smelting and in processes involving exposure to carbon bisulphide, nitrobenzene, and trinitrotoluene short shifts must be enforced. Hours of work must be limited for men working in compressed air or in hot deep mines. The factory physician must make use of all known methods to detect the earliest onset of poisoning by substances such as lead, benzene, and tetrachlorethane.

14.—Selection of Immune Persons

Employment of women and of all persons under the age of 18 in certain lead trades must be forbidden. In gasworks and on blast furnaces, where there is a danger of carbon monoxide poisoning, it is better to employ middle-aged men, who are partially protected by their lesser physical activity and slower respiration. The selection and employment of only immune persons is an ideal the attainment of which must wait on better knowledge. If research could produce satisfactory tests for immunity to various poisons a great deal of suffering and expense could automatically be abolished.

15.—Further Researches

It would be idle to suppose that our knowledge of industrial diseases is a finished chapter. Many diseases of occupational origin remain unknown for years, notable instances being asbestosis and Weil's disease. Clearly there must be others still eluding our search. New processes are constantly springing up. In some countries new solvents for quick-drying paints and varnishes have been allowed to poison men, whereas they should have been first tested on experimental animals. A new method of tin refining recently produced outbreaks of arseniuretted hydrogen poisoning under circumstances which should have been condemned beforehand by chemists, since it is known that the action of water on metallic arsenides is to set free this deadly gas. Until 1933 the co-operation of geologists in attacking the problem of pneumoconiosis had not been sought. The petrological microscope rapidly incriminated silicates such as sericite and sillimanite, whereas previously silica had been held solely responsible. Recently fluorosis of bones, a new horror of industrial medicine, was unearthed. Is this condition widespread among cryolite crushers elsewhere than in Denmark? Do bath enamellers suffer from it? What, if any, will be the ultimate consequences to bone and bone marrow of the deposition of calcium fluoride in the skeleton? Is the high incidence of malignant disease of the nasal sinuses in workers in nickel related to substances used in their work, and is there also a high incidence of lung carcinoma among them? Will the chemist find a suitable substitute for nitrate of mercury in the felting of fur? Will the bacteriologist succeed in producing effective inoculation against Weil's disease? What is to be done about Raynaud's phenomenon, produced by the use of vibrating tools, including pneumatic drills, rams, chisels, and riveting machines? Will the phenomenon, hitherto confined to women, become equally common in men? These and many other problems await solution. We are on the right path, but we have a long way to go.

The King Edward's Hospital Fund for London has made arrangements for Coronation tours of royal palaces and other historical buildings famous throughout the Empire. These privileged visits will take place on April 7, 10, 14, 21, and 28; May 19 and 28. Tickets are 10s. each or £2 10s. for the series, and the entire proceeds are devoted to the Fund. Communications should be addressed to the secretary, King Edward's Hospital Fund for London, 10, Old Jewry, E.C.2.

EPIDEMIC CATARRHAL JAUNDICE IN SCHOOL CHILDREN

BY

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Towards the end of 1936 an outbreak of jaundice occurred in Leicestershire, the villages of Newbold Verdon, Desford, and Syston being chiefly affected. The epidemic was mild and no deaths resulted. Altogether forty school children suffered from the disease, and it is estimated that this figure represents 60 to 70 per cent. of the total number of cases, pre-school children and young adults being also affected.

The first intimation of the outbreak came from Newbold Verdon, when the head teacher of the local school reported in November that five children were absent suffering from jaundice. It was decided that I should undertake a special investigation in order to ascertain whether or not the disease was caused by the *Leptospira icterohaemorrhagiae*.

Nomenclature

Epidemics of jaundice have been recorded since ancient times and have occurred at one time or another in every part of the world. No attempt at classification was made until Weil (1886) published an account of the symptoms in a number of severe cases.

Map showing the relative position of the villages where cases of epidemic catarrhal jaundice occurred.

