lung abscesses. Isolated instances of direct spread of infection to the heart and embolic spread to the brain occurred, with fatal consequences.

Summary

The clinical and pathological records and radiographs of 165 patients with pneumonia complicating influenza admitted to the 10 London undergraduate teaching hospitals in the autumn of 1957 are reviewed. Of these, 20 (12%) were instances of staphylococcal pneumonia.

The non-staphylococcal cases are compared with the staphylococcal cases, together with a further 135 instances of staphylococcal pneumonia complicating influenza during the same period which were derived from hospitals in and near London, mainly in the North-east Metropolitan Region and from the three armed Services.

The severer course and higher mortality in the staphylococcal series are discussed.

The clinical assessment and emergency treatment of fulminating influenza are described, with special reference to bacteriology, choice of antibiotics, steroid therapy, and tracheobronchial intubation.

In order to enlarge experience of the severer forms of influenzal pneumonia and the less common forms of treatment, the records of 79 cases were added to the above series, making a total of 379 cases.

We record our sincere thanks to the many physicians and pathologists who made the collection of so large a series possible, particularly the medical committees of the 10 London undergraduate teaching hospitals, the Senior Administrative Medical Officer, and the medical committees of the hospitals in the North-east Metropolitan Region, and the Medical Directors General and medical officers of the Royal Navy, Army, and Royal Air Force. We are also indebted to individual physicians and pathologists at the following hospitals—Bedford General, Brompton, Buchanan, Lister, Mount Pleasant, North Herts, Royal East Sussex, Royal Northern, Schrodells, St. Andrew's, Bow, St. Helier, and St. Olave's.

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NEUROLOGICAL AND HEPATIC DISORDERS ASSOCIATED WITH INFLUENZA

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From time to time in the history of mankind influenza has taken its toll in terms of human life and suffering. The present pandemic, which appears to have started in North China in the early spring of 1957, spread to Hong Kong, and was first reported from Singapore in May. Cases started to appear in Madras in the same month, after the arrival in the port of s.s. *Rajula* from Singapore. Very soon after, other Indian ports also reported the occurrence of cases. The epidemic thence spread fanwise throughout the country, and by the end of the month most of the towns of South India were affected. The vast majority of these cases conformed to the usual clinical picture associated with influenza and described in previous epidemics (Abrahams *et al.*, 1919; Stuart-Harris, 1945). Early in this epidemic we realized that some of the cases showed unusual clinical features referable to the central nervous system and the liver. These cases were therefore studied in detail, and the observations form the subject of this paper.

Present Investigation

Out of 9,459 cases of influenza admitted to the various military hospitals in South India, a total of 30 showed the features mentioned. All the cases occurred during the peak period of the epidemic in the stations. Sixteen occurred in one station in 17 days. The usual incidence of encephalitis in this station is one or two cases a year. Five cases occurred in another station in 27 days, whereas the usual incidence of encephalitis there is about one a year.

Clinical Features.—These are shown in Table I. They do not pertain to the initial signs and symptoms of influenza but to those of the subsequent illness, during which neurological and hepatic manifestations developed. In a typical case after an attack of influenza the patient was afebrile and progressing well, when suddenly there was an onset of severe persistent vomiting followed very soon by delirium, boisterousness, and coma. Some of these cases had convulsions. In a few, localizing neurological signs were noted. A third of these cases had raised serum bilirubin. Complete recovery occurred in 13, and 17 proved fatal.

Age-and-Sex Incidence.—Age varied from about 14 to 26 years, except one case of a child aged 2 years and 9 months and another of an adult aged 36. All the patients were males. No inference can, however, be drawn from this age-and-sex incidence, since the number of cases affected was small, and the community at risk was of a similar age-and-sex group.

Onset.—As already indicated, after a typical attack of influenza there was an afebrile period varying from one to

seven days in 20 cases. In seven there was no such interval, while in the remaining three the history of respiratory infection was atypical. Vomiting, which was invariably the first symptom, was present in 29 cases and absent in only one. It started suddenly in 27 and insidiously in two (Cases 6 and 24).

Fever.—This was not a constant feature in all the cases. It was high in eight, and the rest had either a low pyrexia or were apyrexial. Headache, which was present in 12, was not a prominent symptom except in one. Some degree of toxaemia was recorded in 13 cases.

