time after the injection of labelled chyle at zero time. The turn-over time of plasma triglycerides was calculated to be 17.3 $\pm$ 2.7 min. The concentration of plasma triglycerides is 3.7 $\pm$ 0.2 μmoles triglyceride per ml plasma. The plasma volume is 4.3 $\pm$ 0.3 ml per 100 g body weight. The turn-over rate of plasma triglycerides was computed to be 2.8 $\pm$ 0.7 μmoles triglycerides per min. The injected chyle contained about 2 μmoles triglyceride per animal.

Discussion. The estimation of the turn-over rate of plasma triglycerides by the tracer technique is based upon the following assumptions: first, that the plasma triglycerides are regarded as a homogenous metabolic pool, second, that the turn-over of the labelled chyle triglyceride is representative of the entire plasma triglyceride fraction. These assumptions are surely not strictly correct, but are in accordance with the results obtained by other investigators[8–10].

We estimated the turn-over rate of plasma triglycerides of pregnant rats to be 2.8 μmoles triglyceride per min per 300 g body weight. In non-pregnant rats the values for the turn-over rate of plasma triglycerides range from 0.5 (HAUDE[6]) to 0.9 (BAKER[8]) to 2.1 (LAURELL[9]) to 3.5 (BELFRAGE[7]); the data are expressed as μmoles triglyceride per min standardized to 300 g body weight.

If we compare these data, we see that the rate for pregnant rats exceeds the rates for non-pregnant rats, except the rate determined by BELFRAGE[7], which is slightly higher. We must consider, however, that the rate determined by BELFRAGE represents the maximal removing capacity of plasma triglycerides, and that this rate was found under unsteady state conditions, because 124 mg chylomicron lipid was injected. The injection of such an amount of triglycerides increased the pool by more than one magnitude of order!

It seemed to us, therefore, that a fall (compared with non-pregnant rats) in the rate of plasma triglyceride removal from the circulation could not explain the hypertriglyceridaemia of pregnancy. Our results support the hypothesis of NAISMITH[11] and RICHARDSON[1] that a rise in hepatic lipogenesis is responsible for the hypertriglyceridaemia of pregnancy.

Zusammenfassung. Die Umsatzrate der Plasmatriglyceride trächtiger Ratten beträgt 2,8 μMol/min/300 g Körpergewicht. Da die Umsatzrate nichtträchtiger Tiere kleiner ist, sprechen die Befunde gegen eine Störung des Abbaues der Plasmatriglyceride während der Gravidität.

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Respiratory Alkalosis in a Panting Lizard (*Sauromalus obesus*)

When the desert lizard *Sauromalus obesus* is exposed to temperatures between 43°C and 45°C, respiratory ventilation and respiratory evaporative water loss increase dramatically[1]. This respiratory response appears to be thermoregulatory (panting) and is apparently mediated by both peripheral and central components of the nervous system[2]. The increase in evaporative cooling during panting is sufficient to keep deep body temperature and brain temperature below an ambient temperature of 45°C for extended periods of time and has a greater cooling effect on the brain than on the remainder of the body[3].

The ventilation necessary to support such significant evaporative cooling is greater than that required to satisfy metabolic demands for oxygen. If the additional ventilation passes over the gas exchange surface of the lungs, a reduction in the P_{CO_2} of the blood should occur, resulting in respiratory alkalosis. This situation is complicated by the fact that lizards, as well as other poikilotherms, regulate pH at a temperature-dependent set point. It appears that they regulate towards a constant alkalinity with respect to the neutral point of water which changes with temperature[4–11]. The purpose of this note is to establish whether the drive for temperature regulation through evaporative cooling or the maintenance of acid-base balance is the dominant regulatory mechanism during panting in the desert lizard *Sauromalus obesus*.

Materials and methods. The animals employed in these experiments were two specimens of a group of lizards which had been previously used to establish the nonpanting relationship between pH and P_{CO_2} of carotid blood and body temperature[11]. Polyethylene catheters were inserted into the right carotid artery and the animals were maintained at 40°C for 3 days. Carotid blood was collected in heparinized capillary tubes directly from the catheter. Carotid blood P_{CO_2} and pH

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were immediately determined with Radiometer microelectrodes. The animals were then quickly transferred to a regulated temperature cabinet at 44 °C. After 4 h of panting, blood was again collected from the carotid catheter and pH and P_{CO_2} measured immediately.

