medical wards, all except one being a Punjabi Indian. Nineteen of the 31 cases had mitral stenosis. The Wassermann reaction was negative in all except two cases, with aortic regurgitation. In five mitral cases, subacute multiple arthritis existed and 13 gave a history of painful swollen joints. Gonorrhœa, syphilis and other specific causes were excluded as far as possible. Hughes concludes :

> In the Punjab, heart lesions, especially of the mitral valve, often bear an ætiological relationship to a disease in certain respects resembling rheumatic fever of temperate climates. This disease shows some or no heart signs with a strong tendency to relapse. Sometimes successive attacks of arthritis are followed by mitral lesion. Some patients complain of rheumatic attacks for years before they come under observation. Unhealthy tonsils occur. The joints swell and become tender, sometimes abruptly, with some fever (102° or 103°) and subside quickly after two or three days. More often the joints swell and subside slowly. Usually there is little fever. The ankles, knees and wrists are most often affected, usually with some stiffness. Response to salicylates was not marked. No cases of rheumatic nodules or of chorea were seen. In short, the mitral disease of these patients seems to be an infection generally subacute or chronic which like rheumatic fever affects the heart or the joints or both together. Whether it is actually a variety of rheumatic fever it is difficult to say, but it is obviously an analogous condition. Possibly it also caused the aortic disease in three cases in which the mitral valve also was affected.

In 1931 Professor Hughes and Yusuf became more definite. Referring to the 1930 series they then wrote :

> Attention was drawn to the fact that in the Punjab rheumatism is an important cause of valvular heart disease, especially of mitral stenosis. Although most frequent in pubescents and young adults it resembles the rheumatic fever of children in temperate climates. Acute arthritis is seldom seen, the disease usually taking a subacute or chronic course with a strong relapse tendency. In some cases with typical heart lesions the history indicates little or no involvement of the joints and in these the cardiac condition is

generally either discovered in routine examination or escapes detection until compensation fails. The joints most frequently attacked are the ankles, knees, wrists, elbows and small joints of the hands and feet but almost any joint may be affected. In the more acute cases especially, the disease first appears in one or two joints and spreads to others a day or so later. We have not seen rheumatic nodules or chorea in any patient, but tonsillitis is sometimes present.

Professor Hughes further now reports the occasional occurrence of uncomplicated aortic regurgitation of rheumatic origin. The two patients were 18 and 21 years old, with well-marked aortic incompetence who gave typical histories of rheumatism but showed no mitral lesion. They were admitted for lobar pneumonia and for malaria, respectively, and the aortic lesion was found only on routine examination. There were no complaints of cardiac trouble; blood pressure 115/40 and 132/50. Wassermann reaction was negative in both cases. The occurrence in India of uncomplicated aortic regurgitation of rheumatic origin was thus substantiated.

Lieut.-Colonel Hodge (1932), now Professor of Medicine, Medical College, Calcutta, reported fourteen selected cases of rheumatic fever from Bengal :

In Bengal the incidence of rheumatic fever is generally accepted. The writer has seen a number of unmistakable cases of rheumatic fever in Chittagong, Chinsura and Darjeeling and Jalpaiguri. Twelve cases are quoted—12 being in children 5 of whom showed chorea and 2 pre-choreic symptoms.

In 1932-33 some ten letters appeared in the correspondence columns of the *British Medical Journal* on 'Acute rheumatic infection in the tropics' (Clarke, Hughes, Keates, Mackinon, 1932), (Clarke, Ross, Dutton, 1933).

The evidence of those against its occurrence carried little weight. Three quotations are appended : (1) 'Twenty-five years of civil surgeoncy in the Punjab has convinced me that rheumatic infection is practically non-existent amongst the indigenous population. If rheumatic fever were at all prevalent, surely one would see cases of mitral disease. I have never seen a case of primary disease of the mitral valve. In 600 post mortems at Amritsar I never saw any evidence of inflammation of the mitral valve'. (2) Rheumatic fever is a disease of temperate climates, and even if I am incorrect in saying that none occurs in the tropics, it is so rare there that I have no doubt that some special difference between temperate climate countries and the tropics will ultimately give the clue which should help to elucidate the cause of this disease'. (3) 'A transfer to the tropics seems to be the best though unfortunately an expensive treatment for rheumatic fever'.

On the other hand, this correspondence produced letters of weight from Lieut.-Colonel Hughes, Professor of Medicine, Medical College, Lahore, and from Lieut.-Colonel Dutton, Principal and Professor of Medicine, Medical College, Patna.