Central Nervous System.—Of all the 30 cases showing neurological involvement, 19 went into coma with or without convulsion after passing through a phase of confusion, delirium, and boisterousness. Four of the patients who developed coma ultimately recovered. Seven were only drowsy, and four others were conscious throughout. The degree of cerebral aberration did not affect the ultimate outcome. Fifteen cases showed localizing neurological signs in the form of involvement of the pyramidal tract or conjugate deviation. One patient (Case 24) developed extensive paralysis of muscles of the trunk and limbs but not of the head and neck. He was conscious throughout and died of respiratory failure.

Other Systems.—Icterus was apparent in only one patient, although serum bilirubin was raised in 10—eight died and two recovered.

Laboratory Findings

The laboratory findings are given in Table II. Total and differential leucocyte counts were carried out in all cases and were within normal limits. Urine examination showed the presence of bile in only one case. Albumin was found in 13 and sugar in four. The presence of sugar in urine, however, was not regarded as very significant, as all these cases were on intravenous concentrated glucose therapy. The serum bilirubin level was raised above 1 mg./100 ml. in 10 cases, the highest level being 4 mg. C.S.F. examination was carried out in 25, but did not show any significant abnormality. Blood-sugar estimation was carried out in 20 cases; the results were within normal limits. Blood urea was estimated in 25. It was raised above 40 mg./100 ml. in four fatal and three non-fatal cases.

Serological findings are given in Table III. In addition to Case 1, as shown in this table, influenza virus A was also isolated from lungs removed at post-mortem examination in Cases 3, 5, 17, and 28. Influenza virus A has also been isolated from the brain of Case 24. The exact relationship of this virus to virus A/Asia/57 is being studied in the Pathology Department of the Armed Forces Medical College, Poona. The serological study for influenza was inconclusive.

The H.I. and complement-fixation tests against arthropod-borne encephalitis viruses were carried out in four cases. The tests were put up against Chinkungunya and AR 339 strain of Sindbis of group A viruses and Japanese B, Murray Valley, Egypt 101 strain of West Nile, and Trinidad 1751 strain of Dengue of the group B arthropod-borne viruses. This study did not incriminate any of these viruses as the causal agent.

Treatment

In the absence of any specific therapy various lines of treatment were tried, either singly or in combination.

TABLE I.—*Clinical Features*

Case No.	Vomiting	Fever	Headache	Toxaemia	Respiratory Involvement	Clinical Jaundice	Neurological Features	Outcome
1	++	High (106° F.: 41·1° C.)	+	+	0	0	Convulsions, coma, plantars extensor	D. 3rd day
2	++	High (104° F.: 40° C.)	0	++	0	+	Delirious, comatose, plantars extensor	,, 6th ,,
3	+++	Low	+	+	+	0	Drowsy, with plantars extensor, later delirious, boisterous, and comatose	,, 3rd ,,
4	+++	,,	0	0	+	0	Drowsy, with plantars extensor Lt., later delirious, boisterous, and comatose	,, 3rd ,,
5	+++	0	0	0	+	0	Boisterous, maniacal, with plantars extensor, later comatose	,, 3rd ,,
6	+++	Low	+	0	0	0	Boisterous, maniacal, with plantars extensor and Rt. cremasteric absent, later comatose	,, 5th ,,
7	++++	,,	+	0	0	0	Drowsy, with plantars extensor, abdominals and Rt. cremasteric absent, later comatose	,, 4th ,,
8	++	,,	+	++	+	0	Delirious, boisterous, with plantar extensor Rt., later comatose, nystagmus during recovery	R. 12th day
9	++	,,	0	0	0	0	Delirious, boisterous with plantar extensor Rt., later comatose	,, 10th ,,
10	++	,,	+	+	0	0	Delirious, restless, plantars extensor, Lt. cremasteric absent, later comatose	,, 9th ,,
11	+	High	+	0	+	0	Drowsy, with vertigo	,, 6th ,,
12	+	Low	+	0	+	0	,, ,, ,,	,, 5th ,,
13	+++	0	0	0	0	0	Conscious throughout, plantarex tensor Rt. till 8th day	,, 8th ,,
14	+	Low	+++	+	0	0	Conscious throughout, plantar extensor Lt. for 2 days	,, 5th ,,
15	+++	0	0	0	+	0	Drowsy till 5th day, giddy till 8th day	,, 8th ,,
16	+	0	+	0	0	0	Drowsy and giddy for 4 days	,, 4th ,,
17	+++	Moderate (102° F.: 38·9° C.)	0	++	0	0	Convulsions, with conjugate deviation, later comatose, with plantars extensor and alternating spasticity and flaccidity of Rt. and later Lt. side	D. 6th ,,
18	++	,,	0	+	0	0	Confused and restless, but conscious, with plantars extensor	R. 6th ,,
19	+++	High	+	+	+	0	Confused and restless	,, 6th ,,
20	++	,,	0	++	0	0	,, ,, ,,	,, 6th ,,
21	++	Low	++	++	+	0	Comatose, plantars extensor	,, 9th ,,
22	0	High (106° F.: 41·1° C.)	0	0	0	?	Comatose	D. 3rd ,,
23	+	Moderate (101° F.: 38·3° C.)	0	0	0	0	Conjugate deviation with jactitation	,, 1st ,,
24	++	0	0	0	0	0	Sudden weakness of respiratory muscles and rest of the body except head and neck. Died of respiratory failure	,, 2nd ,,
25	+++	Low	0	0	0	0	Boisterous, later comatose	,, 2nd ,,
26	++	High	0	++	+	0	Giddy, delirious, boisterous plantar extensor Rt., later comatose	,, 6th ,,
27	+	Moderate (101° F.: 38·3° C.)	0	+	0	0	Boisterous, maniacal, with conjugate deviation, coma, and convulsions	,, 3rd ,,
28	++++	High	0	0	0	0	Delirious and boisterous, later coma, and convulsions	,, 5th ,,
29	++	0	0	0	0	0	Confused, restless, later comatose	,, 2nd ,,
30	++	Low	0	0	0	0	Delirious and boisterous, later comatose	,, 3rd ,,

