degeneration, and not to an acute toxic change. The slight degree of fatty degeneration of the myocardium has already been quoted as evidence of toxæmia. It might, however, be argued that the degeneration was the sequel and not the precursor of the heart failure. The short duration of the heart failure, as estimated by the microscopical as well as the macroscopical changes in the organs, does not support such an argument. The thrombosis of the left femoral vein may either have been secondary to the heart failure or it may have been caused by the action of the psittacosis virus. Some support for the latter view lies in the finding of extensive pulmonary thrombosis by Turnbull (1930) in the first case of this disease reported from the London Hospital.

The conclusion drawn from the examination as a whole is that death was caused by a haemorrhagic bronchopneumonia, acting upon a subject in whom the cardiovascular system was already greatly impaired by degeneration.

The inflammation of the lung resembled that described in the first case of psittacosis examined in this hospital in the abundance of albuminous and haemorrhagic exudate and the presence of conspicuously large desquamated epithelial cells; but it differed in being a bronchopneumonia rather than a pneumonia, in the almost complete absence of fibrinous exudate, in the absence of capillary and arterial thrombosis, and in the presence of bacteria in great number and of great variety. It was also complicated by acid digestion.

In impression preparations made from the spleen and stained by Giemsa the elementary virus bodies described by Levinthal, Lillie, and Coles were found.

NOTE ON THE BACTERIOLOGICAL FINDINGS IN THE ABOVE OUTBREAK OF PSITTACOSIS

BY

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The material received for examination consisted of: (1) blood from patient No. 2; (2) a budgerigar (*Melopsittacus undulatus*) belonging to patients Nos. 1 and 2; and (3) organs obtained post mortem from patient No. 3.

Blood of Patient No. 2.—This was collected in an equal amount of 2 per cent. citrate on November 3rd, 1930, the seventh day of disease. It was received in the laboratory November 5th, and proved to be free from cultivable bacteria. Two mice, M. 235 and M. 236, were inoculated with this citrated blood, each animal receiving 0.5 c.cm. subcutaneously and 0.5 c.cm. intraperitoneally. One mouse, M. 236, died on the thirty-fifth day after inoculation, the other was killed when very ill on the thirty-sixth day. The post-mortem findings were in keeping with the death having been due to the virus of psittacosis. The spleen of Mouse 236 was suspended in 5 c.cm. tyrode and proved to be free from cultivable bacteria. Two mice, M. 255 and M. 256, inoculated intraperitoneally with 0.5 c.cm. of this suspension on December 11th, are still alive and well, February 23rd, 1931.

The Budgerigar B. 157.—This bird, belonging to patients Nos. 1 and 2, and nursed by patient No. 1 before her own illness, had been suffering from diarrhoea, and though it was in fair condition when seen by Dr. Sturdee of the Ministry of Health, November 3rd, he decided to have it killed and examined for the presence of psittacosis virus. Post-mortem examination of the bird showed injection of the intestines, a chamois-leather liver (fatty change), and enlargement of the spleen. The lungs appeared normal. The spleen and a small portion of liver—0.25 gram in all—were suspended in 5 c.cm. N/50 phosphate pH 7.6. The suspension was culturally sterile, and two mice were

inoculated with it. The results obtained in this primary transmission experiment and subsequent passages are given in Table I.

TABLE I

Passage	Material	Mouse	Result
Primary transmission	Suspension of spleen and liver of budgerigar in phosphate; unfiltered; culturally sterile	M. 233	Killed, moribund, tenth day
		M. 234	Killed, very ill, tenth day
1st passage	Spleen of M. 233 suspended in 10 c.cm. tyrode; filtered Chamberland L1 bis	M. 241	Died seventh day.
		M. 242	Killed, moribund, seventh day
2nd passage	Spleen of M. 242 suspended in 5 c.cm. tyrode; unfiltered; culturally sterile	M. 249	Died fifth day
		M. 250	Killed, moribund, fifth day
3rd passage	Spleen of M. 250 suspended in 5 c.cm. tyrode; unfiltered; culturally sterile	M. 257	Killed, moribund, third day
		M. 258	Ditto
4th passage	Spleens of M. 257 and M. 258 suspended in 10 c.cm. tyrode; unfiltered; culturally sterile	M. 261	—
		M. 262	Killed, very ill, fourth day
5th passage	Spleen of M. 262 suspended in 5 c.cm. saline; spleen had been kept frozen from Dec. 23rd, 1930, to Feb. 10th, 1931; unfiltered; culturally sterile	M. 275	Killed, very ill, fourth day
		M. 276	Ditto
6th passage	Spleens of M. 275 and M. 276 suspended in 10 c.cm. saline; unfiltered	M. 281	Killed, very ill, fourth day
		M. 282	Died fourth day
7th passage	5 per cent. suspension of spleens; M. 281 and M. 282 in saline; unfiltered; culturally sterile	M. 287	Ill, second day, still under observation
		M. 288	Ditto

