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INFECTIVE HEPATITIS: THE HISTORY OF AN OUTBREAK IN THE LAVANT VALLEY

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Hepatitis had been present for some time in several places in the coastal area of Sussex to the west of Arundel when the first case was reported from the village of West Dean. This village lies some four miles to the north of Chichester, where the valley of the River Lavant enters the Downs, accompanied by the London road. At Singleton, a mile further on, the river leaves the road and turns to the east to the villages of Charlton and East Dean, which lie in a secluded hollow, and have little communication with the outer world other than by the main road. This lie of the land made the investigation of an epidemic much easier than would otherwise have been the case, and also showed the essential unity of what might have been regarded as sporadic cases of disease. Had there not been separate villages, visited by a single doctor, but separate streets in a large town visited by several, there would probably have been no more comment than on the curious fact that two or three cases of catarrhal jaundice should appear about the same time.

Cases in West and East Dean

About the middle of July, 1939, Betty Swift, a housemaid employed at the local sanatorium, suffered from an attack of hepatitis with jaundice. A fortnight after the beginning of the illness she went to her home in West Dean. None of the six other girls with whom she had lived till then was infected, nor were any of her relatives at home. Her history was discovered only when her brother developed the disease from an unknown source three months later. He worked at Charlton Saw Mills and had visited Chichester, but knew of no case of jaundice in either place.

Case 1.—Bill Swift, aged 19, was taken ill gradually about September 20 with an ache in the middle of his epigastrium. He stayed in bed on the 23rd, but on the next two days got up and went out, and on the 26th returned to work. But he had to give up and go home to bed. He felt very ill, was irritable and a little drowsy, and vomited repeatedly. The next day his urine was dark, and on the 30th he began to turn yellow all over. After a fortnight in bed he returned to work, and was later troubled only by itching.

Among the other workers at the saw mills was Gerald Sparrow, who lived in the village of Cocking, some six miles away. He had not visited any of the known sites of jaundice, nor had there been any cases in his neighbourhood. What was even more curious was that the two worked in different parts of the saw mills, and had not, so far as they knew, had any meeting at all. Yet 34 days after Swift's last appearance in his neighbourhood Sparrow was taken ill.

Case 2.—Gerald Sparrow, aged 18. The onset of illness was sudden at dinner-time, and later in the day his urine was the colour of beer. For the next three days he felt sick, sweated, and had a pain like a stomach-ache across his upper abdomen. On the second day he turned yellow; but he never took to bed, and after three days at home he went back to work and no more was heard of the disease in connexion with him.

Bill Swift had two brothers, but he infected only one of these, and his sister, Mrs. Martin, who lived in the village of East Dean.

Case 3.—John Swift, aged 13, set out for Chichester on October 14 feeling perfectly well; but while he was in a shop his "head went round," he vomited, and he had to be brought home. He was quite well the next day, but on the 16th, at 2 p.m., he was taken ill again with headache, stomach-ache in the epigastrium, and nausea. He went home to bed, and the doctor was called. That day he was feverish, sweating, and drowsy, and he vomited. The next day his urine was discoloured and his nose bled; these symptoms continued until October 21, when jaundice began, later spreading all over his body. Thereafter the symptoms rapidly abated.

Of John Swift's contacts two became infected. His brother Alan fell ill 34 days later, and Brenda Wren, one of twins billeted next door, in 19 days. Brenda might, however, have been infected between September 20 and 26 when Bill Swift last went out 37 days before.

Case 4.—Brenda Wren, aged 7, vomited repeatedly and refused all food. She was sleepy, and later her urine was as dark as beer, and she passed frequent pale stools but never showed jaundice in the face. She was away from school only two days.

Case 5.—Alan Swift, aged 8, was taken ill gradually on November 17 with headache, drowsiness, and malaise, which kept him in bed next day, on which vomiting began. On the 19th he was well enough to get up, and on the 20th to go back to school; but he was glad to return to bed that evening; at the same time his nose bled and jaundice was noticed to be starting. He spent the rest of the week in bed with his symptoms abating.