Cockayne (1912) went a step further and differentiated between Weil's disease and the milder type by styling the former "infectious jaundice" and the latter "epidemic catarrhal jaundice." This classification was commonly used until Hurst and Simpson (1934) produced definite evidence that there were in reality two forms of the milder jaundice, and distinguished the infective hepatic type from the epidemic catarrhal.

In November, 1914, Inada and Ido discovered that the organism responsible for Weil's disease or infectious jaun-

dice was the *Leptospira icterohaemorrhagiae*. All attempts to isolate the organism or organisms causing the other types of jaundice have so far failed.

Historical Review

Outbreaks of epidemic catarrhal jaundice are not infrequent in this country. Morgan and Brown (1927) described an extensive outbreak in the Midlands in which two hundred persons were affected and one fatality occurred. Particulars of an epidemic in Surrey were published by Booth in 1928. Two years later Pickles, outlining an outbreak of catarrhal jaundice in Yorkshire, states that in many ways it resembled the Surrey epidemic. Findlay, Dunlop, and Brown (1931) published an account of numerous small outbreaks of jaundice occurring during the two previous years in Surrey and particularly affecting elementary school children. During the same year Glover gave an account of an outbreak in an unnamed small country town in which are situated three boarding schools, all of which were affected. In 1934 Bashford published an account of an outbreak of epidemic catarrhal jaundice among the workers in several departments of the General Post Office in London.

Montford (1934) describes an epidemic in the Castle Donington district of Leicestershire in which forty-five cases were identified as acute infective jaundice, all except two being children. Frazer (1935), in reporting the occurrence of epidemic catarrhal jaundice among children in Staffordshire, states that the outbreak was of a similar nature to that which occurred during the previous year at Castle Donington, which is about fifteen miles distant.

There appears to be some uncertainty with regard to the classification of these two last-mentioned epidemics, as Barber (1937) classifies both of them as infective hepatic jaundice.

Ramage (1935) describes an outbreak of epidemic catarrhal jaundice in Holland, Lincolnshire, and estimated that 80 per cent. of the cases occurred among school children.

Incidence

The present investigation was confined to the outbreak of jaundice among school children, though, as mentioned above, this figure represents about 65 per cent. of the total number of cases, as pre-school children and adults also contracted the disease, particularly young adults between the ages of 16 and 25.

The distribution of the cases was as follows:

District	Village	Population	Number of Cases (40) Investigated
Ma ke Bosworth R.D.	Newbold Verdon	1,251	26
Barrow-on-Soar R.D. ...	Syston	4,322	7
Marke: Bosworth R.D.	Desford	1,163	4
" "	Cadeby	122	2
" "	Orton-on-the-Hill	191	1

As this epidemic occurred in the autumn and winter months the seasonal incidence corresponds with that recorded in the majority of similar outbreaks.

It is also interesting to note that the age and sex incidence as shown below agrees with the findings in previous investigations in that it is evenly distributed between the sexes, and the highest incidence occurs in children between the ages of 6 and 10.

Age	Males	Females
0-5	2	3
6-10	14	15
10-15	3	3
	19	21

Aetiology

Every effort was made in an endeavour to ascertain the mode of spread of the disease.

Attention was first directed to the water and milk supplies in the villages concerned. Excepting Desford and Syston, each of which has a main water system, it was found that the sources of water supply were multiple, there being a large number of pumps, each supplying one or more houses. The children have, of course, common access to the water supply at the school which they attend, but this fact was not helpful as all the children in any one of the villages affected did not attend the same school. The sources of milk supply were equally varied, and thus no evidence was found of a likelihood that the disease was transmitted by either of these two routes.

The question of infection spreading by means of rats was also considered, but here again no definite evidence was forthcoming. During the past few years the villages have been comparatively free from these vermin; therefore any contamination of food is unlikely.

On investigating the spread among the school children several interesting facts came to light. With few exceptions the children who developed the disease gave a history of having been in close contact with other children who either had the disease at the time or developed it shortly afterwards. The contact was in most cases either a relative or another child habitually sitting in the next seat at school.

