the bowel below the division of the duodenum. Duodenal obstruction was produced by capping the cannula and preventing leakage about it, after which replacement was made by the subcutaneous injection of Ringer's or sodium chloride solutions. Following periods of obstruction of 71½ to 93½ hours the animals were either sacrificed or the duodenal cannula was opened and the accumulated fluid allowed to drain for a period of time. This fluid plus the vomitus was then prorated over the period of the obstruction. In all 3 animals the average secretion during the period of obstruction was slightly less than that found during the control period (Table I).

TABLE I.

Dog No.	Before Obstruction		After Obstruction	
	cc. per hr.	Total hrs.	cc. per hr.	Total hrs.
39	36.5	59	34	71.5
17	34.4	72	30.7	93
46	45.25	36	43	93.5

In certain other experiments we observed that during periods of free drainage, the secretion is greater when the juices are returned than when they are discarded and replacements are made by salt solution. We feel, therefore, that the second group reflects more exactly the usual condition, and that we must assume therefrom that simple duodenal obstruction does not result in a significant increase of the combined secretion of digestive juices above the fasting level.

7374 C

Psittacosis in the Native Australian Budgerigars.

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Soon after the discovery of latent psittacosis in the breeding establishments of California the sanitation of the aviaries became a paramount problem. A proposal was made to destroy the flocks which were proven to be infected and to resume breeding in the disinfected pens with a native stock imported from Australia. Such a plan might be feasible only provided the native birds should be free from disease. According to reliable reports no psittacosis has been observed or reported from the Australian continent. It was thus

anticipated that the budgerigars would possess a high susceptibility to the virus and would therefore lend themselves for exposure and immunity tests.

Permission was obtained to export 200 shell parrakeets through the port of Sydney. The birds had been trapped during the month of June in the vicinity of Adelaide a few days before they left the country. They had no contact with other birds either on land or at sea. On their arrival in 8 large cages, in July, 1933, the green, partly mature and partly immature budgerigars appeared perfectly healthy and remained so under strict isolation in a special room in a separate building removed from any psittacosis work which was in progress.

One month after arrival one bird died in Cage 4. The autopsy was inconclusive but mice inoculated with the spleen and liver suspensions sickened on the 5th to the 7th day and when sacrificed presented enlarged spleens. Aerobic and anaerobic cultures remained sterile. By passage experiments and filtration tests a virus was obtained which produced the typical lesions in mice and infected rice birds by inoculation and *per os*. The inclusion bodies (L. C. L. bodies) were readily demonstrated. Following this unexpected discovery of the psittacosis virus 10 birds each from Cages 3, 4, 5, 6, 7, and 8 were sacrificed and examined by mouse inoculation tests. In 8 shell parrakeets the virus was present in the spleens and livers and in the nasal mucosa also of one other bird. (Table I.) The

TABLE I.
Latent Psittacosis Infections in Australian Shell Parrakeets.

Cage	No. Examined	Anatomically Suspicious	Age	Virus Demonstrated	Size of Spleen
1	2	0	Mature	0	
2	2 (1 died)	0	"	0	
3	10	1 (7x5 mm.)	Early mature	2 (passage)	2.5x2.5x2.5x3
4	14	8	3 "	6 (nasal mucosa)	2.5x3; 4x3.5; 4.5x5
	1 dead	1	12 immature	1	5x5; 3x3; 4.5x3
5	10	1 (4x4 mm.)	All mature	0	
	1 dead	0	——	1	
6	10	1 (4x4.5 mm.)	Mature	0	
7	10	1 (4x4.5 mm.)	9 "	0	
			1 immature		
8	10	0	Mature	0	

highest percentage was found in Cage 4, which housed the majority of immature birds. Only one mature budgerigar (female, Cage 3) presented the anatomical lesions of psittacosis in the form of an enlarged spleen and liver. However, the virus was not found in the tissues. In fact, contrary to the experience gathered on Cali-

fornia parrakeets, it was noted that the size of the spleen is not a definite criterion for a latent infection. Moderately enlarged (2.5 to 5 mm.) spleens classified according to California standards as normal were infectious. Cage 5 furnished non-infected birds but 5 months after the test one parrakeet died and was found to harbor the virus. In order to test the infectiousness of the birds in Cage No. 4 with the highest incidence of latent disease, 4 ricebirds were exposed for 58 days. The birds remained well although 2 of the Australian parrakeets were proven to be carriers. While absolute proof is lacking, it is reasonable to believe that the imported birds should be classed as closed carriers.

