impression of the teachers was that the majority of affected pupils were sensible and emotionally stable.

As to the system affected by the infection, the sudden onset of severe frontal headache associated with vertigo and faintness tended to point to an involvement of the central nervous system rather than of the gastro-intestinal tract, and it was certainly feasible to consider the nausea to be of "central" origin. Conversely, the relatively small amount of vomiting and diarrhoea and the absence of pyrexia tended to exclude the gastro-intestinal system, especially in the absence of any association with a particular food or drink.

The duration of symptoms was rather longer than is usual in this type of condition; the slightly shorter period for boys was probably not really significant, and it may simply reflect differences between boys and girls in psychological response to illness. It was not possible to define a period of communicability, but it appeared to be greater than one week—again rather longer than is usual in outbreaks of this character. The relapse rate was very disturbing, and it was not possible to offer an explanation for this phenomenon, but certain features suggested an hysterical element.

This outbreak serves to illustrate the practical difficulties experienced by a public health medical officer in dealing in a rapid and effective fashion with "group illness" in a localized community of this nature. The initial difficulty was that of making an on-the-spot diagnosis adequate for the purposes of disposal of cases and the taking of immediate action to prevent the occurrence of further cases (it may well be imagined that a school gymnasium giving the impression of a "casualty-clearing station" is hardly an ideal situation in which to practise careful diagnostic technique). In this instance it did not prove too difficult to exclude gaseous poisoning, but this might not have been so straightforward had the school been of an older type using coal-gas and possibly with defective drains. The second problem was that of the logistics of case disposal; it was found that a substantial number of ambulance vehicles (sitting-case cars) had to be used daily, and this involved considerable rearrangement of other non-emergency ambulance duties. The third and possibly most difficult problem was

that of the public-relations aspect of the matter; the condition was soon being discussed by the public at large as the "Coventry mystery virus," but, with the help of the school staff initially and subsequently the co-operation of the press and regional television news broadcasting service, it was possible to utilize the publicity to good purpose.

Summary

An explosive outbreak affecting 404 pupils and two members of staff at three Coventry secondary modern schools is described, in which the prominent symptoms were frontal headache, nausea, and vertigo, accompanied by actual collapse.

Arguments are put forward for considering the outbreak to be of infectious (probably viral) origin, although it is admitted that hysterical elements were probably also present.

An account is given of the attempted measures of control, drawing attention in particular to the part played by exclusion and finally by school closure.

Mention is made of the practical difficulties involved in dealing with an outbreak of this type of "group illness" in a localized community.

We should like to express our gratitude to Dr. J. E. M. Whitehead, Director of Coventry Public Health Laboratory, for his valuable advice during the outbreak. We are also greatly indebted to Mr. W. L. Chinn, Director of Education, City of Coventry, for his co-operation; and to Sister Colman, Miss D. M. Parmcutt, and Mr. T. M. Hope, the head teachers of the three affected schools, for the calm yet smoothly efficient manner in which they coped with the highly dramatic and totally unprecedented situation in their schools.

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Laboratory Infection with Louping-ill Virus: a Case Study*

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Brit. med. J., 1964, 2, 1627-1630

Louping-ill is primarily a disease of sheep in the British Isles, but a few cases have been reported in man; all of them have been in laboratory workers or persons handling sheep, such as abattoir workers, in enzootic areas (Rivers and Schwentker, 1934; Davison *et al.*, 1948; Edward, 1948; Lawson *et al.*, 1949). The aetiological agent of louping-ill is a group B arbovirus closely related to the Russian tick-borne complex which also includes Far Eastern Russian spring-summer encephalitis,

Omsk haemorrhagic fever, Central European tick-borne virus encephalitis, and Kyasanur Forest disease of India. The vector for these diseases is thought to be the infected tick, although Soviet scientists have shown the Central European encephalitis may be acquired by drinking milk from infected goats (Smorodintsev *et al.*, 1954). In the case of louping-ill, natural infection in man presumably occurs after a tick-bite in persons handling tick-infected sheep. The routes of laboratory infection in most human cases have been unknown.