0=Nil. +=Slight. ++=Moderate. +++=Severe. ++++=Gross. D.=Died. R.=Recovered.

Eighteen cases were treated with heavy doses of intravenous vitamins and concentrated glucose. Tetracycline or chlortetracycline in full doses, either parenterally or orally, was administered to 22 cases. Hydrocortisone parenterally, 100 mg. eight-hourly, was used in four cases. Penicillin alone was administered to five cases. It may be reiterated that some of the patients received a combination of vitamins, tetracycline, and hydrocortisone, whereas others received one or two of these products only. No conclusion could be drawn about the effectiveness of any of the treatment given in the present series of cases.

Post-mortem Appearances and Histopathology

All the 17 patients who died were subjected to post-mortem examination, which was performed 2½ to 25 hours after death. Special attention was paid to the lungs, brain, and liver, since all these cases had a history of influenza, and had cerebral symptoms with neurological signs and clinical or biochemical evidence of jaundice.

TABLE II.—*Laboratory Findings*

Case No.	Urine		Serum Bilirubin		C.S.F.	
	Bile	Misc.	Day of Illness	mg./100 ml.	Protein mg./100 ml.	Cells/ c.mm.
1	0	Alb.+ Gr. casts	2	1·25	20	2
2	0	Alb.+	4	3	20	0
3	0	0	3	0·6	30	2
4	0	0	2	0·6	30	3
5	0	0	2	4	30	2
6	0	0	3 5	1·75 3·5	30	3
7	0	Alb.+	3	1	30	3
8	0	Sgr.+	3 8 12	0·4 1·75 0·4	40	3
9	0	0	2 4 10	2 1·5 0·4	35	2
10	0	Alb. trace Sgr.+	3	0·4	30	4
11	0	0	2 5	0·8 0·4	35	4
12	0	0	2	0·2	30	2
13	0	0	3	0·4	30	2
14	0	0	2	0·6	30	4
15	0	0	1	0·4	30	2
16	0	0	—	—	30	3
17	0	Alb.+ Sgr.+	—	—	—	3
18	0	Alb. trace	3	0·3	25	—
19	0	0	3	0·2	—	—
20	0	Alb.+	3	0·2	20	0
21	0	Alb. trace	—	—	—	—
22	0	Alb.+	1	2	50	8
23	0	0	—	—	25	3
24	—	—	2	0·2	—	—
25	0	Alb.++ Gr. casts	—	—	70	4
26	0	Alb. trace Sgr.	5	0·8	30	4
27	0	0 ,,	3	0·2	—	—
28	0	0	5	3	—	—
29	0	Alb.+ PC.+ R.B.C.+	2	4	30	2
30	+	Alb.+ PC.+ R.B.C.+	3	1·5	40	—

Alb.=Albumin. Gr.=Granular. Sgr.=Sugar. PC.=Pus cell. —=Not done. 0=Absent.