Mrs. Martin is Bill Swift's sister and lives in East Dean. There was frequent intercourse between the two branches of the family, but she was able to give September 24, the day on which Bill Swift was up in the middle of his illness, as the one occasion on which she had visited him while he was ailing.

Case 6.—Mrs. Martin, aged 23, began to feel ill during October 22 with terrible pains in the top of the head. She had to go to bed on the third day with the following symptoms, which she attributed to "flu": extreme irritability, sweats that soaked the bed, complete loss of appetite, continuous nausea and occasional vomiting, a full feeling in the stomach, photophobia, and drowsiness. Her urine was dark after the first day or two, but it was not till the fifth that she turned yellow, and the diagnosis thus became obvious. Her recovery was rapid but for troublesome nose-bleeding in the second week.

Mrs. Martin infected two others—her daughter Margaret and a boy next door, Owen Finch. She was known to have been in the Finches' house on the day before she went to bed, but thought that the boy was at school at the time.

Case 7.—Owen Finch, aged 10, vomited on the morning of November 17 and again all night. He had a headache, pins-and-needles in his feet, was drowsy and irritable, sweated till his clothes were soaking wet, and refused his food. On the following day his urine was dark, and on the next jaundice began; he has been quite well since.

Case 8.—Margaret Martin, aged 3, vomited three times in the night of November 24. The next day she was poorly and

irritable and her urine was dark, but she never showed any discoloration of her skin or eyes. On the 28th she had no symptoms and no jaundice; but on examination her liver could easily be felt, quite firm and smooth but not tender, half-way to the umbilicus. The spleen was not palpable.

Finally, and presumably from Margaret although she slept in her mother's room, Susan, the other child in the family, became affected about three weeks later.

Case 9.—Susan Martin, aged 15 months, had an illness in which she seemed "very fractious" and poorly, with sleepiness, diarrhoea, and vomiting. She was "a little yellow." The nurse noted that her urine was dark and her stool pale.

The First Main Stream of Infection

The outbreak had now finished in East Dean, and it was found to flow in two main streams, which it will be best to follow separately. In a house on the outskirts of Singleton lived a family of five adults and one child, friends of the Martins and Swifts, and two of them, Mrs. Hawk and John Hawk, fell ill on November 28 and 29, 37 and 38 days respectively after Mrs. Martin. The only common meeting was at a birthday party on November 9, when all were quite well; but the Hawks had met the Martins individually, though seldom, and also at times when there was no illness. On the other hand, Mrs. Martin's sister had been a constant visitor to both houses throughout the time when the Martins were successively ill, and may thus have acted as a carrier.

Case 10.—Mrs. Hawk, aged 35, was taken ill suddenly at breakfast on November 28 with awful pains all round her stomach that made her sit still and hold a hot-water bottle all morning. She spent the next two days in bed feeling very ill and irritable and with a severe headache. The thought of food made her shudder, but she did not vomit. Her urine was the colour of tea; but she herself did not notice any change in her eyes, though at the end of the week her husband remarked that she looked yellow.

Case 11.—John Hawk, aged 10, had a very insidious onset to his illness. He was well when his mother succumbed, but began to ail during the same week with headache, malaise, irritability, and poor appetite. For a fortnight he vomited intermittently and complained of a constant pain in the top of the stomach. From December 20 his urine was dark for a day and a half and his stools were pale.

The nature of the illness in these two was not at first recognized, and in fact the colour of John's urine was attributed to a surfeit of beetroot. Then, by comparing notes with the Martins, they decided that the illness had been the same as theirs. That they were right was shown by Mr. Hawk's developing a severe attack of the disease 35 days after his wife.

