of which the second was performed in the absence of oxygen with glucose present (filled circles) or without glucose (open circles).

1. *Control efflux*. During the first 60 min of efflux, in the presence of oxygen, the rate coefficient decreased fairly rapidly (10–20 min) to a steady state value in Purkinje fibres and cat papillary muscle. In guinea-pig, however, the rate coefficient continued to decrease during the whole efflux period. This deviation from single exponential kinetics may have different reasons (cells of different diameters or different permeability, complex extracellular or intracellular compartments) and complicates the quantitative estimation of changes occurring during hypoxia in this preparation.

2. *Hypoxia*. In all experiments hypoxia leads to an increase in the rate coefficient: the mean of the rate coefficients during the 60 min hypoxic period differs significantly ($p < 0.05$; paired t-test) from the control value (mean of the rate coefficients 30 min before and 30 min after hypoxia). The maximal effect is already reached 20 to 30 min after the onset of hypoxia, after which a gradual decline in the rate coefficient occurs. The sensitivity of the ^{42}K-efflux to hypoxia is larger for papillary muscles than for Purkinje fibres. It is known that the electrical effects of oxygen lack are also less pronounced in Purkinje fibres[2]. In cat papillary muscle, the increase in ^{42}K-efflux was 22% and 37% respectively in the pres-

ence and absence of glucose; corresponding values were 18% and 24% in guinea-pig ventricle and 8% and 19% in bovine Purkinje fibres.

Discussion. The present results indicate that the increase of K-efflux is a general effect of hypoxia on cardiac cells. Although intracellular K or resting potential were not measured in the present experiments, data from the literature[8-11] indicate that a change in the driving force for K ions is absent or minimal during the first 30 min of hypoxia. The increase observed in K-efflux may therefore be the result of an increase in conductance of the passive K channel. Another possibility to explain the increase in K-efflux has been suggested by HAAS et al.[3]. According to these authors, metabolic inhibition would result in a reversal of an active electrogenic K pump. In order to decide between these two possibilities, measurements of K-efflux, K-influx and conductance under identical conditions would be necessary.

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Production of Temperature Signals in the Peripherally Denervated Spinal Cord of the Dog

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Summary. Selective warming of the spinal cord with ventral and dorsal roots cut can generate panting in the conscious dog.

The spinal cord of mammals contains thermosensitive structures which are linked to the temperature regulating system[1]. In some current models on nervous control of body temperature[2,3], these spinal thermosensitive structures have been thought to act as amplifiers in the afferent and efferent thermoregulatory pathways, the gain of which depends on spinal cord temperature. Thus generation of thermoregulatory responses by thermal stimulation of the spinal cord would require its afferent

or efferent connections to be intact. This hypothesis can be tested by experiments answering the question whether the spinal cord, after being deprived of all peripheral inputs, can generate temperature signals and, by conveying them to supraspinal components of the system, elicit panting whose efferent pathways are not directly influenced by spinal cord temperature.

The experiments were carried out on 3 young Beagle dogs whose body weight was between 6 and 8 kg. Under general anesthesia a laminectomy was performed exposing the lower part of vertebral canal, and ventral and dorsal roots of all spinal segments caudally of Th 10 were bilaterally cut. In dogs 1 and 3 this was done extradurally and in dog 2 intradurally. A U-shaped polyethylene thermode (i.d. 1.14 mm, o.d. 1.57 mm) was placed close to the spinal cord covering all segments caudally of Th 12. By perfusing this thermode with water of suitable temperature, the peripherally denervated lumbar and sacral spinal cord could be selectively heated or cooled. The power transferred to the animal via the thermode, which

Respiratory rate before and during warming of the peripherally denervated spinal cord

a	b	c (°C)	d (°C)	e (min)	f (min^{-1})	g (min^{-1})
1	4	30	46$_{48}$	23	64	96
2	3	30	48$_{52}$	15	22	27
3	7	27	45$_{48}$	35	35	52

a) Animal; b) number of periods; c) air temperature; d) temperature of the water entering the thermodes; e) total length of stimulation periods; f) mean respiratory rate during 5 min before start of stimulation; g) mean respiratory rate during all periods of spinal cord warming.

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was calculated from the flow rate and the difference between inlet and outlet temperature, amounted to 0.005 watt/kg for 1 °C difference between rectal temperature and perfusion temperature. At the most intense levels of stimulation, this could increase mean body temperature at a rate of 0.008 °C/min. The experiments were performed in the conscious animals 3 to 6 days after surgery.