Lieut.-Colonel Hughes wrote :

There is no doubt whatever that (1) at present rheumatic fever whilst not prevalent is by no means uncommon amongst the indigenous population of the Punjab, and (2) that mitral disease is a frequent result of rheumatic infection. I have heard from a colleague of two patients with rheumatic chorea in the same Indian family who both suffered from mitral disease as well. One died of cardiac failure. At the Medical College, Lahore, mitral disease occurred seven times (3.5 per cent) in 200 consecutive post mortems.

Lieut.-Colonel Dutton (1933) wrote :

Experience of my ward cases showed that typical acute rheumatism, rheumatic endocarditis and chorea were quite numerous during the cold weather and reacted to appropriate anti-rheumatic treatment. Many children were admitted with typical rheumatic endocarditis which developed into mitral stenosis, proved later by post mortem. A considerable number of chorea cases were admitted and cured by anti-rheumatic treatment only. There is no doubt whatever in my mind that acute rheumatism with endocarditis is almost as common in India during the cold weather in those provinces which have really cold weather as it is in any temperate climate.

From Vizagapatam, Madras, Kutumbiah (1935) and from Calcutta Banerjea (1935) have published their series of cases of rheumatic heart disease.

Gunewardene (1935) wrote in his *Heart Disease in the Tropics* : 'There is overwhelming clinical evidence that rheumatic fever and its complications are certainly met with in the tropics. In a paper read before the Ceylon Branch of the B. M. A. recently Dr. Cyril Fernando gave notes of 40 cases of rheumatic fever in Ceylon showing convincing evidence that all the usual sequelæ of rheumatic fever are met with in the tropics'.

In 1936 from Miraj in the Bombay Deccan, *i.e.*, well within the true tropics, Dr. Carruthers (1936) of the American Presbyterian Hospital published an excellent study of rheumatic heart disease in Bombay Deccan.

Dr. Carruthers reported his experience at Miraj, Bombay Deccan, and concluded that :

1. Rheumatic fever, as shown by the presence of rheumatic heart disease, although not as prevalent as in other parts of the world, nevertheless is a common condition (in Indians) in the Bombay Deccan.

2. The percentage of heart disease that is rheumatic in origin is greater than in other parts of the world.

3. There would seem to be a slight but definite tendency to a later age of onset in rheumatic heart disease in the Deccan (*see* table).

4. The percentage of rheumatic heart disease without clinical evidence of rheumatic fever is no greater in the Deccan than elsewhere.

Carruthers' 100 heart cases were admitted over the two years 1933-35 forming 5.08 per cent of the total 1,967 medical admissions. Forty-seven per cent of the heart cases were of rheumatic origin, forming 2.44 per cent of the medical admissions.

In Kutumbiah's Vizagapatam series 43.2 per cent and in Banerjea's Calcutta series 33.2 per cent resulted from rheumatic disease.

The age of onset is shown in the following table compared with Cabot's (1926) English figures :

	Bombay Deccan	England
Below 10 years ..	8 or 17.02 per cent	45 or 18.8 per cent
" 20 " ..	21 or 44.68 "	125 or 52.3 "
" 30 " ..	31 or 68.68 "	172 or 72.1 "

Of the 47 Deccan cases, 36 were males and 11 females. The table shows a slight but definite tendency to a later age of onset in India*. Of the Deccan cases, 74 per cent gave a definite history of joint pains and 25 per cent showed these in hospital. There were no choreas. Thirteen or 27 per cent died in hospital, and 7 had autopsies, six showing mitral sclerosis.

Finally in 1937 a note on the prevalence of acute rheumatic infection and of rheumatic endocarditis in India was required for the Office International d'Hygiene Publique to be held in Paris in October 1937, to which end the Public Health Commissioner with the Government of India sought information from the provincial directors of public health and from the annual provincial hospital returns. I had the privilege of reading their replies, which may be thus summarized : Official public health opinion holds that acute rheumatic fever and acute rheumatic endocarditis do exist all over India, but the official opinion is still, with few exceptions, that the prevalence of rheumatic fever is 'rare' or 'very rare'. The medical returns from civil hospitals under 'acute rheumatism' are utterly unreliable since fibrositis, myalgias, arthritis, and many joint conditions, both acute and chronic, are all almost invariably dumped indiscriminately under this head.