Case 12.—Mr. Hawk, aged 33, on January 2 suddenly felt a chill in the back and aching in the loins. He continued to work for two days more, but on the third day had to give up with a temperature of 103.8°. He was irritable and very sleepy, and ate nothing. His sweats were so profuse that the blankets were wet through to the top one. On the 7th his temperature was 97° and he got up feeling stiff; on the next day he went back to work—driving a lorry. But he relapsed on the way home and his temperature rose to 102.8°. On the 10th his temperature was subnormal but he vomited three times, and on the 11th he began to turn yellow and his stool was clay-coloured. His symptoms then left him, and after an unspecified time he was able to go back to work. But a week later the symptoms returned. This time, however, there was no evidence of deviation of his bile. Even at the end of March his "stomach was still readily upset."

The next victim in the Hawk household was John Eagle, who fell ill 22 or 28 days after Mr. Hawk.

Case 13.—On January 24 John Eagle, aged 30, vomited. After this he remained quite well till the 30th, when he was taken ill suddenly while tobogganing and went to bed. The next day he had two rigors, was delirious, vomited twice, and had a temperature of 105°. His doctor naturally suspected pneumonia. His condition remained much the same, except that there were profuse sweats but no more rigors, till on February 3 his parents were sent for. But his delirium ended next day and his condition rapidly improved: on the 3rd he had seemed to be *in extremis*; on the 6th he was "quite bright." His urine was observed to be dark before the 7th, on which date jaundice was definitely noted: his eyes were yellow, but his skin only slightly so. Later his appetite became voracious, and he went home apparently

well. Soon he had to go back to bed, feverish and vomiting, but the jaundice did not return.

Whether the next illness was truly hepatitis or influenza is a matter for conjecture. The patient was taken ill on February 9, 16 days after Eagle—an unusually short incubation period for hepatitis. But he might, like Eagle, have been infected by Mr. Hawk 38 days before.

Case 14.—Hubert Hawk, aged 25, came over hot and cold on February 9. Next day he was all right; but the illness returned on the 12th with headache, pain in the back, sweating, and a temperature of 102°. Unfortunately he left the district, and it was not possible to ascertain his subsequent history.

Mrs. Hawk, who has already appeared as Case 10, was taken ill again 30 days after Eagle took to his bed. Her symptoms recalled to her the original attack. But if there was any doubt about the nature of the first there was none about the second.

Case 15.—Mrs. Hawk was on March 1 seized more or less suddenly with "terrific indigestion" under her shoulder-blades and through the epigastrium. The next day she had to go to bed with a severe headache, shivering, sweats, a temperature of 103.8°, nausea and vomiting, and complete loss of appetite. She could not bear the light, was drowsy but unable to sleep, and had pins-and-needles in her arms and cramps in her legs. The pain lasted only one day, and on the 3rd her urine was dark. Her symptoms now began to abate, and though by the 6th she was intensely jaundiced, "like a Malay," she felt well enough to get up. Her stools were clay-coloured. Later she was troubled with itching; and three weeks after the onset her eyes were still yellow and she had lost two stone in weight.

By now there was only one survivor in the succession of victims in the Hawk family, and he succumbed about 26 days later.

Case 16.—Cyril Hawk, aged 30, "felt rotten" for two or three days. On March 29 he had a bad back and a general ache all over his sides and chest. He had to give up work, and went to bed with a temperature of 101.8°, which persisted for five days. He sweated, was irritable, and vomited twice, but was not troubled by nausea or loss of appetite. He had cramp in his legs and feet. His urine turned dark before jaundice began on April 3, and recovery was then rapid.

The Second Main Stream of Infection

It is now time to return to the other stream of infection, which had apparently ended with Alan Swift's illness on November 17. Nothing more was heard of hepatitis in the village of West Dean until Mr. Raven, the head master of the school, was taken ill early in January. On being informed of his attack a search was made for a connecting link between him and the last known case, and by reference to a school attendance register it was possible to trace a number of children who had been absent during the month of December. At this time colds had been prevalent, but in the end one child was found whose illness was very suggestive.

