lower classes of society, were examined clinically and serologically to determine the incidence of syphilis.

2. There were forty-three children, or 3.9 per cent., giving positive serological reactions. Among these were thirteen whose positive reactions were almost surely due to yaws, leaving thirty, or 2.7 per cent., with probable congenital syphilis.

3. It is estimated that congenital syphilis is present in about 3 per cent. of all black and coloured children of Kingston.

4. Syphilis doubtless reaches a much higher incidence among the entire population. The exact figure cannot be stated, but on the basis of evidence gathered elsewhere concerning the ratio of the incidence of syphilis in children to that for the entire population it may be said to be approximately 12 per cent.

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EPIDEMIOLOGY OF EPIDEMIC DROPSY*

BY

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Epidemic dropsy belongs to the group of obscure diseases, like tularaemia, which are little known outside their endemic homes. None the less this disease is of much interest to students of tropical medicine, and particularly to those of us who work in this part of the world. It is by no means a minor problem in the provinces of Assam, Bengal, Bihar, and Orissa. One would, perhaps, not be far wrong in saying that a practitioner in Calcutta comes across epidemic dropsy as frequently as malaria, if not more frequently. The city, according to Mazumdar (1933), recorded deaths from epidemic dropsy every year between 1905 and 1931. Widespread epidemics involving thousands of citizens and causing heavy mortality occasionally occur. The epidemics in 1909, 1926, and 1927 were particularly disastrous, when 433, 939, and 636 persons respectively are said to have died. Ten times this number must have been the victims of more or less permanent damage to the heart. The disease is not confined to the towns. Outbreaks occur even in the smallest villages, and at times large tracts of country in the eastern provinces of Assam, Bengal, Orissa, and Bihar become involved. Outside these provinces localized outbreaks have been reported from the eastern districts of United Provinces and parts of Madras Presidency. A few epidemics have occurred in Burma, Fiji, Mauritius, and certain other parts of the world, but the disease has been practically confined to emigrants from those provinces.

* A more detailed description of these studies will appear in a set of five papers in the July, 1937, issue of the *Indian Journal of Medical Research*.

Earlier Studies

The history of the disease carries us as far back as reliable records are available. Chambers (1880) believed that the disease had been prevalent since the famine of 1866. Good descriptions of epidemics occurring in the last quarter of the nineteenth century and since then are given by O'Brien (1879), Caley (1878), Payne (1879), McConnell (1879), Crombie (1879), Deakin (1880), Kastagir (1880), McLeod (1893), Rogers (1902), Munro (1908), Cambell (1908), Greig (1911), and many others. How these epidemics arise, what factors favour their continuance and decline, how and why the disease prevalence is maintained at a low endemic level in certain localities, why certain communities suffer more than others, what determines the sex, age, and occupational distributions, what preventive measures can be taken, are some of the problems which have so far defied solution. In epidemic dropsy, as in many other diseases, bad weather, personal diathesis, water, inadequate or unsuitable food, unknown toxic substances, various types of organisms, and even insects have received their share of blame at the hands of various workers. Yet the lack of exact knowledge cannot be said to be due to want of interest on the part of the profession. Among others epidemic dropsy has engaged the serious attention of eminent workers like Rogers (1902), Lukis (1908), Greig (1912), Megaw (1927), Acton and Chopra (1925), and many important contributions have been made. However, in the absence of exact knowledge every practitioner has perhaps either formulated his own theory or has adopted one advanced by others. We cannot do better than sum up the present position in the words of the editor of the *Indian Medical Gazette* (1935) when he says: "Many theories regarding the cause of the disease have been formulated, have lived their day and become history, and have been revived again." However, from amongst a multitude of theories three have received considerable support. These are: (1) rice theory, (2) contagion theory, and (3) mustard-oil theory. We propose to revive one of these theories and present fresh evidence in support of it, in the hope that it will stand critical examination and will not be cast away again into limbo.