2. *Incidence.*—The comparative prevalence of rheumatic fever between India as a whole and other countries, *e.g.*, England, and between the different provinces and areas of India in the present absence of sufficient diagnosis and of accurate statistics is naturally difficult to determine. Statistics for admission to the hospitals of medical colleges and schools and statistics of post mortems done in such teaching institutions would provide the most reliable figures available. Allowance however must be made for 'tropical diseases' which swell the 'total' medical admissions for all causes, and the 'total' post mortems performed in India and which do not of course influence European statistics. The rheumatic fever admission rate and the post-mortem rate in India would be greatly reduced as compared with those of non-tropical countries.

About one-tenth of the total medical admissions and about one-third of total post mortems performed in medical college hospitals are for 'tropical diseases'. Even with this allowance, the figures indicating the true rheumatic fever prevalence rate would be higher in India than those returned because it is only comparatively recently that the not uncommon prevalence of rheumatic fever in India is being recognized, and also because subacute rheumatic infection in Indian children is so difficult of diagnosis. After all it is only comparatively recently in England that the diagnosis and frequency of early rheumatic infection has been clarified.

3. *The stenosis rate or the mitral rate as an index of rheumatic infection.*—The admission rate for acute rheumatic fever or for acute rheumatic carditis (endo- or peri-, or both) only is often regarded as an index of the prevalence of rheumatic infection. But a more reliable index and one easier to obtain is the hospital admission rate for mitral stenosis or for primary mitral disease (stenosis and regurgitation) or for heart failure from mitral disease specially under 30 or 40 years of age. From such primary-mitral-disease figures all cases secondary to aortic disease and degenerative arteriosclerotic lesions, or to myocardial degeneration are of course excluded. At least 95 per cent of all cases of mitral stenosis and probably also of primary mitral disease under 30 or 40 years are due to preceding rheumatic infection. Mitral stenosis or primary mitral disease as an index of preceding acute rheumatic infection is just as reliable an index, or even more so, than is the spleen rate as an index of preceding acute malaria. Such a standard might be well known as the 'stenosis' or mitral rate of rheumatic infection. Such a 'stenosis' rate would include cases due not only to acute but also to subacute and to chronic preceding rheumatic infection.

The post-mortem diagnosis of mitral stenosis, or of mitral valve disease, under 30 or 40 years of age, at the various medical schools of India is reliable, and the proportion of mitral stenosis cases furnishes some rough guide as to the frequency and distribution of acute rheumatic infection. From such statistics those due to tropical diseases have to be excluded when dealing with countries in which tropical diseases are not endemic.

4. *The post-mortem incidence of acute rheumatic infection and of mitral disease post mortems at Indian Medical Colleges.*—In 1925, I collected and analysed the statistics of 10,937 post mortems performed up to that date in the medical colleges of India (Calcutta, Patna, Lucknow, Lahore, Bombay and Madras), with Rangoon and Colombo as representatives of the Near East, also of 4,130 post mortems from the medical colleges of Hong Kong, Singapore and Java as representatives of the Far East, and compared them with 4,378 post mortems from Guy's and St. Mary's Hospital, London, whilst Dr. Ophuls, Professor of Pathology at Stanford University, San Francisco, U. S. A., was good enough to provide me with a comparative series of 3,912 post mortems to indicate the American

*This difference would not satisfy any statistician.—EDITOR, *I. M. G.*

figures. From the Indian and 'Far East' American figures all tropical diseases, *i.e.*, diseases not occurring in England, were excluded, so that some rough comparison becomes possible as to the incidence of rheumatic and of primary mitral diseases amongst these 19,659 post mortems.

The following table indicates that rough comparison but obviously too great importance should not be attached to such figures. They can only serve as some basis for further investigation and discussion.

TABLE I

Post-mortem diagnosis	In the Medical Colleges of				Totals
	India, Rangoon and Colombo	Hong Kong, Singapore and Java	London: Guy's and St. Mary's	San Francisco, U. S. A.	
1. Rheumatic endocarditis	60	0	119	1	180
2. Mitral disease	145	25	43	56	269
3. (1 + 2)	205	25	162	57	449
4. Total post mortems less 'tropical' ..	8,568	3,027	4,378	3,786	19,759
5. Percentage of 3 to 4	2	0.8	4	2	2.2

So far as the figures go, it would appear that post mortems are done for rheumatic endocarditis and for mitral disease combined twice as frequently in London as in India and San Francisco, and four times as frequently as in Hong Kong, Singapore, and Java. There are however obvious fallacies: (1) Although tropical diseases are excluded from the Indian series, yet undoubtedly such diseases do influence the post-mortem figures, *e.g.*, many cases are diagnosed post mortem as anæmia (which is not excluded) and many of these are probably due to ancylostomiasis and to malaria. (2) The post-mortem figures are previous to 1925; since then in India the knowledge of the prevalence of rheumatic fever has increased and this knowledge may to some extent influence the diagnosis frequency. (3) Whereas most cases dying in hospital are post-mortemed in London, only 'unclaimed' bodies can be post-mortemed in most medical colleges in India. However, these figures are the most accurate at present available.