Case 17.—Doris Gull, aged 10, was away from school from December 11 to 13. She said that she had felt cold, had had a headache and a temperature, and had vomited four or five times. When asked to describe the colour of her urine she said without hesitation that it was "very dark brown." Her hostess, however, could remember nothing of the child's illness except that she had had "a cold" with a temperature of 103°. She had not been jaundiced.

Doris Gull's illness therefore seems to have been a mild attack of hepatitis coming 24 days after Alan Swift's and 27 before Mr. Raven's.

Case 18.—Mr. Raven, aged 31, suddenly felt a pain below the ribs just before dinner on January 7. He felt sick, and shivered in the night. The next day he went to school, but had to give up during the morning and go to bed; he took aspirin and for the next three days sweated profusely. Vomiting began on the 9th and was persistent for three days. For the first eighteen days his temperature was never less than 100°. On the 13th his doctor noted the presence of albuminuria. About a week after the beginning of the illness jaundice appeared, spreading all over the body. His urine was the colour of dark sherry intermittently almost from the onset. His bowels were loose and his stools very pale. Recovery was gradual, but he was quite well in a month. On examination on the 18th day, when the pyrexia was coming to an end, he was tender under the costal margin, but neither liver nor spleen could be felt.

There can be little doubt that Mr. Raven was the means of infecting the next five cases—his wife, and four children who attended the school. As regards these latter the incubation period can be assessed with an unusual degree of accuracy. The head master was taken ill on the day before the beginning of term. On the first day of term he went to school in the morning only, and it must therefore have been between 9 a.m. and noon on January 8 that he passed on the infection. Of the children infected two were taken ill on February 6, one on the 7th, and one on the 8th, giving an incubation period of 29, 30, and 31 days respectively. And as they lived in the village, an isolated farmhouse, and the hamlet of Binderton, a mile to the south, there was no likelihood of their having caught it elsewhere. Mrs. Raven, whose infection could scarcely have been contracted earlier than the 7th, the day on which her husband first felt ill, herself succumbed 24 days after him.

It is unnecessary to enlarge on the clinical features of most of these and subsequent attacks. Suffice it merely to say that they conformed in all essential particulars with those that have already been described, with the exception that in them for the first time in this outbreak conjunctivitis was noted. The eyes were not deeply bloodshot, but had a suffused and bleary look; and while it was not a symptom which the relatives volunteered, it was one about which they seemed to have no doubts. Though it is more typical of spirochaetal jaundice, this sign was found by Bates (1936) to be a valuable diagnostic test.

Case 19.—Mrs. Raven, aged 34, fell ill gradually on February 1 and turned yellow on the eighth day. Her eyes were slightly congested then, and she was much troubled by itching during convalescence.

Case 20.—Edward Daw, aged 9, of Binderton, began a typical attack on February 6. His eyes were pinkish before they turned yellow.

Case 21.—Stephen Rook, aged 8, also of Binderton, sickened for an attack with jaundice on February 6.

Case 22.—Mary Crow, aged 9, in a remote farmhouse, sickened for a similar attack on February 7.

Case 23.—Tom Chough, aged 12, West Dean, was taken ill in school on February 8 and developed slight jaundice on the 10th. His eyes looked "as if he had got a cold in the corners."

The subsequent history of the families of these victims is interesting. Stephen Rook passed on the disease 20 days later to a girl billeted in his home, but she took it so mildly that dark urine was the only certain evidence of its nature at the time, and her hostess noticed this only because she had been warned to be on the look-out for it. At this point the chain of evidence snaps, for the last case in the outbreak also came from the same house and occurred on April 29, 62 days later. At the time circumstances prevented a proper inquiry, but in retrospect it is tempting to halve 62 and to suggest that a missed case intervened on about the 31st day.

Case 24.—Helen Drake, aged 10, living with the Rooks, felt sick on February 26 and was kept in. The following day her urine was dark, and she spent the next six days in bed. She had no appetite and felt ill, but never turned yellow.

Case 25.—Elizabeth Rook, aged 13, fell ill with a cough and malaise on April 29. She continued very poorly, with general and gastric symptoms, till May 10, when jaundice was well established. Her eyes had been red.