Although all those in contact with a case did not develop the disease it would appear that a very close personal contact is necessary for the spread of epidemic catarrhal jaundice, and whatever the causal organism it is no doubt transmitted by droplet infection. It would also seem almost certain that a child was infectious during the incubation period of the disease.

It was difficult to determine the probable incubation period, and although in Newbold Verdon the cases tended to occur in batches with three-day to five-day intervals between them, little can be deduced from this fact. In several instances where two children in the same family were affected there was approximately a month between the dates of the first symptoms in each case, and it is quite reasonable to assume that the second victim contracted the disease from the first. On summarizing the length of time between the date of the first symptoms and the alleged contact with the disease, it is found in the forty cases under observation that this period more nearly approaches four weeks than one week.

The distribution of the epidemic in the county was at first difficult to account for, there being no direct communication between the villages concerned, with the exception of Newbold Verdon and Desford. Investigation, however, disclosed the fact that a number of young adults from the villages work in factories some distance away. It was also learnt that cases of jaundice had occurred among these factory workers, and indeed the epidemic probably originated through this channel from some extraneous source.

Clinical History

The clinical features of the disease were remarkably constant throughout, though there was some variation in

the severity. In one instance symptoms were completely absent until jaundice appeared.

The onset was gradual, and for several days before definite symptoms developed the child complained of a headache with perhaps nausea, loss of appetite, and a feeling of lassitude and sleepiness. At this stage the tongue was usually covered with a slight fur and the breath fetid. In most cases bradycardia could be detected, while in some the face was flushed, though the temperature was found to be normal. Diarrhoea was sometimes present, but constipation was more usual. Vomiting, which is the first definite symptom, then occurs and varies in severity, some children only vomiting once, while others continued for an hour or more. This vomiting has been attributed by some writers to a duodenitis. Pain in the upper abdomen accompanied the vomiting, in several instances being so severe that the parents thought the child had appendicitis. The pain diminished as the vomiting subsided, but a slight tenderness on palpation persisted for some days over the region of the gall-bladder, which was palpable in several cases. There appeared to be a correlation between the rise in temperature and the severity of the disease, for in those cases which were febrile the symptoms were more marked.

From one to fourteen days after the acute symptoms developed jaundice appeared and the child at once began to feel better. At first the conjunctivae were affected, then the skin of the face, neck, arms, trunk, and lower limbs became yellow. In some instances the jaundice was less intense and did not spread beyond the eyes or face, while in others it was very intense and the patient exhibited the peculiar orange flush of the face that has been described in previous epidemics. About the same time as jaundice appeared the faeces became pale and the urine dark, though an occasional dark bile-stained stool was passed. Itching of the skin was only present in those children where the jaundice was widespread and deep in colour. Convalescence was complete in about a fortnight and no complications occurred.

The chief symptoms in order of frequency in the forty cases investigated are here summarized.

Jaundice	occurred in	40 cases
Faeces pale	"	40 "
Urine dark	"	40 "
Vomiting	"	36 "
Pain in the epigastrium	"	34 "
Loss of appetite	"	34 "
Lassitude	"	34 "
Sleepiness	"	31 "
Headache	"	30 "
Dizziness	"	21 "
Temperature	"	18 "
Rambling	"	17 "
Itching	"	12 "
Diarrhoea	"	8 "
Sore throat	"	1 case

Laboratory Investigation

Investigation under this heading was undertaken by the Leicester City and county laboratories. The following is a summary of the specimens submitted for examination, and the findings:

Blood.—Specimens of blood were taken from two cases, each on the sixth day of the disease. On microscopical examination of the centrifuged deposit no spirochaetes were detected. Another specimen, taken from a case on the forty-ninth day after the first symptoms developed, was also submitted and the serum, tested for agglutination against the following, proved negative in all dilutions: *B. typhosus* H and O, *B. paratyphosus* A and B, *B. dysenteriae* Sonne, *B. dysenteriae* Shiga, *B. dysenteriae* Flexner, *B. aertryche*, and *B. abortus* Bang. Blood films were taken at various stages of the disease from all the forty cases investigated

and a differential count made. A number of the films showed an eosinophilia sometimes as high as 15 per cent., but as this high count is frequent in routine films taken from country children, where the cause is usually threadworms, no significance is attached to the present findings. In the majority of the films, however, a monocytosis was evident, varying from 2 to 19 per cent., while in a number of others taken from contacts who did not develop the disease the monocytic counts were normal.