If the percentage frequency of rheumatic and of mitral disease combined is worked out on the post mortems performed for deaths from circulatory system disease (which probably gives a true figure) the percentage for India and for London is precisely similar (*vide* Table II).

Moreover, if the figures for India and for London be further analysed (*see* table III), the interesting inference can be drawn that in India possibly preference is given to diagnosis of 'mitral disease' rather than to one which more definitely indicates the probable causative factor.

The American figures of post mortems for circulatory disease include a very large number due to arteriosclerosis, atheroma and other degenerative cardiovascular diseases, so that a comparison becomes difficult.

It is of some interest to examine the relative frequency of rheumatic and mitral post mortems at the different Indian medical colleges. These are set out below (table IV), but here again too much reliance should not be placed on

TABLE II

Post-mortem diagnosis	In the Medical Colleges of				Totals
	India, Rangoon and Colombo	Hong Kong, Singapore and Java	London: Guy's and St. Mary's	San Francisco, U. S. A.	
1. Rheumatic endocarditis	60	0	119	1	180
2. Mitral disease	145	25	43	56	269
3. (1 + 2)	205	25	162	57	449
4. Total post mortems 'circulatory diseases' ..	807	49	631	1,255	2,742
5. Percentage of 3 to 4	25	50	25	2	14

TABLE III

Post-mortem diagnosis	India, Rangoon and Colombo		London: Guy's and St. Mary's	
	Actuals	Percentage of circulatory post mortems	Actuals	Percentage of circulatory post mortems
1. 'Rheumatic' mitral disease	60	7	119	18
2. Mitral disease	145	18	43	7

deductions drawn from them. Further investigation is required.

As judged by these post-mortem figures, the order of frequency of post mortems for combined rheumatic endocarditis and mitral disease is

TABLE IV

Post-mortem diagnosis	In the Medical Colleges of								Totals
	Calcutta	Patna	Lucknow	Lahore	Bombay	Madras	Rangoon	Colombo	
1. Rheumatic endocarditis ..	0	0	1	0	25	9	2	23	60
2. Mitral valve disease ..	10	0	7	14	22	25	12	0 (?)	90
3. (1 + 2)	10	0	8	14	47	34	14	23	150
4. Circulatory post mortems	112	1	18	27	176	319	84	70	807
5. Percentage of 3 to 4 ..	9	0	44	50	27	11	17	33	26
6. Total post mortems (excluding 'tropical').	1,052	137	145	303	2,498	2,702	1,114	617	8,568
7. Percentage of 3 to 6 ..	1	0	5	5	2	1	1	4	2

low-lying areas near the sea coast or rivers, especially where liable to floods, and during heavy rains, *e.g.*, in the hills during the monsoons when rain falls and the diurnal range of temperature is maximal. Damp clayey soils for obvious reasons also predispose. Dry hot areas (*e.g.*, near the Sind desert) may be expected to furnish a low incidence. More direct predisposing conditions are pharyngeal catarrhs, recurrent tonsillitis and exposure to chill by permitting the causative germ of rheumatic infection to enter the system through this portal. Adequate preventive measures are thus indicated : good dry warm housing, climates, soils and clothing, good food and physique, a healthy naso-pharynx and a sufficient income. On theoretical grounds some believed that rheumatic infection would be rare in the tropics because heat and its attendant perspiration are preventive and curative. A well-recognized ætiological factor is damp cold *plus* a draught which produces a 'chill'. Dry cold is not a usual precursor unless there be a chill of exceptional severity. But in India sudden chills after free perspiration are very common. A great draught in conditions of great humid heat and great perspiration, but with little fall in general atmospheric temperature, will produce an individual chill as readily as a lesser draught with lesser heat and perspiration with a considerable general climatic fall in temperature.