But though there was no more jaundice to report from the houses so far infected, the history of illness had not come to an end. On February 27, 20 days after Mary Crow had been taken ill, her two brothers had to stay in bed. They were alleged at the time to have had "colds," but it is tempting to suggest that there may have been some other explanation of their illness, though impossible to prove it.

Cases 26 and 27.—Hugh and Willie Crow, aged 4 and 7, were feverish on February 27 and were kept in bed for three days. Hugh had a sore throat, Willie had not; Willie vomited, Hugh did not. But they both had headaches, refused all food, slept the whole of the first day, and had bleary eyes.

Another interesting example of a similar type of illness came from the Daw household. Edward had had hepatitis on February 6. Some 30 days later, between March 8 and 11, the rest of the family save his father went down with "flu."

Cases 28 to 32.—Mrs. Daw and four children aged 3 to 13. Individual details are lacking, but headache, nausea, drowsiness,

complete loss of appetite, and "pinching" pain in the stomach were all given as common early symptoms. Later they developed coughs. One vomited; but none appeared to show any evidence of jaundice.

One further case remained—that of Florence Reeve, who was employed in a local hotel and was taken ill on April 17. Again no case of hepatitis was known in her neighbourhood, and the only cases in the district had been unconnected with her; but a little sister, aged 9, had been poorly for a week in March with "flu," in this case characterized by tiredness and refusal to eat her food. There had been no bile in the urine and no vomiting, and no one had taken much notice of her indisposition. Had this child, then, acted as an unrecognized link between Florence and the cases in the school about February 7, 69 days before?

Case 33.—Florence Reeve, aged 19, was taken ill gradually from April 17 and began to turn yellow on the 23rd.

The Clinical Features and their Significance

The typical attack of hepatitis has been well represented in the foregoing case histories. Its symptoms are not specific enough to make a diagnosis easy except in times when the disease is known to be prevalent. But its eventual recognition is certain. In its severest form the differential diagnosis is even more difficult. In this series Case 13 was at first thought to be one of pneumonia. Where headache, drowsiness, and fever are the predominating symptoms, with occasional vomiting, and maybe photophobia, the picture may resemble that of meningitis. Where pain is very severe an "acute abdomen" may seem to exist. And there is always the typhoid group of fevers lurking in the diagnostic background. Moreover, one attack gives no lasting immunity (Cases 10 and 15), and so a history of previous infection does not entirely exclude a diagnosis of hepatitis. No doubt help in recognizing hepatitis at an early stage (and particularly in distinguishing it from Weil's disease) could be obtained from a white blood count, which in hepatitis would usually give a figure below 8,000 with either a relative lymphocytosis or a monocytosis, or both (Martland and Winner, 1939). But most cases will remain in doubt till bile appears in the urine, an event which nearly always precedes the appearance of jaundice in the eyes, and is therefore well worth special observation.

An interesting variant on the usual course of the illness is the relapsing type, seen in Cases 1 and 5, in which an apparent recovery is followed rapidly by the return of symptoms and the appearance of jaundice. Now reference to a rather scanty literature on post-mortem appearances, which has been well summarized by Findlay, MacCallum, and Murgatroyd (1939) and is supported by biopsies carried out by Roholm and Iversen (1939), shows that the evidence is overwhelmingly in favour of the disease process being a diffuse hepatitis without involvement of bile ducts. But there are a few apparently well-authenticated cases in which the reverse has been the case. The varying colour of the stools, too—in some cases clay-coloured, in others not (Martland and Winner, 1939)—seems to suggest that the obstructive element is a very variable one; and, moreover, there is no doubt at all that the liver can be severely damaged without jaundice developing. Can this relapsing type of illness bring the two theories into line? On such a hypothesis the first attack would be a diffuse hepatitis not producing jaundice; then as the patient begins to show signs of recovery the obstructive element is added and he turns yellow. There would thus be some cases—the majority—in which the jaundice was purely hepatic in origin, others occasionally in which it was obstructive, and others in which the two forms coexisted or followed one another.