Urine.—Altogether twelve specimens of urine were examined for spirochaetes where the period of the disease varied from the seventh to the thirty-third day. A centrifuged deposit from 15 c.cm. of urine was injected peritoneally into two series of guinea-pigs, all of which survived three weeks. In addition the specimens of urine, which were deeply bile-stained, were examined microscopically. An occasional leucine crystal was present in one specimen, but the others showed no leucine or tyrosine crystals, red blood corpuscles, or casts. No spirochaetes were found in any centrifuged deposit.

Faeces.—A specimen from a typical case was collected in the acute stage and submitted for examination for any abnormal bacteria. The result was negative.

In my opinion the above results can be accepted as definite evidence against the epidemic being either of spirochaetal origin or due to any of the other possible organisms for which tests were carried out.

Differential Diagnosis

Weil's Disease (*Spirochaetosis icterohaemorrhagica*).—Weil (1886) was the first to publish an account of this disease. Since then a number of similar outbreaks have been described and referred to as Weil's disease, but it was not until 1914 that the disease as described by him became definitely linked with the *Leptospira icterohaemorrhagiae*. In November of that year two Japanese workers, Inada and Ido, inoculated a guinea-pig with blood from a patient suffering from the symptoms of Weil's disease, and subsequently discovered the spirochaete in the animal's liver. Since then a good deal of work has been done in various epidemics, notably that by Dr. Adrian Stokes during the great war, who proved that rats carried the organism. Many outbreaks of spirochaetal jaundice have occurred on the Continent and in Asia. With regard to this country, outbreaks of Weil's disease are very rare. Buchanan and Gulland (1923) report the finding of the spirochaete in the blood and urine of miners in East Lothian who were suffering from jaundice. Since then other small outbreaks in Scotland and England have been reported. The onset of the disease is sudden and is ushered in with general malaise, severe headache, violent muscular pains, giddiness, loss of appetite, high fever, epistaxis, and usually diarrhoea. The symptoms become more acute as the disease progresses, and nervous disturbances increase, delirium frequently being present. From about the third to the fifth day jaundice appears and is accompanied by enlargement of the spleen and liver and tenderness over the latter organ. The urine contains albumin and perhaps blood. Disturbances of the digestive system are marked, there being furred tongue, vomiting, and abdominal pain. The temperature falls about the twelfth day and the jaundice begins to fade about the same time. Convalescence is slow, and leaves the patient weak and exhausted. The death rate is relatively high, and has been estimated in one epidemic even as high as 45 per cent. The *Leptospira icterohaemorrhagiae* can be isolated and identified microscopically in a centrifuged deposit of the blood taken about the third day of the disease, in a similar deposit of the urine in the later stages, or by animal inoculation with the same material and subsequent recovery of the organism from the liver and spleen. The above are the

usual symptoms that occur, but some variation in type and severity has been noticed in several of the epidemics described.

Infective Hepatic Jaundice.—Hurst and Simpson (1934) showed conclusively that this type differed from the closely allied epidemic catarrhal jaundice. Several epidemics are classified under this heading by Barber (1937), particularly those recorded by Montford (1934) in Leicestershire and Richards (1933) in Derbyshire. The clinical picture is quite distinct from that in either Weil's disease or epidemic catarrhal jaundice. Usually there are no pre-icteric symptoms of any kind. Pale faeces and dark urine occur immediately prior to the appearance of jaundice, which is accompanied by headache, general malaise, occasional vomiting, diarrhoea, and a slight rise in temperature. The liver and spleen are enlarged though epigastric tenderness is absent. The jaundice is less marked but of longer duration than that of epidemic catarrhal jaundice. Convalescence is prolonged and the patient is left very weak. The mortality rate, though low, is higher than that in epidemic catarrhal jaundice. Appropriate tests disclose an hepatic insufficiency, and in the few post-mortem examinations described hepatic necrosis was present.