Post-mortem Material from the Nurse (Patient No. 3).—This consisted of lung, liver, and spleen obtained at the post-mortem examination carried out by Dr. Dorothy Russell. A 5 per cent. suspension in tyrode made from the pooled organs gave a copious growth of a variety of organisms accounted for by the presence of numerous secondary invaders in the lung. This suspension was filtered through a Chamberland L1 bis candle, and two mice, M. 253 and M. 254, were inoculated with the filtrate, each receiving 1 c.cm. intraperitoneally. Both mice became ill ten days after inoculation. One, M. 254, died on the twentieth day, the other recovered. The spleen of M. 254 was suspended in 5 c.cm. N/50 phosphate pH 7.6, and proved to be free from cultivable bacteria. With this suspension further mice were inoculated, with a fatal result, and the strain has now been carried to the tenth generation in mice. This strain rapidly increased in virulence, and from the fifth passage onwards killed mice in two days. This is a point of some interest, for the great majority of human strains of psittacosis virus never attain any high degree of virulence and are difficult to maintain.

Identification of the Strains of Virus by the Neutralization Test.—The death of the two mice inoculated with the blood of patient No. 2 was in all probability due to psittacosis virus, but positive passage was not obtained; so this point remains unsettled. The two strains of virus, B. 157 and "Bangor" obtained from the budgerigar and the organs of the nurse (patient No. 3), were almost certainly psittacosis virus; but in order to settle this point beyond dispute the effect of neutralization with a specific serum was determined. The serum employed was one produced in mice, M. 176, M. 177, and M. 178, with a strain of virus "G.B." isolated from budgerigars. Previous work (Bedson and Western, 1930) had shown that this serum neutralized not only the homologous strain, but a strain isolated from a parrot and one from a Java sparrow as well. Mixtures of serum and virus (spleen suspension in saline) were made in which the final concentration of serum was 1 in 100, and the concentrations of virus 1 in 1,000 and 1 in 10,000. These mixtures were allowed to stand at room temperature and then inoculated intradermally in the guinea-pig in a dose of 0.2 c.cm. At the same time titrations of the two viruses were made in the same animal. The results of this experiment are given in Table II. The signs denote the degree of reaction in the guinea-pig's skin.

The strain "Bangor" is obviously the stronger, and probably the end point was not reached, but the strain B. 157 would certainly have gone no further than the 1 in 10,000 dilution. The serum therefore neutralizes approximately 100 m.l.d. of each strain of virus, a finding in

keeping with previous titrations of this serum with other strains of psittacosis virus (Bedson and Western, 1930).¹ Thus proof is forthcoming that the two strains B. 157 and "Bangor" are immunologically identical, and that they are strains of psittacosis virus.

TABLE II

Virus	Treatment	Dilutions of Virus		
		1 in 100	1 in 1,000	1 in 10,000
B.157 ...	Virus in saline (titration)	+	±	±
	Virus with immune serum M.176, M.177, and M.178	-	-	-
Bangor	Virus in saline (titration)	++	+	±
	Virus with immune serum M.176, M.177, and M.178	±	-	-

CONCLUSIONS

Certain points arise out of the consideration of these cases. First, psittacosis is still liable to occur in this country, and should be borne in mind when one or more patients in a household are found to be suffering from an influenzal type of illness, with early signs in the lungs and perhaps some typhoid-like symptoms, such as epistaxis, abdominal distension, vomiting, constipation, or diarrhoea. Especially should such a diagnosis suggest itself if influenza is not prevalent in the surrounding neighbourhood at the time. Secondly, parrots are not the only birds which cause psittacosis in man. In the report of the Ministry of Health,² lovebirds, thrushes,

and canaries, among other birds, are stated to have been the cause of human illness. It is apparently not certain how many types of birds suffer from diseases communicable to man, so the fact that a patient keeps birds should probably always arouse suspicion in doubtful cases of human illness. Certainly all bird keepers should take care always to wash their hands after attending to the birds, and especially before taking food. Thirdly, an unusual factor in the cases described in this article is that three generations of human psittacosis followed from the original sick budgerigar. Cosman, quoted in the Ministry's report, mentions a woman who caught psittacosis from a patient she nursed and afterwards infected her own child, but such a sequence is sufficiently unusual to be worth noting. Fourthly, the infection of a trained nurse in the course of her ordinary duties shows the care required in dealing with psittacosis, and incidentally, therefore, the necessity of early diagnosis of the disease. The nurses attending on these patients were warned to treat them like cases of typhoid, and this is probably all that can be done. So far as is known, this is the first instance in this country of the infection of a hospital nurse with psittacosis by her patient, and, even so, two out of the three nurses in attendance escaped the disease.