In several reported cases louping-ill in man has been described primarily as non-fatal meningoencephalitis with certain characteristics common to the whole group of Russian tick-borne encephalitides (Rivers and Schwentker, 1934; Davison *et al.*, 1948; Edward, 1948; Lawson *et al.*, 1949; Rivers and Horsfall, 1959). These features are: (1) abrupt onset and a diphasic course of symptoms; (2) an influenza-like initial phase marked by fever, systemic symptoms, and leucopenia; (3) a second phase characterized by central nervous system symptoms

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and leucocytosis. In Omsk haemorrhagic fever and Kyasanur Forest disease the initial systemic phase is marked by haemorrhagic manifestations and thrombocytopenia.

During an attempt to isolate Philippine haemorrhagic fever (P.H.F.) virus from Filipino patients' sera two viruses (8982 and 8983) were "isolated," and after intensive laboratory study these viruses were found to be indistinguishable from louping-ill (L.I.) virus. A technician accidentally inoculated himself with this agent while working in the laboratory. His illness was indistinguishable clinically from reported cases of Kyasanur Forest disease, but was actually caused by infection with a laboratory contaminant—the L.I. virus.

Case Report

On 11 April 1963 a technician accidentally pricked his finger while inoculating mice with P.H.F. "isolates" (8982 and 8983). On 14 April he noted malaise, and one day later had generalized muscle soreness, nasal congestion, smarting of the eyes, and fever which reached 102° F. (38.9° C.) by the evening of 15 April (Fig. 1). A local Chinese physician saw the patient, diagnosed

but symptoms of malaise, myalgia, anorexia, nausea, and insomnia continued. The petechiae gradually faded during this period and the pharyngeal ulcer healed. Repeated stool examinations were negative for blood.

On 1 May, 16 days after admission, he began to have fever again, which gradually rose to a peak of 104° F. (40° C.) on 3 May, then fell to 102° F. (38.9° C.) on 5 and 6 May. Daily physical examination failed to reveal the presence of a complicating illness. The other symptoms continued and the patient vomited several times. No petechiae were seen during this second febrile episode, and there was no evidence of bleeding tendency. The fever lasted seven days, after which the patient made an uneventful though rather slow recovery. During the entire illness there was no clinical evidence of involvement of the central nervous system or meningitis. The deep tendon reflexes were consistently brisk, especially in the lower extremities, but equal bilaterally. There were never any pathological reflexes. Muscle strength, checked frequently, remained grossly normal. The patient refused lumbar puncture.

Leucopenia occurred during the initial phase of his illness; the lowest white-cell count was 2,450/c.mm. on 19 April. From 22 to 25 April leucopenia and thrombocytopenia were present simultaneously. On 22 April very few platelets could be found in peripheral blood smears and the total count remained below 100,000

FIG. 1.—Clinical features of patient's illness, correlating symptoms, signs, and laboratory data. The biphasic nature of the illness is depicted graphically.

influenza, and administered unknown medications on 15 April. Past family and social histories and a systemic review did not contribute pertinent information except that the patient had been working in virology laboratories for three months. He had worked with yellow fever, trachoma, Japanese encephalitis, L.B.-1, Sagiyama, influenza, polio, adenovirus, E.C.H.O., and L.I. viruses.

Physical examination on 16 April revealed a Chinese male of small stature who appeared flushed and acutely ill. His blood-pressure was 126/78; pulse 108; respirations 20; temperature 102.4° F. (39.1° C.). The conjunctivae were injected and the eyelids red and slightly oedematous. The mucous membranes of the mouth were reddened and oedematous. Sinus tachycardia and suggestion of gallop rhythm were observed. Hip girdle muscles were tender on palpation. There was no rash and, at this time, no petechiae or ecchymoses.

The patient was put to bed, given a free diet, and encouraged to drink at least 3,000 ml. of fluids daily. He was febrile for the first six days, the fever gradually falling from 103 to 99° F. (39.4 to 37.2° C.) over this period. Malaise and myalgia, especially of the hip girdle muscles, continued and he began to experience nausea two days after admission. On 18 April he began to have slight epistaxis, and on 20 April a few scattered petechiae 1–3 mm. in diameter were noted on his face, neck, and forearms. On 21 April the posterior pharynx was injected and a 4 by 8 mm. ulcer was observed on the posterior soft palate. From 22 to 30 April he was free of fever

until 23 April, after which the count gradually returned to normal (Fig. 1). The thrombocytopenia was further documented by an abnormal clot-retraction test (plasma as well as whole blood), borderline low serum prothrombin time, and positive tourniquet tests. The Wintrobe (1961) screening test for abnormal blood coagulation supported the probability of a "first stage" coagulation defect as opposed to a circulating anticoagulant. On 23 April an attempt was made to detect platelet agglutinins in the patient's plasma, using the technique described by Stefanini and Dameshek (1955). The test was negative.

