BIOLOGY

Locomotion in the Australian Marsupial *Antechinomys*

THE so-called 'jerboa marsupial' of Australia (*Antechinomys*) is widely held to be bipedal and convergent on bipedal saltatory murids or jumping mice (*Notomys*); various species of these occur in identical localities with *Antechinomys* in rather arid parts of Australia from Western Australia to Queensland. This interpretation of locomotion in *Antechinomys* is to be found in most general works on Australian marsupials and, as an example of evolutionary convergence, in zoological text-books[1–4].

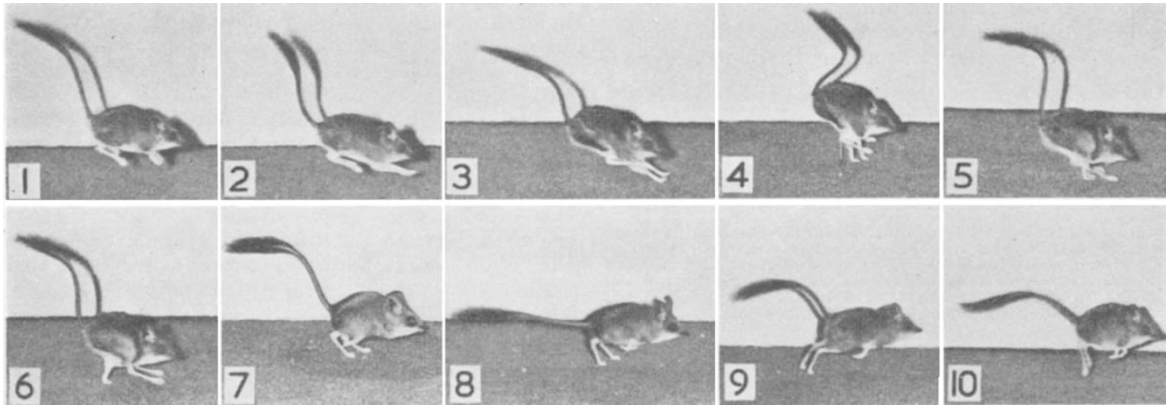

Fig. 1

During the past year I have kept four *Antechinomys spenceri* and one *Notomys richardsoni* in captivity; they are often allowed free to run in my home. Electronic-flash photographs and ciné-film have been taken of them, and these reveal that *Antechinomys* is consistently quadrupedal. Fig. 1 illustrates typical stages in the gallop. The sequence illustrated is made up from selected electronic-flash photographs arranged in the order shown to be natural in cinematographic film taken under similar conditions. This sequence takes from 0·2 to 0·25 sec to complete.

The jumping-mouse, *Notomys richardsoni*, photographed under these conditions is bipedal and saltatory.

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[1] Jones, F. Wood, *The Mammals of South Australia*, Part 1 (Govt. Printer, Adelaide, 1923).
[2] Troughton, E. Le G., *Furred Animals of Australia*, seventh ed. (Angus and Robertson, 1962).
[3] Parker and Haswell, *A Textbook of Zoology*, **2**, seventh ed. (Macmillan, 1962).
[4] Troughton, E. Le G., in Keast, A., Crocker, R. L., and Christian, C. S., *Biogeography and Ecology in Australia* (Junk, 1959).

Mechanism of Anaphylactic Death in the Mouse

GUINEA-PIG anaphylaxis has for many years been considered the classical example of anaphylaxis. In this animal, challenged intravenously, anaphylactic shock is believed to be due to liberation of histamine with subsequent contraction of smooth muscle. Contraction of the bronchial musculature completely shuts off the alveoli and prevents exhalation of air, and the animals die of asphyxia. If the shocking dose of antigen is given by the intraperitoneal or subcutaneous route, death in many cases is not due to asphyxia, but occurs slowly (protracted anaphylaxis), and cannot be attributed to respiratory failure[1,2]. In other animals, anaphylactic death has also been associated with a 'selectively' shocked organ. Thus, in the rabbit, the constriction of pulmonary arterioles and extreme dilation of the right heart lead to acute fatal heart failure. In the dog, marked liver congestion seems to be responsible for death[3].

In the mouse, anaphylactic shock is always accompanied by respiratory distress, cyanosis and some degree of œdema. Symptoms associated with smooth muscle contraction are evident, but not striking. Other characteristic symptoms are marked lack of activity and uncoordinated motor response. A mouse which has just died of anaphylaxis has collapsed and unobstructed lungs, a heart that is still beating and an uncongested liver. All the rest of the abdominal organs appear normal, except for a generalized injected and œdematous appearance of the intestines and stomach. Consequently, death of the mouse is not due to obstruction of the lungs or liver or to cardiac failure. The mouse undergoing anaphylaxis does show signs of respiratory distress and anoxia. Within 1 min after antigen injection an intense spasm of the arterioles and venules occurs followed within 5–20 min by a marked relaxation of these vessels and a fall in blood pressure. All the vessels become dilated with slow-moving blood[4]. In the experiments to be reported, mice undergoing anaphylaxis appeared to experience an actual loss in blood volume, and replacement of the blood volume protected them from anaphylactic death.

Experiments were conducted to explain the obvious respiratory distress always observed in mouse anaphylaxis. It is known that the capillaries become strikingly dilated and that circulation is retarded[4]. During anaphylaxis mice are extremely difficult to bleed; little blood can be obtained by cutting the tail veins and arteries or by puncturing the infra-orbital sinus. If the heart of a mouse which has just died of anaphylactic shock is cut open, little bleeding occurs. These observations suggested that a marked loss of effective blood volume has occurred or that dilation of vessels has produced circulatory collapse.

The following experiments were performed to determine the cause of this circulatory failure. Mice were immunized with 0·5 mg of egg albumen in incomplete Freund's adjuvant, and 21 days later the hæmatocrit values were

Table 1. HÆMATOCRIT VALUE CHANGES DURING ACTIVELY INDUCED ANAPHYLAXIS

Mouse No.	Sensitized mice*		Normal mice*	
	Pre-challenge	Post-challenge	Pre-challenge	Post-challenge
1	49	72	51	50
2	45	60	44	45
3	52	70	49	47
4	45	65	46	46
5	52	74	49	46
6	50	73	49	49
7	47	64	48	46
8	48	65	46	46
9	51	67	51	50
10	47	69	51	49
Average	48·6	67·9	48·4	47·4

* All sensitized mice eventually died of shock, while all the normal controls survived without symptoms of anaphylaxis.