Simple Catarrhal Jaundice.—Usually of a very mild character and due to a mechanical obstruction of the biliary passages; said to be due to colds, chills, etc., and often occurs in children. The symptoms of this type and of epidemic catarrhal jaundice are very similar, and it is often difficult to differentiate between the two. Owing to the absence of bile the stools in simple catarrhal jaundice are pale and remain so until obstruction to the bile flow is removed. In the epidemic type, although the stools are for the most part pale, an occasional bile-stained stool is passed.

Enteric Group.—Jaundice rarely occurs in this group of fevers, and although the symptoms resemble in some respects those in epidemic jaundice the agglutination tests and bacteriological examination of the excreta reveal the presence of the organisms belonging to this group.

Acute Yellow Atrophy of the Liver.—Of very rare occurrence, particularly in children. The post-mortem findings are somewhat similar to those in infective hepatic jaundice.

Treatment

All the cases which occurred in this epidemic were under the care of the local general practitioners, and treatment chiefly consisted of confinement to bed for a few days and the relief of symptoms.

It was not considered desirable or necessary to close any of the schools involved. Those children who had been in contact with the disease, also the senior children from the affected villages attending central schools, were, however, excluded for several weeks, and it was impressed upon the parents of the former to keep them in strict isolation during the period away from school. The parents of children who had contracted the disease were advised to prevent them mixing with other children until convalescence was complete.

Summary and Conclusions

1. The recent occurrence of jaundice in Leicestershire as detailed above was undoubtedly an outbreak of epidemic catarrhal jaundice and was very similar to the epidemic in the Midlands described by Morgan and Brown (1927).

2. School children principally were affected, and it is estimated that they comprised from 60 to 70 per cent. of the total number of cases.

3. There was no evidence that the disease was spread through the medium of water or milk supplies, or by rats.

4. It is evident that epidemic catarrhal jaundice is spread by close personal contact, probably by means of droplet infection.

5. The incubation period is about four weeks, and children with the disease appear to be infective during this stage.

6. No causal organism was found, though undoubtedly it exists, probably in the form of a virus as suggested in previous epidemics.

I am indebted to Dr. J. A. Fairer, county medical officer of health, for permission to publish the particulars of this investigation.

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EXTERNAL VERSION FOR BREECH PRESENTATIONS *

BY

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Compared with vertex presentations the incidence of breech presentations is low but the foetal mortality is high. Pierson (1923), Westman (1931), Dearnley (1931), Studdiford (1932), Geothals (1936), and Williams (1924) placed the incidence of breech deliveries between 2.3 and 3.7 per cent. Kedar Nath Das (1926) placed it at 3.3 per cent., and the latest report of the Wadia Hospital (1935) at Bombay gave it as 3.1 per cent. Ryder (1923), Pierson (1923), King and Gladden (1929), Gibberd (1931) Studdiford (1932), Morton (1932), Donovan (1933), Cannell and Dodek (1934), Gordon, Garlick, and Oginz (1934), Barney (1934), Geothals (1936), and St. Mary's Hospital report for 1935 record the foetal mortality as varying from 8 to 26 per cent.; in complicated and premature breech presentations it was as high as 43 per cent. and 48.6 per cent.

Scope of Investigation

Desiring to find out the state of affairs at the Bhagat Hospital for Women and at my private hospital I have reviewed 5,028 deliveries—3,028 from my private hospital and 2,000 from the Bhagat Hospital.

In the first 1,240 deliveries at my hospital and 2,000 at the Bhagat Hospital all cases of breech presentation diagnosed during pregnancy were allowed to continue as such: these are reviewed as Group I. The next 1,788 consecutive cases at my hospital were treated by external version whenever a breech presentation was diagnosed during pregnancy; these represent Group II. All emergency breech cases and those undergoing breech delivery

* Abridged version of a paper read before the Bombay Obstetric and Gynaecological Society.