INFLUENZA "A": AN ACCOUNT OF A MINOR EPIDEMIC

BY

T. H. DONNELLY, M.B., B.S.

Flight Lieut., R.A.A.F.

H. P. HUGHES, M.B., Ch.B.

Flying Officer, R.A.F.V.R.

D. ROBERTSON, M.B., B.Ch.

Flying Officer, R.A.F.V.R.

AND

ELLIOT PHILIPP, M.A., M.R.C.S.

Squadron Leader, R.A.F.V.R.

On March 30, 1943, a Canadian airman went sick with symptoms corresponding to those known to the lay public as "influenza." He was tired and had slight cough and headache; eye movements were painful and he felt hot and then shivery. On examination his temperature was 102° F., pulse 84, and respirations 18. There was slight pharyngeal inflammation, but no glandular enlargement or nasal catarrh; lungs, heart, and all other systems were normal. He was put to bed. His temperature remained above 101° F. for 36 hours, and then dropped to normal. The patient was fit for discharge within four days of admission.

On April 3 an English officer reported sick with similar symptoms and signs. No special investigations were made, and after four days in bed he was perfectly fit again.

On April 14 an airman reported sick. Symptoms were similar to the previous two cases except for slight harshness in breath sounds at the base on the right side. Temperature was normal after 60 hours in bed. On the third day of illness slight pain was felt in the right side of his chest; breath sounds were diminished, but temperature became quite normal. After two days, as the patient did not seem to be making quick recovery in spite of the normal temperature, he was sent to hospital for x-ray, which showed tuberculous infiltration in the right apex, and this result was confirmed by tuberculosis-positive sputum.

On April 16 a W.A.A.F. reported with a temperature of 101° F. She had headache, a slight cough, and a sore throat. Put to bed, she recovered in three days. On the next day an airman reported with similar signs and symptoms, which also cleared in three days; but from that date cases occurred in increasing numbers, as shown on the graph of incidence of the epidemic (Fig. 1). Ten cases were reported on April 21;

FIG. 1.—Curve of incidence of the epidemic.

it then appeared obvious to us that an epidemic of a virus infection was occurring, and it seemed likely that the infection might be one of true influenza. Investigations were carried out, and on April 23 some of the 30 patients then in bed were picked out at random for (1) chest radiography; (2) differential blood count; (3) throat swab; (4) blood culture; (5) sputum examination; (6) titrations of antibodies in blood against influenza viruses A and B.

By this date the patients being admitted had much severer symptoms and very definite signs. They felt too ill to report sick, and several were found lying on their beds in their quarters feeling very poorly. In most cases headache was

severe; there were slight soreness of the throat, nasal catarrh, and some nausea. There was much sweating with shivering between times, and also a variety of severe signs and symptoms which we discuss below.

On examination temperatures were found to be between 102° and 103° F.; the eyes were very red and the faces flushed, suggestive of early morbilli, with, in some cases, enlargement of cervical and submaxillary glands. Tongues were furred, pharynges injected (but with no membrane) and in some cases of a violaceous tinge. In the chest, post-tussive "sticky" crepitations were heard in all areas. Breath sounds were harsh and vesicular in type, with prolonged expiration. Low backache was a feature of most cases. The urine was normal, except for traces of albumin in one or two cases. Of the 12 chests x-rayed, seven had no abnormality. Only one showed marked congestion with some consolidation at the right base.

The differential white blood cell count in a typical case was: total white cells, 5,300 (polymorphs 71.5%, lymphocytes 23%, monocytes 5%, eosinophils nil, basophils 0.5%). The highest total white cell count was 7,600 (polymorphs 54.5%, lymphocytes 31.5%, monocytes 12%, eosinophils 1%, basophils 1%). Throat swabs in most cases showed moderate growths of pneumococci, *Strep. viridans*, *N. catarrhalis*, or *Strep. haemolyticus*. Blood cultures revealed nothing. Sputum examinations disclosed little of interest except in the case with tuberculosis.

Titrations of antibodies against influenza viruses A and B by the Hirst method (inhibition of the agglutination of fowl red cells by influenza virus) gave these results:

Case	Sample	Virus	
		A	B
1	I	1/32	1/32*
	II	1/192	1/64
2	I	1/48	1/16*
	II	1/384	1/32
3	I	1/256	1/96
	II	1/256	1/96
4	I	1/96	1/64
	II	1/128	1/32
5	I	1/128	1/32*
	II	1/1024	1/32
6	I	1/64	1/16*
	II	1/1024	1/16

* In each case blood sample I was taken within two or three days of the onset of the disease, and blood sample II about 14 to 20 days later.