The Figure shows an experiment in dog 1 at an air temperature of + 30 °C. 3 periods of spinal cord warming were performed by perfusing the thermode with water of
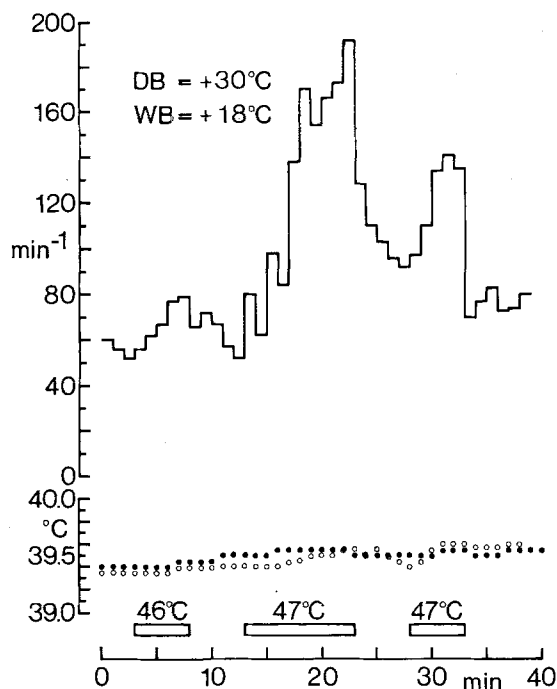
Selective warming of the peripherally denervated spinal cord in a conscious dog and its effect on respiratory rate (above). Filled circles: Rectal temperature. Open circles: Temperature in the vertebrate canal at the level of the lowest intact segment. Bars and numbers: Stimulation periods and temperature of the water used to perfuse the thermodes.

46–47 °C. In all warming periods, clear increases of respiratory rate up to a peak value of 195 min^{-1} were found. Since rectal temperature and peridural space temperature at the level of the lowest intact segment (Th 10) changed insignificantly[4], this panting response was elicited by signals originating in the peripherally denervated part of the spinal cord. The rapid rate of respiration was accompanied by all the characteristic signs of panting which occur in the intact animal when it is exposed to an external or internal heat load: i.e. increased salivation, opened mouth and protrusion of the tongue. However, in contrast to the typical pattern of response to spinal cord warming in intact dogs[4], the highest levels of respiratory rate were found towards the ends of the stimulation periods. This may have been caused by the paralysis of the lower part of the body, which generally rendered panting difficult and seemed to oppose the thermal drive, when higher respiratory rats were expected.

The Table comprises the results of 14 periods of warming the denervated part of the spinal cord in the 3 animals. In 2 of the 3 animals, the respiratory rate during the control periods was higher than the normal resting values in conscious dogs[5]. This was due to the elevated air temperature, which was chosen to facilitate responses. However, in all cases respiratory rate increased during spinal warming and declined after spinal warming had been finished. This effect was least pronounced in animal 2, in which the spinal roots were cut intradurally.

In conclusion, the experiments have shown that the lumbar and sacral spinal cord, after being deprived of all inputs via dorsal or possibly ventral[6] roots, can still generate temperature signals and convey them to supraspinal centers to produce panting. These findings extend earlier results about the generation of thermoregulatory cold defence responses in dogs[7] and pigeons[8] with dorsal roots cut.

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Fermentative Digestion of Food in the Colobus Monkey, *Colobus polykomos*

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Summary. Fermentation of leafy food occurs in the enlarged saccus gastricus of the colobus monkey with the formation of volatile fatty acid, as in the rumen of ruminant animals. About half of the digestible organic matter and cellulose of the diet is digested in this way.

Although herbivorous mammals do not secrete cellulolytic enzymes, they are able to digest cellulose in their food by subjecting it to microbial fermentation. The microbes are accommodated either in an enlarged compartment of the stomach, as in ruminant animals, or in an enlarged caecum and colon, as in horses. While there are many herbivores that employ gastric fermentation, among the primates only the colobus, langur and proboscis monkeys, the Colobinae, appear to have adopted this mode of digestion[3,4]. This subfamily lead an arboreal life, feeding mainly on leaves, fruit and seeds. The first part of the stomach, the saccus gastricus, is greatly

enlarged[5] and it is here that food is fermented yielding gases and volatile fatty acids similar to those produced

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