(1) Lahore, (2) Lucknow, (3) Colombo, (4) Bombay, (5) Rangoon, (6) Madras, and (7) Calcutta. If the Guy's and St. Mary's London figures (25 per cent + 4 per cent) were added they would be placed fourth or fifth after Colombo or Bombay.

5. *The importance of rheumatic infection.*—This lies not in the initial attack, nor in the relapses, but in the gradual progressive fibrosis of the mitral valve which so often follows and which kills the young Indian patient in his late teens or early twenties. Three questions of practical importance arise : What are the ætiological factors ? Why are more cases of rheumatic infection not diagnosed ? What steps are required to improve the diagnosis ?

6. *Ætiological factors in acute rheumatic infection.*—The available figures are not yet sufficiently scientific nor numerous in India to decide with reference to such ætiological factors as sex, caste or area of frequency. There seems little doubt however that as in Europe the incidence falls far more heavily on the poor and especially on those ill-nourished and debilitated by previous disease and infection. Moreover, the incidence may be expected to be greater (as elsewhere throughout the world) in cold and damp seasons, areas and dwellings, *e.g.*, in

7. *Why are more cases of rheumatic infection not diagnosed ?*—Some reasons why more

cases are not diagnosed in Indian children are: (1) The existence and prevalence of rheumatic fever in India have only recently been generally recognized, and in fact is even now questioned and at times denied by some outside medical teaching colleges and schools. Even now, most authorities speak of it as 'rare' or 'very rare'. (2) The general unwillingness of Indian families to bring their children or young adults to hospital and to leave them in hospital without relatives, and that for an apparently trivial illness which in its subacute form displays only moderate fever with limb or joint pain and not those with striking and dramatic symptoms which are typical of some diseases with which Indian families are readily familiar, *e.g.*, cholera, tetanus, rabies, etc. Parents are not alive to the serious cardiac conditions which may not infrequently arise. (3) The want of special accommodation and of special physicians for children's diseases in India, *e.g.*, the comparative absence of children's beds, of children's wards, of children's hospitals, and of special out-patients for children with physicians specializing only in children's diseases, not only in the hospitals attached to medical colleges and schools (where they are essential) but also in time in all civil, Dufferin and private hospitals. (4) The diagnostic difficulty. Moderate fever for a week or two even with severe joint, limb or heart pain is not recognized as serious in a country where severe fever often with severe febrile pains is of daily occurrence. (5) The want of better teaching by children's specialists in Indian medical colleges and schools, both under-graduate and post-graduate. Hence, the present generation of medical men, whether of degree or licentiate standard, is not as fully trained as they should be in the diseases of children.

8. *Steps required to improve diagnosis* may be mainly summed up within the two words, education and provision, *e.g.*—(1) Information to medical officers in Government service, by circulars from the administrative medical officers and through their post-graduate classes, and to private practitioners, by articles to the medical press and by papers read before medical associations and societies. (2) Special attention by the school medical service and medical inspection of school children. (3) Teaching by specialists in the diseases of children to all medical students in their curriculum as now laid down by the Medical Council of India. (4) A definite quota of questions on children's diseases in all parts of the appropriate qualifying examination papers and *viva voce*, *e.g.*, in medicine, pathology, hygiene, obstetrics and surgery. (5) Instruction to nurses, preparing for the Nursing Certificate examinations of the provincial State Medical Boards. (6) The general public information concerning the occurrence and prevalence of rheumatic infection and of its sequelæ, through the public health services, the Red Cross, women and child welfare centres, with their health visitors and midwives, with lectures to the educated public and especially to mothers through such agencies. (7) By providing special children's hospitals, wards, beds and out-patients in all medical colleges and medical school hospitals and as soon as possible in all hospitals and dispensaries. (8) A revised and more accurate nomenclature in diagnosis and the annual hospital returns, *e.g.*, not 'acute rheumatism', which might reasonably include acute lumbago or acute sciatica, but 'acute rheumatic' infection, to include acute rheumatic fever (polysynovitis), acute rheumatic carditis (peri- and endo-), rheumatic chorea, and nodules and chronic rheumatic mitral disease.

Summary

1. Recent views concerning the occurrence of acute and chronic mitral rheumatic infection in India are reviewed.

2. The experience of teaching physicians attached to medical college hospitals that such infection most certainly occurs in all its forms and is not uncommon is noted.

3. In comparing the incidence of rheumatic infection in India and Europe, and throughout India, the most reliable standards would be (*a*) the hospital admission, (*b*) post-mortem rates of the Indian medical colleges and schools, and (*c*) the clinical experience of their attached physicians.