Examination of material obtained by sternal aspiration on 22 April showed cellular marrow, containing megakaryocytes adequate in number and appearance. Platelets were slightly re-

duced in the marrow. The myelocyte:erythrocyte ratio was 7.5:1. Serial determinations of haemoglobin, haematocrit, bleeding-time, and prothrombin time were normal. The clotting-time (Lee-White) was 12 minutes on 23 April and 14 minutes on 3 May. Red blood cell indices and morphology were normal. Haemoglobin electrophoresis revealed haemoglobin A. Urinalyses during the initial febrile episode showed one- to two-plus proteinuria and a few red cells, white cells, and granular casts. Subsequently the urine sediment became normal. On admission S.G.O.T. and S.G.P.T. were 24 and 11 units respectively; they rose to 88 and 41 units on 22 April. On 19 April an electrocardiogram showed non-specific ST and T wave changes in leads II and III when compared with entirely normal tracings taken after recovery. Bromsulphthalein retention was 12% in 45 minutes on 25 April. Total and direct bilirubin, thymol turbidity, cephalin flocculation, serum proteins, and protein electrophoretogram, alkaline phosphatase, blood urea nitrogen, and blood glucose determinations were all within normal limits. The chest x-ray picture was normal.

Isolation and Characterization of Virus from Patient's Acute Serum.—Acute blood samples were drawn on the first and second days after onset of illness. Undiluted serum and whole blood were inoculated intracerebrally (0.01 ml./mouse) into groups of 1- to 2-day-old suckling mice, and into cultures of HeLa cells¹ (0.1

¹ Originally obtained from Microbiological Associates, Albany, California.

ml./tube). Both groups of mice became moribund four to five days after inoculation, and their brains were harvested on the fifth and sixth days after inoculation. HeLa cells showed a cytopathic effect (C.P.E.) when inoculated with either whole blood or serum after incubation at 35° C. for nine days in a roller drum. When stationary incubation was used, only the cells inoculated with whole blood showed C.P.E. at first passage. Stationary incubated serum-inoculated tubes showed C.P.E. beginning six days after second-passage inoculation with nine-day fluids from the first passage. The viruses isolated from whole blood and serum were designated 9013 and 9014 respectively. The viruses were characterized by mouse and tissue-culture neutralization tests (T.C.N.T.). Mouse neutralization tests and neutralization index calculations were performed using the methods of Hammon (1956). T.C.N.T. were performed as previously described (Green *et al.*, 1963). Procedures used for the propagation, inoculation, and maintenance of HeLa-cell cultures have been described in a previous report (Green *et al.*, 1963).

Identification of Virus Isolated from Patient.—Viruses (9013 and 9014) isolated from both blood and serum of the patient were tested by T.C.N.T. against known rabbit antisera to various group B arboviruses and to 8982 and 8983, the "isolates" with which the patient was working when his accident occurred (Table I). No relation to dengue, Japanese encephalitis, West Nile, Murray Valley encephalitis, or yellow fever viruses was found. Both isolated viruses were neutralized by 8982, 8983, and L.I. antisera, indicating that the "isolates" were closely related to L.I. (8982 and 8983 were subsequently shown to be indistinguishable by complement-fixation box titration. We concluded that they were actually contaminant L.I. virus originating in our laboratory.) In Table II (mouse neutralization test) the very similar log neutralization indices

TABLE I.—Identification of Virus Isolated from Patient as Louping-ill by Tissue-culture Neutralization Tests in HeLa Cells

Virus Specimen No.	Original Specimen Source	Virus Dose TCID ₅₀	Titre*											
			Rabbit Hyperimmune Sera†											
			8982	8983	L.I.	Dengue 1	Dengue 2	Dengue 3	Dengue 4	Dengue 5	J.E.	W.N.	M.V.E.	Y.F.
9013	Blood	25	80	160	320	0‡	0	0	0	0	0	0	0	0
9014	Serum	5	160	320	320	0	0	0	0	0	0	0	0	0

* Titre expressed as the reciprocal of the geometric mean of the highest initial serum dilution preventing cytopathic effect of the virus and calculated on the basis of a 50% endpoint.