REFERENCES

- ¹ Bedson, S. P., and Western, G. T.: *Brit. Journ. Exper. Path.*, 1930, ii, 502.
- ² *Public Health and Medical Subjects*, No. 61, H.M. Stationery Office.

A CASE OF CEREBELLAR ABSCESS

BY

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Cerebellar abscess is not a very rare condition, and recovery occurs after operation in a small percentage of cases. It is thought, however, that the following case may be of sufficient interest to report, on account of its dramatic course, and also because of important considerations of treatment that it raises.

CLINICAL HISTORY

On January 8th, 1931, Dr. A. Moscrop Walker of Wallasey asked me to see, in consultation, a girl, E. M. T., aged 13, who had acute middle-ear suppuration, with involvement of the mastoid. I was informed that the ear infection had set in suddenly at a boarding school a fortnight previously, and that there had been no antecedent ear trouble. The mastoid symptoms were only of a few days' duration. On examination I found the left ear discharging. There was also tenderness and swelling over the mastoid and above the ear. The pulse rate was 120, and the temperature 99.4° F. The patient was moved to the Wallasey Cottage Hospital, and an operation was performed the same night.

The entire mastoid was in a condition of acute osteitis, and there were numerous small collections of pus scattered throughout the mastoid cells. There was also pus in the mastoid antrum. The mastoid was widely opened, a large rubber tube being used for drainage.

The patient was more comfortable after the operation, and for the first five days appeared to be progressing satisfactorily. It was observed, however, that she was rather apathetic and disinclined for food, but no special misgiving was felt on that account. On January 14th she vomited twice and became more lethargic, but there was no headache, and the temperature and pulse were normal. Later in the day she refused food, and during the night passed rapidly into a semi-comatose condition, with occasional meningeal cries. On the following day the coma deepened and she responded to questions in monosyllables only, and after long delay. She lay curled up

on the right side. Incontinence was present. The eyes were half open, fixed, and staring. The pupils were unequal, and the right one was a little irregular in outline. The right eye was very slightly deviated downwards and outwards. There was no nystagmus and no papilloedema. The mouth was open and the tongue dry and furred. The temperature was 97.5°, and the pulse rate 76.

The case appeared to be one of brain abscess, but whether in the cerebrum or cerebellum it was impossible to say. Under ether anaesthesia the temporo-sphenoidal lobe was explored, without pus being found. The more difficult exploration of the cerebellum was then commenced, but before the dura mater of the posterior fossa was exposed, the condition of the patient became so alarming that the operation had to be rapidly terminated.

On January 16th the coma was deeper and the patient could not be roused. Lumbar puncture was performed, and 10 c.cm. of clear cerebro-spinal fluid was withdrawn under pressure. It was decided to make a further attempt to explore the cerebellum. A general anaesthetic being out of the question, 1/6 of a grain of morphine was given hypodermically, and the operation of the previous day was resumed as the patient lay in bed. An area of dura mater, the size of a sixpence, in front of the lateral sinus, was at length exposed. The dura was incised and a knife inserted into the cerebellum to a depth of one inch in different directions, but without result. A Record syringe with a stout needle was then tried, and at a depth of one and a half inches one drachm of pus was withdrawn. A small rubber tube, the width of a slate pencil, was used for draining the brain abscess. It was stitched to the skin to prevent displacement.

The withdrawal of pus was immediately followed by deeper breathing. The coma lifted slowly and consciousness was not fully restored for two days. Incontinence continued for nearly a week. When the patient was sufficiently conscious to co-operate in the eye tests, the changes observed were as follows: (1) nystagmus, on movement of the eyes to the left; (2) conjugate deviation of both eyes to the right, in the normal resting position; (3) diplopia, the right eye was inclined downwards and outwards; (4) the eyes had a fixed, staring appearance, spontaneous movements were very limited, and the patient was unable to move the eyes together in different directions and hold them there. She did not experience any