Another interesting feature is the existence of hepatitis without jaundice, a fact generally overlooked in previous reports, though Bates recalls the presence among 65 cases

of 6 who "showed no jaundice of conjunctiva or skin although bile was present in the urine and the liver was enlarged," thus providing a striking clinical confirmation of Case 8 above. Cullinan (1939) quotes a case reported to him by A. W. Franklin in which "a child who had been vomiting and unwell for a few days had a slightly tender liver but no jaundice. In the morning there were bile pigments in the urine; in the afternoon they had disappeared." Jaundice, in fact, is a symptom—only one symptom, and a very variable one at that—of a disease in which it is of no greater significance than a scarlatinal rash is in the case of haemolytic streptococcal infection. To refer to the underlying disease as "jaundice" is thus to fix attention on a single feature, with all the danger, now so familiar in the case of scarlet fever, of obscuring the existence and importance of cases in which this feature is not present. Hepatitis can exist with jaundice or without, with bile obviously present in the urine, and with bile noticed only by the expectant observer. It would be reasonable to suppose that the same disease process may exist in what is apparently a completely abortive form with nothing but temporary symptoms attributed to "acidosis," "gastritis," "a bilious attack," or any of the dietary indiscretions which are always easily to be found. (Besides a white blood count the intradermal histamine test described by Cullinan would no doubt be a help in differentiating these illnesses if it could be applied.) The idea of hepatitis as an unrecognized but not usually an unrecognizable illness may at any moment assume great practical importance, and perhaps has only avoided doing so already by remaining to a very large extent a disease of children. Hepatitis has followed the use of human serum for the purpose of immunization (Findlay *et al.*, 1939). An abortive attack of the disease in a blood donor might have disastrous results, the scale of which would depend only on the question whether a single transfusion or pooled immune serum was involved.

Some Epidemiological Considerations

Infectivity.—That the patient becomes infective very early in the disease there can be no doubt. Mr. Raven, for instance, had been ill for less than twenty-four hours when he passed the disease on to the four school children. It is quite likely that by the end of about a week from the onset of symptoms the patient has ceased to be a danger to others. This is very much the same conclusion as that arrived at by Pickles (1939), who has allowed sufferers to return to school at the end of a fortnight and has had no case of infection after their return. In the series of cases just described there is no instance of infection being certainly, or even probably, due to a sufferer after this time.

Incubation Period.—Where the precise moment of infection is so vague it is obviously usually impossible to find the exact incubation period. The case of Mr. Raven provides an almost ideal basis for the calculation, and gives 29 to 31 days. Most authorities now agree with Booth and Okell (1928), who fix the limits at 20 to 41 days. All the cases in this series fall within this period except perhaps Case 4, Brenda Wren, who, as already noted, may have belonged to the longer incubation group of 37 days. There was no evidence at all that the period could be the short one of about 4 days favoured by Glover and Wilson (1931).

Means of Spread.—The most arresting, if not the only well-authenticated, instance of hepatitis being spread by water is the Mount Allison outbreak described by Fraser (1931). In the Lavant Valley series there was no common supply or other evidence pointing to such a source. Nor could food be suspected. Various animals suffer from hepatitis—horses (Findlay *et al.*, 1939), dogs (Pirrie, 1937), pigs (Anderson and Tulinius, 1938), rodents (Bell Walker, 1938)—but though special attention was given to the possibility of animal vectors no such could be discovered, and there was no likelihood of an insect vector being involved. All the evidence points to direct spread from case to case. There is a possibility that a healthy carrier was

responsible for transferring the disease from the Martins to the Hawks, though in this case the evidence was not as conclusive as in two cases already reported from the county (Newman, 1939). Finally, there can be no doubt of the extreme importance of missed cases, which, as is shown by Cases 8, 10 or 11, 17, and 24, may be just as dangerous as florid cases with jaundice.