† All hyperimmune sera used in this test had been checked by mouse neutralization test with homologous virus. All had log NI of at least 3.0—that is, were capable of neutralizing at least 1000 LD₅₀ dose of virus.

‡ 0 = < 10.

J.E. = Japanese encephalitis. W.N. = West Nile. M.V.E. = Murray Valley encephalitis. Y.F. = yellow fever. T.C.I.D. = 50% tissue-culture infectious dose of virus. Four tubes were used for each dilution of antiserum.

TABLE II.—Mouse Neutralization Test of Patient's Convalescent Serum, Compared with Normal Rabbit Serum (N.R.S.), Against Viruses 9013 (Isolated from Patient), 8982, and Louping-ill

Virus	N.R.S.	Patient's Convalescent Serum	Log Neutralization Index
9013	8.62	5.00	3.62
8982	8.47	5.36	3.11
L.I.*	8.36	5.00	3.36

* The prototype L.I. virus used in this laboratory was originally obtained from Major E. Beuscher, Walter Reed Army Institute of Research, Washington, D.C. It had been through 50 sheep passages and 18 mouse passages when used by us.

indicate that viruses 9013, 8982, and L.I. are indistinguishable, and that the patient's convalescent serum contained antibodies to these viruses in significant titre. The T.C.N.T. test (Table III) shows that he had a fourfold rise in antibody titre to the agent in question. We therefore conclude that he had an infection with the L.I. virus. The acute and convalescent sera of Filipino patients suspected of having P.H.F., including that serum from which 8982 and 8983 were "isolated," had no titre to L.I. virus.

A mouse-brain suspension of virus 8983 was sent to the Rockefeller Foundation Virus Laboratories for characterization. Fig. 2 shows the results of an absorption H.I. experiment performed by Dr. Delphine H. Clarke. L.I. serum absorbed with 8983 antigen gives an identical falling titre curve with L.I. serum absorbed with L.I. antigen. These results can only indicate identity of L.I. and 8983. Antigenic distinction is now possible between L.I. and Central

European tick-borne encephalitis, Omsk haemorrhagic fever, Kyasanur Forest disease, and Far Eastern Russian spring-summer encephalitis (Clarke, 1961).

TABLE III.—Tissue-culture Neutralization Test of L.I., 8982, and 8983 Viruses

Virus	Virus Dose TCID ₅₀	Serum Neutralization Titre					
		Patient's Sera			Immune Rabbit Sera		
		Acute (4/16/63)	Conv. I (4/29/63)	Conv. II (5/6/63)	8982	8983	L.I.
8982	16	28	113	320	211	211	211
8983	32	28	40	184	160	160	57
L.I.	50	80	226	320	65	65	65

T.C.N.T. of L.I., 8982, and 8983 viruses, with acute and convalescent sera from patient. Cross-titration of 8982, 8983, and original L.I. strains are also included. The serum neutralization titre is expressed as the reciprocal of the geometric mean of the highest initial serum dilution preventing C.P.E. of the virus used and calculated on the basis of a 50% neutralizing endpoint. All sera were heated at 56° C. for 30 minutes to inactivate any virus which may have been present. Conv. = convalescent.

FIG. 2.—Absorption—HI comparison of L.I. and virus strain 8983. L.I. serum absorbed with 8983 virus.

Discussion

Case reports of louping-ill in man have never mentioned haemorrhagic manifestations as part of the clinical picture but have always included central nervous system manifestations. However, bleeding tendencies and thrombocytopenia are hallmarks of Omsk haemorrhagic fever and Kyasanur Forest disease; these diseases are caused by viruses believed to be strains of the same Russian tick-borne complex virus as L.I. (Pond *et al.*, 1953). In a report of the 1958 epidemic of Kyasanur Forest disease Work *et al.* (1959) described the following features as characteristic of their cases: (1) sudden onset of fever and/or headache; (2) severe muscular pains, neck pain (meningismus), and prostration; (3) inflammation of the scleral and palpebral conjunctivae; (4) vomiting and other gastro-intestinal symptoms; (5) haemorrhagic signs; (6) a papulo-vesicular eruption on the soft palate; (7) an occasional febrile exacerbation in the third week; (8) prolonged convalescence. It is interesting that our case manifested all of these symptoms and signs with the exception of headache and meningismus; the bleeding tendency was mild in contrast to that of the cases of Work *et al.*, and in this respect resembled more closely the patients with Kyasanur Forest disease described in 1961 by Webb and Rao.