Observations in the Field

These three theories formed our working hypotheses, and it was mainly on these premises that we proceeded to collect exact information in the field. Investigations were made in seven different localities where epidemic dropsy was raging. These included rural, semi-rural, and industrial areas in Bihar, Bengal, Assam, and Orissa, thus representing different living conditions. House-to-house visits were made and important data were collected on printed schedules specially designed for mechanical tabulation. In three localities data were collected for every individual and for each family, whether stricken or not. In other places investigations were confined to the affected families and to such unaffected families as were of special interest. Reasonable precautions were taken to ensure the veracity of the information recorded. Detailed information was obtained from 964 patients and 2,581 healthy persons, comprising 310 affected families and 290 unaffected families. In addition, certain relevant data were collected from 1,727 unaffected families having 9,678 members. The main points that emerged after critical analysis were:

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(*a*) Morbidity rate varies greatly from place to place.

(*b*) The disease is almost exclusively confined to the Bengalees and to those who have adopted their way of living, especially in regard to food habits.

(*c*) Both sexes are equally liable to suffer.

(*d*) Epidemic dropsy claims its victims irrespective of their religious grouping, depending upon local circumstances.

(*e*) Higher castes among the Hindus are the worst sufferers and the menial classes usually escape.

(*f*) It is mainly a disease of the middle-class people.

(*g*) Age distribution is very striking; suckling babies do not suffer, the disease rarely occurs in babies under 3 years of age, and the morbidity rate is very low up to 5 years. Above this age group there is not much difference.

(*h*) Rice, as the principal article of diet of patients, can be excluded in rare instances only.

(*i*) The habit of discarding water in which the rice is boiled does not afford any protection against the disease, as would be expected if it were due to a water-soluble toxin. On the other hand, more cases are found among those who reject rice-water than among those who do not. This is probably linked up with economic status.

(*j*) Outbreaks of epidemic dropsy occur as frequently amongst people habitually using sun-dried and hand-pounded rice as among those taking parboiled and milled grains.

(*k*) There is no evidence of bad storage of rice as one of the factors associated with incidence of the disease nor is there any clustering of cases around a common stock of rice.

(*l*) The incidence of the disease and the consumption of rice grains having opacity are not found definitely associated.

(*m*) Not a single case is observed among those who deny the use of mustard oil as an article of food.

(*n*) Strong evidence has been obtained to associate a particular brand of mustard oil produced at a certain time with an outbreak of the disease at Jamshedpur.

(*p*) Cases develop more frequently among those who give history of contact with patients, particularly family contact, than among those who do not, but this factor is usually linked up with common messing.

(*q*) Congestion in houses is negatively correlated with the incidence of the disease (due regard having been paid to the population at risk). This factor is probably linked up with economic status.

Examination of Theories

We may now examine the three theories stated above in the light of these observations.

1. *Rice Theory*.—According to the rice theory in its present advanced form epidemic dropsy is caused by a water-soluble toxin which is ingested with rice. The toxin is elaborated inside the grain by certain members of the *B. vulgatus* group. Medium-grade rice, chiefly the variety locally known as "balam," is the most suitable pabulum. Infection takes place most readily in parboiled and polished rice, in which the natural protective enzymes are destroyed. Storage under warm and damp conditions is favourable for infection. Infected grains may be readily differentiated from the healthy ones by the central opacity, which can be easily seen with the naked eye, especially if the grains are steeped in water or glycerin. This is at present the officially accepted theory.

It will be readily seen that the facts as presented above under items (*e*), (*f*), (*g*), (*i*), (*k*), and (*l*) are not in accord with the rice theory. Special attention in this connexion may be drawn to item (*i*). Besides, we were unable to produce the central opacity in rice grains by storing the proper kind of rice under conditions which are believed to favour its development. Another objection to the rice theory is that, while rice forms the staple food in many parts of India and in other countries, the geographical distribution of epidemic dropsy is very restricted. Samples showing large proportions (more than 90 per cent.) of typical centrally opaque grains were obtained from certain districts of Madras Presidency, where epidemic dropsy was unknown, and similar grains were also obtained from paddy gathered directly from the fields. Moreover, we were unable to demonstrate the presence of spore-bearing aerobes in the opaque part of the grain when surface sterilization had been efficiently carried out. The rice theory therefore failed to afford explanation of the observed facts. As we shall see later, direct experiments on human subjects also pointed towards the same conclusion.

