

Clinical cases of diphtheria in these parts of the country are almost unknown among Indians; the writer saw only one case of diphtheria during the five years of his stay in Nainital district. It is not, of course, suggested that this was the only case that occurred from the disease during that period. It does appear, however, that the probable explanation of the phenomenon is that sub-clinical infections of the disease are quite common.

Frost (1928) while discussing the epidemiology of diphtheria stated that ratios of infection to immunity and of infection to disease vary widely, in the same population, with age, race, circumstances of known exposure, and, in different populations, with difference in latitude. Certain unpublished studies made by the writer on acute poliomyelitis, a disease epidemiologically very similar to diphtheria, seemed to suggest that these ratios may vary in the course of the same epidemic. Statistics are available on the results of Schick tests done in India on Indians by Rambo (1929) and on Europeans and Anglo-Indians by Fox, McDonald and McCombie Young (1923). These are given below :

RAMBO'S STATISTICS		FOX'S <i>et al.</i> STATISTICS	
Age group	Per cent positive	Age group	Per cent positive
Under 3 years	72.2	1-5 years	54.5
3-6	10.7	5-10 "	51.5
7-10	14.0	10-15 "	41.6
11-14	2.6	Over 20 "	66.6

These statistics indicate that, age for age, the Anglo-Indians and Europeans have a higher susceptibility to diphtheria than Indians. The results are similar to those obtained in the U. S. A. where the white population have at corresponding ages a greater susceptibility than the coloured.

There were only four pseudo-reactions noticed by the writer among Schick tests made. Rambo had 2 such among 197 cases, whereas Fox *et al.* had 12 pseudo-reactions among 271 tests done on Anglo-Indians and Europeans. These reactions have been ascribed to local anaphylactic response to the protein of autolysed diphtheria bacilli in the broth. The statistics at our disposal are inadequate, but if the figures indicate a general law regarding racial variation, some explanation will have to be sought for the phenomenon. The question will then be: How do the Europeans and Anglo-Indians get sensitized to the diphtheritic protein to a greater degree than do the Indians?

#### Summary

1. There are reasons to believe that diphtheria is endemic in India.

2. The morbidity from diphtheria is, for some obscure reason, not high among Indians.

3. The susceptibility to diphtheria rapidly decreases with advance of age in this country, in spite of the fact that as a disease it is not so prevalent. This suggests that infections incapable of producing clinical manifestation of disease are common.

4. More extensive Schick-testing among various races in India are required to elucidate certain important phenomena of the disease.

#### Acknowledgments

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## A Mirror of Hospital Practice

### TOXIC EFFECTS OF EMETINE ON THE CARDIOVASCULAR SYSTEM

By R. N. CHOPRA, C.I.E., M.A., M.D. (Cantab.)  
LIEUTENANT-COLONEL, I.M.S.

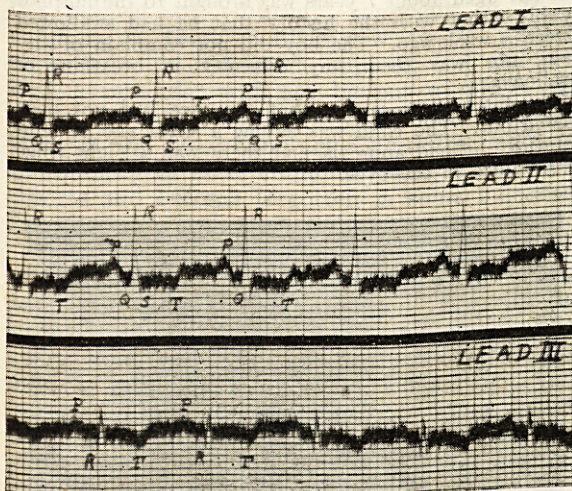
and

B. SEN, B.Sc., M.B.

(Carmichael Hospital for Tropical Diseases, School of Tropical Medicine, Calcutta)

