Cerebrospinal Fluid pH, pCO_2 and Bicarbonate of the Conscious Dog, Under Exposure to High Ambient Temperature

In a previous investigation (SIEMON et al.[1]) we demonstrated that during panting of awake dogs, the blood gases can be altered in the direction to alcalosis. Furthermore it was shown (PLESCHKA[2]) that in contrary to conditions at normal body temperature, at elevated body temperatures respiratory alcalosis stimulates rather than depresses ventilation. Consequently it was reasonable to look for alteration of cerebrospinal fluid (CSF) which might explain these results.

Methods. During short anaesthesia with Thiogenal® a polythene catheter was placed into one femoral artery. In subsequent surgical procedures the membrana atlanto-occipitalis was exposed by cutting the muscles of the neck in the midline. To prevent pain, a local anaesthetic (Hostacain®) was repeatedly injected into the dissected muscles. 1 h after recovery from anaesthesia (the dogs at this time could run about freely) the following procedure was made. First we took an anaerobic sample of arterial blood at neutral ambient temperature of 24 °C. At the same time we made a puncture into the cisterna cerebello-medullaris, taking an anaerobic sample of nearly 2 ml CSF. The pCO_2 and the pH were immediately measured directly by electrodes (Radiometer Copenhagen). The bicarbonate of the blood and of the CSF was determined by the manometric method of VAN SLYKE. In addition respiratory rate and body temperature were measured. After these determinations at neutral ambient temperature, the dogs were brought into a climate chamber with a constant high ambient temperature of 36 °C and normal humidity. At the onset and at the maximum of thermally induced panting, the described measurements were made again.

Results. In the Table the mean values of all measurements are shown. Comparing the acid-base parameters for arterial blood and CSF one can see that at neutral temperature as well as at high ambient temperature the arterial blood pCO_2 is always lower than the CSF pCO_2, while arterial blood pH is always higher than the CSF pH. According to this pattern the CSF bicarbonate is higher than that of arterial blood. It is also remarkable that CSF pH and pCO_2 alterations are parallel to the blood gas alterations. We therefore have to take into account that during panting not only the blood gases but also the acid-base state of CSF is altered to respiratory alcalosis.

Conclusion. From the results of present experiments, the following conclusions may be drawn: (1) There is no change of pH, pCO_2 or bicarbonate of the CSF which might explain the ventilatory drive during panting, inspite of the alcalosis prevailing in the arterial blood. (2) Since alterations of the acid-base state of CSF and blood during panting parallel each other, it is possible that both influence the effector mechanisms of the temperature regulation[3].

Zusammenfassung. An 13 wachen Hunden wurde bei normaler und bei erhöhter Umgebungstemperatur der Kohlensäurepartialdruck, die Wasserstoffionenkonzentration und der Bikarbonatgehalt des arteriellen Blutes und des Liquor cerebrospinalis gemessen. Danach ergab sich, dass diese Werte sowohl im arteriellen Blut als auch im Liquor cerebrospinalis während der Wärmepolypnoe Änderungen im Sinne einer respiratorischen Alkalose aufzeigen.

Period		Control		Onset of panting		Maximum of panting	
ϑU (°C)		24		36		36	
		$\bar{x}$	S.D.	$\bar{x}$	S.D.	$\bar{x}$	S.D.
ϑR (°C)		39.4	± 0.7	40.0	± 0.7	40.1	± 0.6
f (min^{-1})		35	±21.7	186	±84.7	277	±81.0
pCO_2 (Torr)	blood	35.8	± 5.6	35.7	± 7.7	31.6	± 9.2
	CSF	41.1	± 2.6	39.9	± 3.6	36.0	± 5.8
pH	blood	7.382	± 0.035	7.397	± 0.038	7.432	± 0.081
	CSF	7.331	± 0.031	7.344	± 0.039	7.376	± 0.068
HCO_3^- (m*M*/l)	blood	20.1	± 2.7	19.8	± 2.7	19.6	± 3.3
	CSF	22.3	± 1.3	21.8	± 1.3	20.8	± 1.6

Rectal temperature (ϑR), respiratory rate (f), CO_2-pressure (pCO_2), pH and bicarbonate (HCO_3^-) in relation to ambient temperature (ϑU). $\bar{x}$, mean values of 13 experiments; S.D. standard deviation.

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[2] K. PLESCHKA, Pflügers Arch. ges. Physiol. *308*, 357 (1969).

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