It is when we come to the means by which one patient infects another that accepted theories least bear scrutiny. Throughout the epidemic it was apparent that the disease spread in series in each family, a feature already noted by Pickles as being without counterpart in any other disease. He attributes this to the fact that it is not very infectious, and then for only a short time. He considers that the means of spread is by droplets. That it is not very infectious there can be little doubt: witness its failure to establish itself widely in either of the two schools at risk. But if infection is by droplets, why did the victims appear in series? If Mrs. Hawk, for instance, was ejecting thousands of infective droplets, why was Mr. Hawk the only one involved when three known susceptibles were constantly present? And that this could not have been a mere coincidence, or due simply to the closer contact between husband and wife, is shown by the same fact being observed in the Swift, Martin, and Rook households. Again, the lack of close contact noted by Bashford (1934) appears strikingly in this series. Sparrow and Swift were quite unknown to one another. Even more remarkable is the fact that when Mr. Raven went to school on the morning of January 8 and infected four children, there was no general assembly as there usually is on the first day of term, and he was quite positive that he could have done no more than pass through the rooms where the children were sitting. In the light of this we are forced back to the rather unsatisfactory explanation—if it can be called an explanation—of the infection being air-borne.

But if an air-borne infection accounts better than the droplet method for the infection of remote contacts, it is as ineffective as the droplet method in explaining the phenomenon of infection in series. Receptivity to infection in this disease, whatever its means of direct transference, cannot have been continuous, or else infection in parallel would have been commoner. The only apparent explanation is that receptivity must be phasic or intermittent. The liver and bile ducts, which all agree to be the organs involved, are essentially phasic in their activities. On this assumption immunity to the noxa of hepatitis is far from absolute, but waxes and wanes over short periods of time; and the chances of a congregation of people all being in the same phase of susceptibility at the same moment are so much reduced as to make the probability of spread in parallel much less. At the same time this would account for a person escaping, even though exposed to an intermittent or short infectivity, only to succumb at a later date to otherwise precisely similar conditions.

The Attack Rate.—The attack rate in the houses involved is shown in the following table:

Age:	20+	15+	5+	0+	Total
Exposed	33	4	27	8	72
Clinically infected	7 (21%)	3 (75%)	12 (44%)	2 (25%)	24 (33%)

This again, making allowance for the abnormally high incidence in the adolescent (over 15) group due to the small numbers, confirms Pickles's observation that "in country districts . . . all those who have not previously suffered are fair game, whatever the age"; but the schools were clearly not the only important factor in facilitating the spread. Pickles is one who subscribes to the common belief in the relatively higher susceptibility of a rural population, pointing out that an outbreak in Leeds on the same scale as his own in Wensleydale would have produced 25,000 cases. This apparent immunity of the towns is an interesting phenomenon not apparently to be accounted for by repeated exposure to clinical cases. It may be, though, that besides the question of individual insusceptibility the abortive case is of significance in this connexion. If mild hepatitis is a common disease it might well be the means of producing a more or less stable state of herd immunity, each town dweller being repeatedly in contact with "bilious attacks."

The same reason might explain the fact that adults are in general less liable to attack than those aged 6 to 10, on whom the highest incidence nearly always falls.

Control.—Infective or epidemic hepatitis is an infectious disease directly transmissible from man to man, and the question therefore arises, What steps can be taken to check it? The first essential is that its infective nature should be generally recognized, and this is not the case at present even within the medical profession. Doctors remember it as having been described in the textbooks under the section of diseases of the liver, and their difficulties are increased by a confusion in nomenclature which is so pronounced that one observer (Lisney, 1937) classifies "infective hepatic jaundice" and "simple catarrhal jaundice" as diseases to be differentially diagnosed from "epidemic catarrhal jaundice." The name "infective hepatitis" is a good one because it signifies the infective nature of the condition, does not stress the single symptom of jaundice, which may be absent, and indicates what is certainly the commonest, if not the invariable, pathological process. Control of an infectious disease usually involves early diagnosis, notification, isolation of the victim, and restriction of the activities of contacts. Early diagnosis is, as already pointed out, an ideal unlikely to be attained at first. Notification would certainly be incomplete owing to the failure to recognize abortive cases; but the information provided would be of great epidemiological interest, and the knowledge that the disease had appeared in a particular area might help towards the early recognition of other cases. Isolation of the sufferer would do little to check the spread of a disease which had probably been already transmitted. And in view of the long incubation period and the generally mild character of the illness, it would scarcely be worth while restricting the activities of a contact for some five weeks unless there was a reasonable certainty that he would become a more serious menace during that time than is actually the case.