Dr. C. H. Andrewes, F.R.S., of the Medical Research Council, under whom Miss K. C. Mills so kindly did the tests, reported: "No case showed a significant change in antibody level against B, whereas four showed a good rise against A. (These have been marked with an asterisk.) As to the diagnosis of Cases 3 and 4, it has been noticed in numerous outbreaks, both in the United States and over here, that cases giving no evidence of infection with influenza A virus turn up in the same outbreak as virus A infection. It has been suggested that when influenza hits, as it sometimes does, a patient who already has good A antibodies the infection may cause no further rise. This may have been the case with Case 3." Dr. Andrewes (1942) has discussed the question in an address.

On April 24 only one case was admitted, but April 27 brought the "crest" of the second wave of the epidemic. Cases admitted after that date became progressively less severe, and only one or two were admitted after May 12.

Discussion

Although only 68 cases actually reported sick it is known that there were many airmen who felt "a bit off colour" during the period of the epidemic and who probably suffered from a "subclinical" attack of influenza A. Of our severe cases, one patient showed chest complications at a very early stage, although by portable x-ray findings no pneumonia was actually present. Clinically his case seemed to be quite a definite pneumonia, and was treated as such. Another patient showed as his first severe sign marked and persistent irregularity of the heart rhythm. He was treated with "absolute rest" for three days, at the end of which time normal rhythm without adventitious signs had been re-established. Severe low backache was experienced by two or three patients, and had there not been an epidemic of influenza this might easily have

been mistaken for a renal condition. We would like to point out that had we seen the case with the bad chest or the case with irregularity of the heart or those with the very severe backache at any other time than during an obvious epidemic we would have sent them into hospital under varying diagnoses, and thus almost certainly have spread the epidemic.

We consider that the period of incubation is between 24 and 48 hours, but it may be as high as 96 hours.

Treatment

All patients were placed on a fluid diet on the first day of the disease and all received diaphoretic mixtures. The diet was increased each day, and the diaphoretics were continued until the patients were fit to leave bed. Cough, where troublesome, was treated with a codeine linctus.

Sulphapyridine treatment was resorted to for all patients whose temperature had risen above 103° F. The dosage was 2 g. as an initial dose and 1 g. four-hourly. No patient had more than 8 g., and the temperature was normal in each case before that dosage had been taken. It did appear as though the sulphapyridine had definitely assisted the drop in temperature. The literature gives conflicting reports on the efficiency of sulphapyridine in influenza A, but it seems logical that in a disease in which the temperature is high owing to virus infection and in which most throat swabs demonstrate the presence of pneumococci or streptococci the use of sulphapyridine or some other drug of the group in severe cases is indicated.

The weakness that so many writers describe after influenza was not apparent in this series of cases, and all patients were fully fit seven days after leaving bed. These seven days were spent doing light duties only. There were no serious complications, except in the case proved to have tuberculosis. It may well be that had this patient not developed influenza his tuberculosis would not have been brought to light at the early stage at which it became apparent.

As stated by many writers, the epidemic occurred in two waves; and we noticed also that the curve of incidence of the cases was not dissimilar in shape to an average temperature curve of the severer cases (Fig. 2). It was observed, too, that

FIG. 2.—Average temperature curve.

the epidemic began in warm dry weather with a mild breeze which persisted from April 14 to April 21. The weather then deteriorated, and there was some rain; but on the 24th it improved, and became quite warm by the 26th. It may be that, somehow, the weather had an effect on the incidence of the disease, as rain fell again on April 28 or 29.

The ultimate object, of course, of the study of any outbreak is to indicate some way of preventing another occurring and, in the case of influenza, especially of preventing such a catastrophe as the 1918-19 epidemic. We can only state that, in our opinion, isolation is of the very greatest importance. Every case of even mild cold that occurs during a known epidemic should be rigidly isolated. By far the greatest proportion of our cases occurred on a satellite aerodrome, and all cases from that aerodrome were taken to the main aerodrome to be nursed in houses separated from all the other buildings on the camp. The only patients reported from the main camp after April 18 were medical personnel and ambulance drivers. Approximately half the medical personnel looking after the cases developed influenza. These medical personnel did not leave sick quarters or associate in any way with other members of the camp. It is probably for this reason that no cases occurred among the thousands of airmen on the main camp.