4. Allowance must however be made for tropical diseases which affect the Indian as compared with European admission and post-mortem rates.

5. A stenosis rate or mitral rate is suggested as a more reliable index for the incidence of rheumatic infection than an admission rate for acute rheumatic polyserositis or carditis.

6. Amongst 10,937 post mortems from the medical college hospitals of India the incidence for acute and chronic mitral endocarditis combined was 2.2 per cent of the total post mortems and 14 per cent of the post mortems for total circulatory diseases.

7. If the post-mortem rate for rheumatic mitral (7 per cent) disease and for primary mitral disease (cause unspecified) (18 per cent) be added together the same figure is reached for India as in London post mortems (18 per cent for rheumatic and 7 per cent for unspecified). It would therefore appear in India that primary mitral disease (of young adults) is not so far recognized as being mainly due to rheumatic infection.

8. The relative order for acute rheumatic infection and for mitral disease as judged by those 1925 figures at the Indian medical colleges is (1) Lahore, (2) Lucknow, (3) Colombo, (4) Bombay, (5) Rangoon, (6) Madras, and (7) Calcutta. But future investigation will make large differences in these figures.

9. The importance and ætiology of rheumatic infection is briefly referred to.

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POST-ARSPHENAMINE BLOOD DYSCRASIAS

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BLOOD dyscrasias as a result of modern anti-syphilitic treatment are relatively rare and grave complications. The literature on the subject is scanty. Folley and Moore claim to have described the first case in the American literature. McCarthy, Loveman and Wilson have recorded seventy cases in their special study on the subject. In general, blood dyscrasias following the administration of arsphenamines are characterized by the symptomatic and pathological expression of depressed bone-marrow function, the type of reaction depending upon the type of injury and the particular elements affected. Piney's (1931) hæmatological studies have confirmed the fact that a condition resembling agranulocytosis produced by benzol poisoning is rarely caused by salvarsan and similar substances. The following figures are quoted by Moore (1933) to show the relative frequency with which each preparation of arsenic produces this group of toxic reaction :—

Arsphenamine	..	10 cases
Neo-arsphenamine	..	43 „
Sulpharsphenamine	..	14 „
Silver-arsphenamine	..	1 „

Aubertin, Blancstein and Lehmann have observed two cases of agranulocytosis in syphilitic patients after treatment with acetylarsan. These patients according to their report had also severe anæmia. No cases have been so far reported with tryparsamide. Though the above figures show that the neo-arsphenamine group produces the greatest number of blood reactions, it is considered by the Council of Pharmacy and Chemistry of the American Medical Association that sulpharsphenamine is the worst of the offending agents.

Clinically four different types of reactions are described :—

1. Symptomatic purpura.
2. Thrombocytopœnic purpura.
3. Agranulocytosis.
4. Aplastic anæmia.

There may occur a combination of these syndromes. The frequency of each type is shown by the following figures in the reported cases :—

Thrombocytopœnia	..	12 cases
Thrombocytopœnia with agranulocytosis	..	7 „
Agranulocytosis	..	15 „
Aplastic anæmia	..	36 „

This depends upon the degree of damage to the thromboplastic, leucoblastic and erythroblastic tissues, respectively. In the severe type of aplastic anæmia the hæmopoietic activity of the bone marrow is completely suppressed and all the formed elements are thereby involved.

The clinical picture of thrombocytopœnia is essentially one of purpuric hæmorrhages from the skin, mucous membrane and viscera. Blood platelets are markedly diminished. The other elements are usually not affected. Recovery is said to be the rule.

In the other type of cases with granulocytopœnia, fever, and soreness of the throat, with or without necrosing ulcerations of the bucco-pharyngeal tissues, are the usual signs. A blood count reveals the marked fall of leucocytes caused by an absolute granulocytopœnia. Very low figures down to 136 per c.mm. are reported. Other septic complications may set in with lethal termination. In cases associated with platelet deficiency purpuric hæmorrhages and visceral hæmorrhages may complicate the picture. Usually the anæmia associated with this group of disorder is slight and non-progressive. The gravest of all the types is aplastic anæmia. Progressive anæmia is evident from the onset in addition to thrombocytopœnia and agranulocytic symptoms. The course is downhill. Blood shows a progressive and often rapid

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10. Some steps required to improve the recognition of the prevalence of rheumatic infection are detailed.

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