2. *Contagion Theory*.—It has been vaguely stated that the disease spreads from person to person by close personal contact. An influential section of the profession supports this theory. No definite views have been advanced with regard to the nature of the organism, the portal of discharge, the transmitting agent, or the portal of entry. Item (*q*) in the above summary threw doubt on the validity of this theory. It also fails to give adequate explanation of items (*b*), (*e*), (*f*), (*g*), and (*m*). It has been observed by many workers as also by us that introduction of patients in advanced stages of the disease into unaffected families is in many instances not followed by the development of secondary cases, even though conditions are very favourable for transmission of disease from person to person through droplets or contact. To test further the validity of this theory an experiment was conducted in a semi-isolated community.

Forty-six young men lived in a barrack side by side and worked by shifts as warders in a local jail. According to messing arrangements they were divided into five unequal groups. In one of these groups five out of seven went down with epidemic dropsy within a week. The servant who cooked for them was also a victim. Members of this group, as also of the other groups, occupied beds next to one another. This arrangement was altered so that the patients now occupied seats in between the healthy persons. The experiment continued for about four weeks, but no fresh cases developed.

It would thus appear that common food rather than personal contact was the factor responsible for the incidence of cases in this community. As it happened the affected group was the only one which purchased mustard oil from the bazaar. All the other warders obtained their supply from the jail.

3. *Mustard-oil Theory*.—This also is ill defined inasmuch as no definite views have been advanced with regard to the nature or the source of the substance in the oil which causes symptoms. The theory is popular among the lay people, in whom malice against the Marwaris (who sell mustard oil but do not themselves take it) plays an important part, because they never contract the disease. Adulteration with cheaper grades of oil, or even with mineral oil, is suspected. This theory has very few adherents amongst responsible members of the profession.

There is a striking correspondence between the endemic area and the part of the country in which mustard oil is habitually used by the bulk of the population. This fact is highly suggestive. Further, even in this area the disease is, in our experience, strictly confined to the communities that are consumers of mustard oil; a minority of the population, mostly emigrants from other provinces, who use *ghee* (clarified butter) for cooking purposes, escape. Indeed all the facts enumerated in the summary given above fit in with the hypothesis that certain consignments of mustard oil are responsible for the causation of the disease. In this connexion the epidemic which occurred in October, 1936, at Jamshedpur is of special interest. Being a purely industrial town its population,

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which totals about 100,000, is very cosmopolitan. Jamshedpur had been free from epidemic dropsy for some years when, in October, 1936, the first case occurred. Within five weeks 222 cases were reported, involving sixty-six families. Of these fifty-two were Bengalee families living on the usual Bengalee diet. The non-Bengalee victims were also found to have taken to Bengalee diet; at any rate rice and mustard oil were used by them in considerable quantities. The most remarkable feature of this epidemic was that the affected families had a common source of mustard-oil supply. Eighteen of them obtained one particular brand of oil direct from the mill, of whom fourteen received it in original sealed containers and four loose, and thirty-five obtained it from the grocers. In ten instances the grocers stored this brand, but the customers had not specially asked for it. In the case of three families the evidence was not so clear. There were no cases among the large clientele of the other three mills which supplied mustard oil to the town. Another important point is that the cases only appeared amongst people who had purchased the oil during October and the first week of November. The suspected brand of oil had been quite popular for some time, and nothing untoward had happened previously. Thus the suggestion is that a particular consignment of oil of this brand was associated with the epidemic. It may be mentioned here that the manufacturers claimed that the oil was unadulterated and of good quality. The genuineness of the oil was confirmed by chemical analysis, kindly carried out for us by the Professor of Public Health Laboratory Practice on a sample collected from one of the affected families.

Thus we see that the field studies, laboratory investigations, and observations under controlled conditions so far described threw doubt on the validity of the "rice" and the "contagion" theories. They gave, however, a definite lead in favour of the mustard-oil hypothesis stated above. Since we were able to procure samples of oil in original containers from the affected families it was now possible to test this hypothesis by feeding human volunteers on the suspected oil under strictly controlled conditions.

Feeding Experiments on Human Volunteers

The results of these experiments are very striking. It is not proposed to go into the details of the experiments, but a few interesting facts are stated below.

Three experiments were performed, in each of which twelve healthy young subjects who were willing to take the risk and who were ready to give an undertaking that they would continue on the special diet for the required period, were selected. In the first two experiments the subjects were divided into four groups of three each, and the experiments were arranged as below:

Group A: "diseased" rice and "suspected" mustard oil.
Group B: "diseased" rice and jail-produced mustard oil.
Group C: clear rice and "suspected" mustard oil.
Group D: clear rice and jail-produced mustard oil.