So far as other preventive methods go, it would certainly be worth while trying experimentally to vaccinate large bodies of men against influenza A a few months before the reckoned time (if it is possible to reckon!) when another epidemic should occur. The work of Horsfall (1939, 1941) in this connexion is especially interesting.

Sterilization of the air by ultra-violet light and propylene-glycol spray has been attempted elsewhere, but in view of our findings that wet weather coincided with the decrease in the waves of the epidemic it may be that simple moistening of the air would have some beneficial effect.

Summary

An account is given of a small epidemic of influenza A.

Indications as to the treatment of such an epidemic when occurring on a military camp are given.

The importance of bearing in mind the possibility of influenza when certain symptoms not normally "textbook" arise, as well as the necessity for excluding tuberculosis, is stressed.

Our thanks are due to Dr. C. H. Andrewes and Dr. A. Morton Gill for the very kind advice and help given, as well as to Wing Commander Hudson, Wing Commander Church, Squadron Leader Wynroe, Squadron Leader Campbell, and our own sick-quarters staff for their great assistance.

BIBLIOGRAPHY

- Adamson, J. D., and Fleet, R. O. (1942). *Canad. med. Ass. J.*, **46**, 121.
 Andrewes, C. H. (1942). *Proc. roy. Soc. Med.*, **36**, 1.
 — et al. (1941). *Lancet*, **2**, 387.
 DeCoursey, C., and Thuss, O. (1941). *Ohio State med. J.*, **37**, 964.
 Emdin, W. (1942). *S. Afr. med. J.*, **16**, 101.
 Gill, A. Morton (1938). *British Medical Journal*, **1**, 504.
 Hirst, G. K. (1942). *J. exp. Med.*, **75**, 49.
 Horsfall, F. L., jun. (1939). *Ibid.*, **70**, 209.
 — et al. (1941). *Ibid.*, **73**, 335.
 Lush, D., Stuart-Harris, C. H., and Andrewes, C. H. (1941). *Brit. J. exp. Path.*, **22**, 302.
 Martin, W. P., and Eaton, M. D. (1941). *Proc. Soc. exp. Biol.*, N.Y., **47**, 405.
 Price, F. W. (1941). *Textbook of Medicine*, London.
 Smith, W., Andrewes, C. H., and Laidlaw, P. P. (1933). *Lancet*, **2**, 66.
 Stokes, J., and Henle, W. (1942). *J. Amer. med. Ass.*, **120**, 16.
 Stuart-Harris, C. H. (1941). *J. R.A.M.C.*, **77**, 123.

ESTIMATION OF HAEMOGLOBIN BY PHOTO-ELECTRIC ABSORPTIOMETERS

BY

J. M. PETERSON, D.Sc.

Lecturer in Experimental Physiology, the Physiology Institute, Cardiff
AND

D. H. STRANGEWAYS, M.A., Ph.D.

Assistant Lecturer in Histology and Embryology, University College, Cardiff

Changes in intensity of light and in the electric properties of the photo-cell have been advanced as sources of error in the estimation of haemoglobin by means of photo-electric absorptiometers (M.R.C. Report, 1943). This objection to a method which at the moment appears to offer the best means of accurate estimation of haemoglobin may be justified when it refers to absorptiometers which employ a single photo-cell and which indicate the light-absorbing power of the solution by the extent of deflection of a galvanometer. When, however, a null technique is employed with an apparatus incorporating two opposed photo-cells, error due to these changes has been found, in practice, to be eliminated. The procedure by which such error is avoidable can be simply stated without detailed description of method.

Outline of Procedure

The concentration of haemoglobin is measured in terms of the width to which a slit has to be adjusted to pass through the haemoglobin solution that quantity of light which gives a definite output of current from the photo-cell on which the transmitted light is focused. With the light to one photo-cell passing both through the filter whose light absorption in terms of haemoglobin is known and through the adjustable slit set at the haemoglobin value of the filter, the light to the other cell is varied to give a null reading on the galvanometer. The standard filter is then replaced by the blood solution to be measured and the light adjusted by means of the slit till the null reading is again obtained. On replacing the standard filter and resetting the slit to the filter value the instrument should give the null reading. If there has