Infective hepatitis has repeatedly appeared under war conditions, sometimes on a large scale, and may at any moment become an important cause of incapacity. Once a case has appeared, whether it be in a private house, school, hostel, or Service unit, the medical officer should be on the look out for secondary cases. Contacts should not be restrained from their usual activities, but should be warned that the appearance of gastric symptoms in about a month's time must be reported immediately, so that these can be treated at once, until either the diagnosis has been disproved or the short period of infectivity is passed.

Summary and Conclusions

Infective or epidemic hepatitis, synonymous with "catarrhal jaundice," is a fairly common infectious disease which spreads direct from person to person and tends to appear in series at intervals of about a month. Its period of infectivity is short.

There is a need for a simplification of its nomenclature and a wider recognition of its infective properties.

Jaundice is a common but not an invariable symptom, and the abortive case without it is of pathological interest and of epidemiological importance, in relation both to the direct spreading of the disease and to the use of human serum for therapeutic purposes.

Clinically the disease usually runs true to type, but may simulate not only Weil's disease but acute catastrophes of the lungs, abdomen, or central nervous system. The appearance of bile in the urine is usually the first definite evidence of the nature of the illness. Second attacks are possible.

The evidence suggests that susceptibility is phasic. The droplet theory of its means of spread is not altogether satisfactory.

It is apt to appear under wartime conditions, and the best hope of checking its spread lies in the prompt recognition and removal of secondary cases rather than in an indiscriminate adherence to the usual methods of controlling infectious disease.

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VALUE OF PROCAINE INFILTRATION IN THE DIAGNOSIS AND TREATMENT OF FIBROSITIS

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Fibrositis is the name introduced by Gowers to describe the painful "rheumatic" condition of the muscles and fasciae which has been variously designated "muscular rheumatism," "myalgia," "myositis," and "myofascitis" in general; as "stiff neck," "torticollis," "rheumatic cephalalgia," "brachialgia," "pleurodynia," "lumbago," "sciatica," and a host of other names depending upon the locality of the symptoms. Its indefinite aetiology and pathology, and the lack of biochemical changes, have caused many clinicians, particularly in America, to doubt its existence as a clinical entity, and it must be confessed that, considering the vague descriptions often given, combined with the confused and varied terminology, this is small wonder. Therefore we put forward a strong plea for standardization in the nomenclature and a return to the original name given by Gowers until our knowledge of the aetiology and pathology is placed on a sound basis.

Incidence

Fibrositis is one of the commonest maladies of temperate climates; it has been stated that sooner or later most individuals suffer from one or more attacks in the course of their life. It is rare in tropical and Arctic regions (Hill, 1939). Davidson and Duthie (1939) have estimated that 300,000 new cases of rheumatic disease requiring medical attention occur annually in Scotland alone; of these, 75% are of the fibrositic group. Copeman (1940) has drawn attention to the high incidence of the malady in the B.E.F.

It affects both sexes and all ages, but is commonest in middle and later life, particularly in the male (Bach, 1935). Occupation and increased liability to trauma may account for this. Exposure to damp and chill is also a factor determining its incidence, so that a large number of cases will undoubtedly occur in the coming winter as a result of the "shelter-life" necessitated by air raids.

Aetiology

The aetiology of fibrositis is unknown, and, like most conditions the cause of which is obscure, many agents have been incriminated. Among the more generally accepted