Mild epistaxis was the only evidence of gross loss of blood; there was no gastro-intestinal bleeding on repeated stool examination, no haemoptysis, and no haematuria. Petechiae were first noticed at about the time that thrombocytopenia occurred near the end of the first febrile period. There seemed to be only one

crop of petechiae, which gradually faded. The thrombocytopenia was quite transient; a platelet count below 100,000 was present for less than 48 hours, but about five days were required for it to rise again to over 200,000. The thrombocytopenia was further documented by abnormal clot-retraction of both plasma and whole blood and borderline low prothrombin consumption times during this period. The bone-marrow examination at the same time disclosed numerically adequate and morphologically normal megakaryocytes; therefore the fault did not seem to be one of platelet production. It is our feeling that the cause of the thrombocytopenia in this setting is overconsumption of platelets, probably as a result of capillary damage, with resultant capillary haemorrhages (petechiae). Microthrombi, formed in areas of capillary damage, cause deposition of platelets as well as usage of the other coagulation factors which, if widespread enough, might result in transient depletion of all the factors concerned in blood coagulation, including platelets.

No necropsy data are available on human cases of louping-ill. Pathological material on Omsk haemorrhagic fever has suggested to Russian workers that the basic lesion involves the capillaries (Gajdusek, 1953). In a few reported human necropsies of Kyasanur Forest disease there was extensive oozing of blood into the lungs and massive gastro-intestinal haemorrhage (Work *et al.*, 1959). Both leucopenia and thrombocytopenia are seen in Omsk haemorrhagic fever and Kyasanur Forest disease. Webb and Rao (1961) noted phagocytosis of erythrocytes, leucocytes, and platelets by peripheral blood monocytes in their patients with Kyasanur Forest disease. This may be an additional explanation for the thrombocytopenia and leucopenia observed in our case.

Previous reports of louping-ill in man have stressed the variability of the clinical picture and have pointed out that either the prodromal influenza-like phase or the later encephalitic phase may be absent entirely (Rivers and Horsfall, 1959). The reports have stressed that louping-ill should be suspected whenever a person exposed to the L.I. virus acquires a febrile meningoencephalitic illness. This case illustrates that louping-ill in man need not be manifested as a central nervous system disorder.

Immunologically, a very close relationship exists among all members of the Russian tick-borne virus complex; that haemorrhagic manifestations may occur in louping-ill suggests also a clinical relationship between L.I. virus and the known haemorrhagic strains of this virus complex.

It follows from the foregoing that the diagnosis of louping-ill in man cannot be made on clinical grounds alone. We should enlarge upon this point to emphasize that in the individual patient the clinical symptomatology with most arbovirus infections is so variable that an accurate diagnosis based solely on clinical data is not possible.

Summary

The case of a laboratory technician accidentally infected with the virus of louping-ill is presented. His illness could not be distinguished clinically from Kyasanur Forest disease as described by Work *et al.* (1959) but differed from previously reported human cases of louping-ill in two ways: (1) there were no central nervous system symptoms and (2) there were thrombocytopenia and mild haemorrhagic manifestations. The implications of this clinical relationship of louping-ill to Omsk haemorrhagic fever and Kyasanur Forest disease are discussed.

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Autoimmunity and Thyrotoxicosis

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Brit. med. J., 1964, 2, 1630-1632

It is well known that various degrees of autoimmune thyroiditis, characterized by focal chronic inflammation, usually with the presence in the blood of thyroid autoantibodies, are to be found in most patients with thyrotoxicosis. In the past we, and probably many others, have assumed that the hyperplastic

thyrotoxic thyroid gland, which is very rich in the "microsomal" epithelial autoantigen, provides an abnormally strong antigenic stimulus, and that this is responsible for the commonly associated autoimmunity. However, the following three pieces of evidence have led us to doubt the validity of this assumption.

I. Familial Associations of Thyroid Disorders and Thyroid Autoimmunity

It has been shown that in some families a high incidence of thyroid autoantibodies is associated with a high incidence

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