The infective agent when first secured was relatively weak. The mice inoculated with the spleens became definitely sick around the 10th to the 14th day. Few died but when sacrificed at the time of the illness or on the 20th day after clinical recovery the virus was invariably demonstrated in the spleen by inoculation and by the presence of L. C. L. bodies in the impression smears from the organs. On the 4th passage the virulence was slightly enhanced and rice birds injected with 1 cc. of a 20% suspension of mouse spleen succumbed on the 6th, 8th, 13th, and 19th days respectively. A total of 11 mouse passages was made within 23 weeks (7 California viruses were simultaneously passaged 10 times in 13 weeks). California raised immature shell parrakeets were readily infected with the Australian virus. Some died in from 6 to 50 days and conveyed the disease to others which were exposed in the same cage.

Incomplete cross-protection tests suggest a close relationship to the California virus. In view of the existence of latent psittacosis it was anticipated that the susceptibility of the Australian parrakeets would be low. Two fatal and 6 carrier infections were produced in 8 Australian budgerigars which had been injected with a ricebird passage virus fatal for mice in a dilution of 10^{-5} on the 11th day. On the 57th day 2 carriers yielded the virus on the nasal mucosa and 3 in the contents of the cloaca. Thirty-nine shell parrakeets from 5 different cages were then injected intramuscularly with ½ cc. of a 20% mouse spleens California parrakeet passage virus which killed mice in dilutions of 10^{-7} on the 12th day (Table II). Of 10 deaths 6 were conclusively attributed to the psittacosis infection; 2 birds died from relapses on the 74th and 98th days although the anatomical lesions were suggestive of an acute process. Only 8 of the 29 surviving parrakeets were proven carriers. The recovery rate during the period (37 to 146 days) was 73%. This figure corresponds closely to that of 77% established in a similar

TABLE II.
Susceptibility of Australian Parrakeets to California Virus.

Cage No.	No. Birds	Deaths	Carriers	Non-infected
1	8	2 (9th and 25th days)	6 (57th day)	0
	Exposure 4	1 (106th day)	0	3 (128th day)
	" 2	1 (25th day)	—	1 (25th ")
3	6	2 (21st and 74th days)		
		1 (negative 40th day)	—	3 (83rd ")
4	6	1 (36th day)	—	3 (83rd ")
		2 (negative 36th day)	—	
5	10	1 (48th day relapse)	1 (49th ")	2 (49th ")
		7x7.5 mm.	1 (83rd ")	2 (83rd ")
				3 (146th ")
6	7	1 (98th day)	1 (79th ")	2 (79th ")
		8x11 mm.		3 (83rd ")
7	10	1 (14th day)	3 (37th ")	2 (37th ")
		1 (negative 104th day)	1 (83rd ")	1 (83rd ")
			1 (146th ")	
	53	10 positive 4 negative (26%)	14 (27%)	24 (47%)

experiment on mature California budgerigars from an infected aviary. In exposure experiments in which 6 parrakeets were brought in contact with sick and dying ricebirds only 2 contracted psittacosis. Twenty-four budgerigars from Cages 1 and 2 mingled for 174 days with a flock of California parrakeets in which the incidence of latent infection was estimated at 50%. None of the exposed birds died and only one revealed at autopsy an enlarged spleen without virus. The nature of this resistance is unknown and since it is not associated with neutralizing antibodies it is difficult to determine whether it is the result of a sub-clinical infection or of maturation immunity. Observations on California shell parrakeets from infected and non-infected breeding establishments indicate a lower recovery rate in the birds from psittacosis-free stock. The differences are sufficiently striking to suggest that a higher recovery rate follows previous contact with the virus.

The demonstration of latent infection together with a high resistance and recovery rate strongly supports the conclusion that psittacosis is an endemic disease of the native Australian parrakeet. The birds examined were closed carriers of a weak virus. California experiences indicate that such infections fail to give rise to human psittacosis. However, it must always be remembered that missed human infections particularly in the owners of aviaries and pet shops are by no means uncommon. In the light of the newer knowledge these groups deserve further scrutiny on the Australian Continent.