The main difference in the two experiments was the source from which the "suspected" oil was obtained. The oil used in Experiment I was collected from an affected family in Assam, while that used in Experiment II was obtained from an affected family at Jamshedpur. No untoward symptoms developed in any volunteer in the first experiment, except in one of those taking suspected oil. He complained of loss of appetite and constipation. Later he developed a cold with slight rise of temperature, but was restored to normal condition after a few days of rest and milk diet. In the second experiment, on the fifth day after the commencement of the special diet, two persons in Group A and one in Group C missed their morning meal and all members of Groups A and C complained of pain in the joints and of symptoms of gastro-intestinal disturbance. Later some of them developed fever and three developed oedema of legs with flush and pitting on pressure, which are characteristic of epidemic dropsy. Two of the oedema cases belonged to Group A and one to Group C. The last-mentioned patient was taken out of consideration, because on closer examination he showed evidence of chronic filariasis. All the controls remained perfectly healthy and cheerful. That was the first occasion on which it had been possible to reproduce signs and symptoms of epidemic dropsy in human subjects. The value of these results is further enhanced on account of the history behind the suspected oil which was employed in this experiment.

However, it remained undecided whether oil alone could produce the condition or if it was necessary to combine "diseased" rice with it. A third experiment was therefore carried out in which "diseased" rice was excluded. The source of suspected mustard oil was the same as in the last experiment. The results of this experiment were very striking. After the same premonitory symptoms which were observed in Groups A and C of Experiment II well-marked oedema of the legs developed in all the subjects taking the suspected oil. In three cases there was dilatation of the heart. In one patient foetal type of rhythm and in another systolic bruit were heard; there were no symptoms referable to the nervous system. In short, in the judgement of competent physicians the signs and symptoms in these cases were identical with those found in mild cases of epidemic dropsy. The controls remained perfectly healthy.

Discussion

In the absence of exact knowledge of the causal agent epidemic dropsy can only be defined as a clinical entity. Descriptions of different observers vary to a certain extent. It is therefore possible that this condition, like so many other diseases, such as croup and the enteric fevers, may in course of time be split up into two or more distinct diseases on the basis of their aetiology, which may possibly be entirely different for these really distinct but allied clinical entities. The clinical feature on which we have mainly relied for distinguishing the condition which we have called epidemic dropsy from what has been described as "wet beri-beri" is the entire absence of signs of involvement of the nervous system. In this respect and in general symptomatology the cases met with in the course of field investigations and those developed under experimental conditions were exactly alike. At any rate the present position may, we think, be stated in the following words:

The clinical condition known as epidemic dropsy (or beri-beri by lay people), as commonly met with in endemic or epidemic form in Assam and Bengal and in parts of Bihar and Orissa and occasionally outside these boundaries, mostly among emigrants from these provinces, is caused by some unknown substance or substances which enter the system through the ingestion of food cooked in certain consignments of mustard oil. The oil may be genuine and unadulterated. The nature of the poisonous substance is not known. There is some evidence in favour of the view that the deleterious substance is not a normal constituent of mustard oil and that it is more likely to be a chemical poison rather than something in the nature of a living virus. However, inquiries are in progress to clarify this point and to develop, if possible,

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a test by which bad oil may be distinguished from the harmless one.

While the problem of the aetiology of epidemic dropsy cannot be said to have been completely solved, a definite advance has been made and a stage has been reached when preventive measures, based on scientifically proved facts, can be recommended, though their application is limited to a certain extent by the absence of knowledge about the nature and origin of the deleterious substance in the mustard oil. It should now be the duty of the health administrator to trace the oil which has caused the disease to its source as soon as he comes across cases of epidemic dropsy, and to prevent its further distribution. He should also prohibit its use for cooking purposes. The discarded oil may be used for oil baths, as it is probably harmless when applied externally. Besides, there are many other uses to which this oil can be put. The length of the incubation period or the lag phase—five days for premonitory symptoms and nine to twenty-three days for development of oedema, as noticed in our human experiments—should be of assistance in tracing the bad oil. By adopting the measures recommended above it should be possible to prevent the spread of the disease among those who have not yet partaken of the oil and to reduce the severity of the disease in those who have taken it in just sufficient quantities to cause premonitory symptoms.