Results. Figure 1 shows the relationship between arterial P_{CO_2} and body temperature (solid line) in *Sauromalus obesus* at non-panting temperatures. By extrapolation (dotted line), one would predict the P_{CO_2} to be about 45 torr in an animal not panting at a body temperature of 44 °C. The actual P_{CO_2} of the panting lizards at 44 °C ranged between 18 and 20 torr (closed circles in Figure 1). The P_{CO_2} of these same animals not panting at a body temperature of 40 °C ranged between 37 and 41 torr. Similarly, the extrapolation of the non-panting relationship (dotted line Figure 2) to a body temperature of 44 °C predicts a pH of 7.06. The carotid pH of panting animals ranged between 7.29 and 7.36 (closed circles in Figure 2). The pH was between 7.15 and 7.17 in animals not panting at a body temperature of 40 °C.

Discussion. When the body temperature of the turtle *Pseudemys scripta elegans* and the lizard *Iguana iguana* is increased, the ratio of respiratory minute volume to oxygen uptake (convective requirement) decreases. As a result of this ventilatory adjustment with increasing body temperature, blood P_{CO_2} increases, blood pH decreases and a constant OH^-/H^+ ratio of the blood is maintained[12, 13].

CRAWFORD and KAMPE[1] have shown that the lizard *Sauromalus obesus* decreases tidal volume and increases respiratory rate dramatically when the body temperature is increased from 40 to 43.5 °C. The tidal volume decreases from 1.46 ml to 0.47 ml and the respiratory rate increases from about 13 breaths/min to about 65 breaths/min. This leads to a 2-fold increase in ventilation. We interprete the decrease in P_{CO_2} and increase in blood pH in *Sauromalus obesus* after 4 h of panting to be the result of a substantial increase in the ratio of respiratory minute volume to oxygen uptake. However, if the increase in pulmonary ventilation relative to oxygen uptake is primarily the result of increased dead space ventilation, little change in blood P_{CO_2} or pH would be expected. Therefore it appears that, when *Sauromalus obesus* pants, a considerable portion of the increased ventilation passes over the gas exchange surface of the lungs which is equivalent to alveolar ventilation in mammals.

At all non-panting temperatures, *Sauromalus obesus* maintained an OH^-/H^+ ratio of 6:1[11]. After 4 h of panting, the ratio was 18:1. According to interpretation of HOWELL[6], this increase constitutes an alkalosis. We conclude, therefore, that prolonged bouts of panting in *Sauromalus obesus* results in respiratory hypocapnia and alkalosis. A similar alkalosis occurs in mammals[14–17] and birds[18,19] subjected to heat stress. It appears that in panting animals under heat stress the demand for respiratory cooling over-rides the ventilatory drive usually associated with acid-base regulation[20].

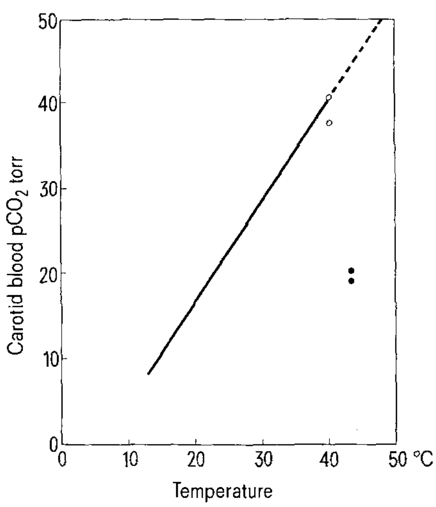

Fig. 1. The P_{CO_2} of carotid blood of 2 specimens of *Sauromalus obesus*. The solid line represents the relationship between P_{CO_2} and body temperature[11]. Open circles are P_{CO_2} at 40 °C and not panting; closed circles are P_{CO_2} at 44 °C after 4 h of panting.

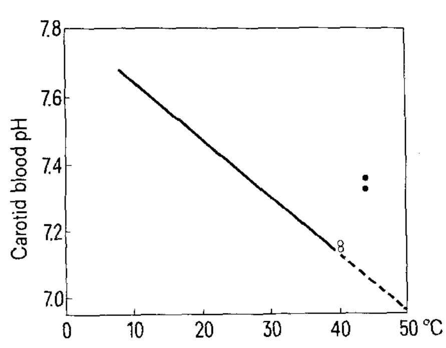

Fig. 2. The pH of carotid blood of 2 specimens of *Sauromalus obesus*. The solid line represents the relationship between pH and body temperature[11]. Open circles are pH at 40 °C and not panting; closed circles are pH after 4 h of panting.

Zusammenfassung. Steigerung der Körpertemperatur der Wüsteneidechse *Sauromalus obesus* von 40 °C auf 44 °C erzeugte Wärmehecheln mit respiratorischer Alkalose im arteriellen Blut. Die Versuche zeigen, dass der wärmeregulatorische Atemantrieb denjenigen zur Aufrechterhaltung des Säure-Basen-Gleichgewichtes übersteigt.

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