TABLE III.—*Serology*

Case No.	Influenza H.I. Titre						Isolation of Virus
	F.M.I.		B6		New Virus		
	1st Sample	2nd Sample	1st Sample	2nd Sample	1st Sample	2nd Sample	
1	256	—	—	—	Neg.	—	+
6	—	256	—	128	—	0	—
8	—	256	—	2,048	—	32	—
9	128	128	32	64	8	16	—
11	128	256	32	32	0	0	—
18	128	128	—	—	0	16	—
19	0	0	128	128	0	0	—
20	256	2,048	—	—	0	0	—
21	256	256	—	—	0	8	—

F.M.I. strain=Virus A. B6 strain=Virus B. New virus=A/Asia/57. —=Not done. 0=Nil.

Lungs.—Naked-eye appearances varied from an intense congestion to patchy haemorrhagic pneumonia. Histologically the general picture was a complete denudation of the ciliated epithelium of the mucous membrane of the bronchial tree. Haemorrhagic consolidation was also present in many of these cases. Case 24, from whose brain influenza virus A was isolated, showed the characteristic bronchial mucosal change without any pulmonary involvement.

Brain.—Naked-eye appearances varied from moderate to severe congestion with petechial haemorrhages in the white matter in all cases, and histology confirmed the presence of diapedesis of cells into the Virchow–Robin space in some. In a few cases neuroglial proliferation was also present. Case 24 showed evidence of neurone degeneration, including satellitosis and neuronophagia.

Liver.—On naked-eye examination no specific change was noticed. Appearances varied from a normal to a mild congestion or a patchy yellowish discoloration. Histologically there was evidence of fatty change not confined to any zone. In 12 out of 17 fatal cases there was evidence of portal or periportal round-cell infiltration. In the majority of cases there was no necrosis of liver cells.

Discussion

In association with various epidemics and pandemics of influenza, respiratory and abdominal complications have been described. Of these the former have been responsible for most of the fatalities. To our knowledge the neurological and hepatic features manifested by our patients have so far not been recorded in the literature, including reports of the recent pandemic (Lim *et al.*, 1957; Holland, 1957; Smith *et al.*, 1957). Non-specific non-localizing cerebral symptoms have been described by many (Cole, 1918; Benjafield, 1919; Abrahams *et al.*, 1919). Edmundson and Hodgkin (1957) have also mentioned facial neuralgia and peripheral neuritis as complications or as possible concomitant diseases with influenza.

All the cases which showed the unusual neurological or hepatic manifestations occurred during the height of the influenza epidemic in the various localities. Every one of them had a preceding influenza-like illness. In the fatal cases incontrovertible evidence of respiratory infection with influenza virus was present. These facts would indicate that the unusual features are in some way related to the influenza infection. Hyperbilirubinaemia was not invariably present, and where serum bilirubin levels were abnormal they were not very high. Cholaemia could not therefore account for the neurological manifestations. Besides, the type of neurological signs seen have not, in our experience, been associated with liver-cell failure. Apart from coma, convulsions, etc., localizing features like pyramidal signs were present in both comatose and non-comatose patients. These facts would indicate that we are here dealing with an encephalitic syndrome.

The clinical manifestations of these patients do not resemble those of the sporadic or epidemic cases of encephalitis reported from Jamshedpore (Chari and Swamy, 1955) or from Lucknow (Kaul, 1955), nor do they conform to the features of arthropod-borne encephalitis (Webb and Pereira, 1956). Serology also excludes the latter group. Such aetiological factors as measles, mumps, and vaccination were not responsible for the abnormal neurological signs.

Although the post-mortem examination (and virus culture) and histopathology have confirmed the presence of respiratory infection with influenza virus, the morbid appearances both of the liver and of the nervous system are variable and indefinite. They have been recorded here for the sake of completion, but it must be mentioned that no definite inference can be drawn from them. Influenza virus A has been isolated from the brain in one patient (Case 24), who also had marked histopathological changes in the C.N.S. Influenza virus has not, to our knowledge, been

isolated from the brain so far, and its significance cannot therefore be assessed without further study. This is being carried out in the Pathology Department of the Armed Forces Medical College, India. It is difficult to state whether the neurological and hepatic syndromes were caused by a new mutant of influenza virus, by the activation during the influenza epidemic of an associated neuro-viscerotrophic virus, or by a hybridization of viruses of the type produced by Burnet (1957) in his experimental genetic studies on viruses.