Summary

1. A brief statement is given of the results of epidemiological studies in connexion with outbreaks of epidemic dropsy in seven localities in Bihar, Bengal, Assam, and Orissa, representing rural, semi-rural, urban, and industrial conditions.

2. An epidemiological experiment designed to test the contagiousness of the disease in a semi-isolated community is briefly described.

3. Three principal theories of the aetiology of the disease are discussed, and it is shown that the observed facts fail to support the "diseased-rice" theory and the "contagion" theory. A very satisfactory explanation of these facts is provided by the hypothesis that epidemic dropsy, as commonly seen in these provinces, is caused by a deleterious substance contained in certain consignments of mustard oil.

4. Experiments on human subjects, living under strictly controlled conditions, are described. They show that signs and symptoms of epidemic dropsy are produced in healthy young men by giving them food cooked in a brand of mustard oil which is strongly suspected, on epidemiological grounds, of being responsible for an epidemic—the "diseased" rice having been altogether excluded from the experimental food.

5. Practical application of these findings is discussed.

Acknowledgements

We gratefully acknowledge the help and co-operation which we received from our colleagues in the Calcutta School of Tropical Medicine, and the All-India Institute of Hygiene and Public Health, and from the officers of the Jail Department. In this connexion the names of Brevet Colonel R. N. Chopra, C.I.E., K.H.P., I.M.S., Major-General D. P. Goil, K.H.P., I.M.S., Lieut.-Colonel Vere Hodge, I.M.S., Dr. R. N. Chowdhury, Lieut.-Colonel M. A. Singh, I.M.S., Major S. L. Patney, I.M.S., and Lieut.-Colonel M. Das, I.M.S., deserve special mention. We are thankful to the Indian Research Fund Association for financing the inquiry. We also wish to convey our thanks to the official and non-official agencies which extended their co-operation in the course of field investigations.

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AN INEXPENSIVE FLOW METER AND HUMIDIFIER FOR ADMINISTERING OXYGEN

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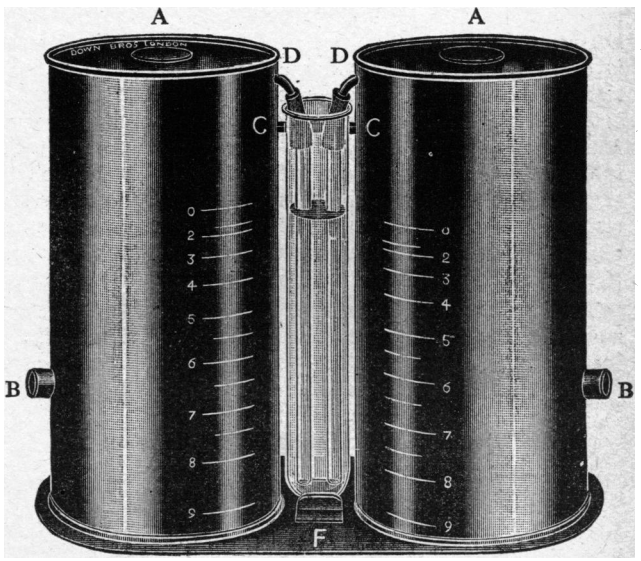
For the efficient administration of oxygen the rate of flow must be known, and this is particularly important now that its relation to the percentage of oxygen in the alveolar air has been established for many different methods of administration. The dial flow meter which is often added to the reducing valve of the oxygen cylinder, because it is really a pressure gauge, may give inaccurate results, and the apparatus recently described by Marriott and Robson is expensive since most of the parts have to be specially made and each apparatus must be calibrated by experiment.

The Apparatus

The present flow meter consists of two tins with lids (A) 7 inches high and 3½ inches in diameter, soldered on to a piece of tin plate (see Figure). These tins are connected 1¼ inch below the top by an accurately drawn German silver (nickel) tube (C–C) 3½ inches long, with a diameter between 107 and 108 thousandths of an inch; the tube projects for some distance into each tin. The oxygen passes in and out of the flow meter by the side tubes (B); the nickel tube provides a resistance so that the pressure in the proximal tin is higher than in the distal tin, the flow being measured by the difference between these pressures. The pressure gauge consists of



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