S. D., a European female, 48 years old, was admitted into the Carmichael Hospital for Tropical Diseases, under the senior author on 23rd January, 1934, with irritation of the skin all over the body and a general feeling of weakness, depression and debility. The patient suffered from malaria five years ago and was treated with quinine. A year later she had an attack of acute amoebic dysentery and was treated at the School of Tropical Medicine, London, where a large number of *Entamoeba histolytica* were found in her

stools. The treatment consisted of a course of emetine-bismuth-iodide by the mouth lasting for 12 to 14 days, the drug being taken every night in gelatine capsules. The first two doses were vomited, and after six doses of the drug, the patient's pulse became rapid, she felt extremely weak and was consequently kept strictly confined to bed for a week. The symptoms of dysentery disappeared, but the rapid pulse and general weakness persisted, at the time of her discharge from the hospital. She regained her normal health in about a month. The patient returned to India and during the hot weather of 1933 had another attack of malarial fever and was treated with quinine. Soon after this she started passing blood and mucus with her stools, and on examination these showed *E. histolytica*. She was put on emetine, one injection daily for the first six days and after that one every alternate day till a total of nine injections had been given. After the ninth injection, although the symptoms of dysentery disappeared and examination of the stools showed no *E. histolytica*, the patient's pulse became very rapid, she felt very weak physically, was mentally depressed, and had an itching sensation on the skin all over the body which was very uncomfortable. The patient was kept in bed and put on a mixture containing digitalis which she took for about three weeks. The rapid pulse and the general depression however persisted, she could not undertake any physical exertion, and she lost weight. She came to the Carmichael Hospital for Tropical Diseases for investigation and treatment.



The patient on admission was thin, anæmic and debilitated, and complained of a feeling of exhaustion. Physical examination revealed a rapid pulse (90 to 100 p.m.) of low tension, the heart sounds were weak and flabby, and the blood pressure was 106 m.m. systolic and 70 m.m. diastolic. The urine showed no albumin or any other abnormal constituents. There was a good deal of itching of the skin, but no patches of urticaria were visible. Repeated examinations of her stool showed neither protozoa nor any non-lactose-fermenting bacteria. No helminthic ova were found and no malarial parasites could be detected in the peripheral blood; culture for the plasmodium was also negative. Electrocardiographic examination was made soon after admission with the following results:—

P-R interval—0.10 secs.

P-T interval (systole)—0.35 secs.

T-P interval (diastole)—0.24 secs.

Heart rate—104 per minute, regular.

Potential of R wave—1.2 multivolts.

Inversion of T wave in second lead with T-P interval (diastole) less than P-T interval (systole) indicates myocardial damage with tachycardia of sinus origin.

The interesting points about this case are the toxic effects produced by emetine on two occasions when the drug was administered for intestinal amebiasis. On both occasions the main effects were on the cardiovascular system. The author and his co-workers (1924) showed that marked histological changes of a degenerative character occur in the heart of rabbits after injections of emetine. In this patient the indications of myocardial changes were clearly demonstrated by electrocardiographic tracings. Emetine also has a damaging action on the capillaries, and the senior author has seen several cases in which itching of the skin and even urticaria and large pruriginous plaques were produced and persisted for a month, after a course of emetine. The patient improved with cardiac tonics (iron, arsenic and strychnine), and an electrocardiogram taken three weeks later showed a distinct improvement.

#### REFERENCE

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### A CASE OF CEREBRO-SPINAL FEVER SUCCESSFULLY TREATED BY INTRAVENOUS INJECTIONS OF UROTROPINE

By B. L. CHOPRA, L.R.C.P., L.R.C.S., L.M., D.P.H.  
D.T.M. (Liverpool)

District Medical Officer, North Western Railway  
Delhi

A MAN, 35 years of age, was admitted into this hospital about the first week of December 1933 with a temperature of 103°F. He was examined and found to have Kernig's sign and all other clinical signs of cerebro-spinal fever.

A lumbar puncture was done and about 30 c.cm. of turbid fluid, flowing at slightly increased pressure, were drawn off. This was examined bacteriologically and showed meningococci in abundance. Next day another lumbar puncture was done, 30 c.cm. of fluid drawn off and 20 c.cm. of anti-meningococcal serum injected intrathecally. Next day another lumbar puncture was done, and the same amount of fluid was drawn off, and 20 c.cm. of anti-meningococcal serum was injected. As the serum was not having much effect on the temperature and general condition of the patient, daily intravenous injections of urotropine (5 c.cm. of 40 per cent solution) were given. The first injection brought the temperature down and it remained normal throughout the remaining period of his stay in hospital, except on one or two occasions when it was about 99°F.

Twelve intravenous injections of hexamine were given in all. Each injection seemed to improve his condition. Whenever he complained of headache and uneasy feeling a lumbar puncture was made and about 30 c.cm. of fluid removed each time. Five such punctures were made.

On the 31st December (25th day of disease) the patient complained of pain in his joints and looked slightly toxæmic with a temperature of 98.8°F. An intravenous saline (half a pint) was administered and after that day he made an uneventful recovery and