Summary

A short account of the origin and spread of the pandemic of influenza of 1957 is given. In India cases started appearing in Madras in May, 1957, and very soon after it spread to the rest of South India.

Out of a total of 9,459 cases of influenza admitted to military hospitals in South India, 30 developed manifestations of neurological and hepatic involvement. Sixteen of these were reported from one station in 17 days and five from another in 27 days.

These manifestations usually developed after an afebrile period following a typical attack of influenza.

They were ushered in by incessant vomiting followed by delirium, boisterousness, and coma. Half the patients showed localizing neurological signs; raised serum bilirubin was present in one-third.

All the 17 fatal cases were subjected to post-mortem examination. Respiratory system showed appearances typical of influenza. Influenza virus was isolated from the lungs in five of them. The liver showed a variable and indefinite picture. The brain showed congestion and diapedesis of cells. In one case neurone degeneration was present and influenza virus A was isolated from the brain substance.

Serological tests did not indicate an infection with arthropod-borne encephalitis viruses.

From the evidence presented it appears probable that the clinical manifestations of neurological and hepatic involvement are closely related to infection with influenza virus and are of a type not so far described in any other form of encephalitis reported from India. The significance of the morbid anatomy and histology and the exact causal relationship of the influenza virus to the syndrome are, however, not clear.

Our thanks are due to the medical officers who helped in the management and follow-up of the cases and to the officers and staff of the Pathology Department of the Armed Forces Medical College, Poona, for their help in the investigation of cases and examination of post-mortem material. We acknowledge the help and co-operation of the Virus Research Centre, Poona, in undertaking serological studies. Our thanks are also due to Dr. I. G. K. Menon, Deputy Director, Pasteur Institute, Coonoor, for his co-operation. We are grateful to various officers commanding military hospitals in Southern Command, D.M.S. Army, and the D.G. A.F.M.S., for permission to publish the report.

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ASIAN INFLUENZA (1957) IN ALLERGIC PATIENTS

BY

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The clinical, epidemiological, and pathological aspects of Asian influenza have already been described (*Brit. med. J.*, 1957; Smith *et al.*, 1957; Swee and Dourado, *Lancet*, 1957; Roberts, 1957; Zhdanov *et al.*, 1957; Fry, 1958; Martin, 1958).

Altogether I treated 344 cases of influenza in general practice during the epidemic in 1957, the first case being seen on August 25, 1957, and the last on November 3, 1957. The practice is an industrial one with 5,560 patients, and I am one of three partners. It was estimated that there were 216 allergic patients in this practice (Hamilton and Bendkowski, 1954). The number of allergic patients has risen with the increase in the size of the practice, and there are now about 250. I treat most of these, and have had an opportunity of studying the clinical course of Asian influenza among them.

Classification

The 344 influenza patients were divided into three groups: (1) allergic (124 cases); (2) non-allergic, but who came from allergic families (59 cases); and (3) non-allergic (161 cases). The distribution of allergic diseases in patients suffering from influenza is shown in Table I.

TABLE I.—*Severity of Symptoms Among the* 344 *Patients*

Allergic Disease	No. of Cases			Total
	Very Severe	Severe	Mild	
Allergic Patients				
Bronchial asthma ..	49	4	2	55
Allergic rhinitis ..	20	8	2	30
Respiratory allergy	9	2	2	13
Urticaria and angio-neurotic oedema	7	7	6	20
Atopic eczema ..	2	1	—	3
Contact dermatitis ..	1	1	1	3
Total ..	88	23	13	124
Other Patients				
With family history of allergy	12	25	22	59
Non-allergic ..	27	45	89	161

Findings

Each patient was examined and assessed according to severity of symptoms. This was judged by temperature, headaches, aches and pains, dizziness, vomiting, cough, dyspnoea, sweating, and prostration. Table I shows the severity of symptoms among the allergic patients and among the others.

TABLE II.—*Main Finding on Physical Examination*

Clinical Finding	No. of Cases		
	Allergic	With Family History of Allergy	Non-allergic
Laryngitis	15	29	105
Bronchitis	48	28	48
Bronchopneumonia ..	11	1	5
Lobar pneumonia ..	3	—	3
Bronchitis with asthma ..	47	1	—
Total	124	59	161