inhibitory synaptic potentials and does not contain type I small intensely fluorescent cells[15] is consistent with noncholinergic, nonadrenergic regulation of ganglion cAMP metabolism[2,4]. Whether or not prostaglandins play a role remains to be determined, but they are good possibilities for causing cyclic nucleotide accumulation during ganglionic activity[6].

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Hibernation in golden hamsters (*Mesocricetus auratus*, W.) exposed to 5% CO_2[1]

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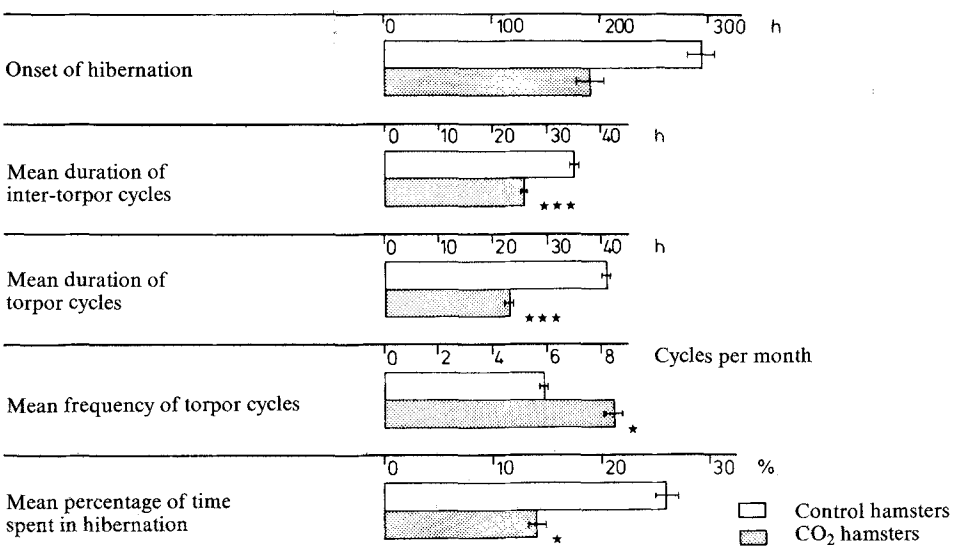
Summary. Chronic exposure of golden hamsters to a gas mixture containing 5% CO_2, 21% O_2, and 74% N_2 favors entry into hibernation. In the hibernating golden hamster, however, chronic CO_2 exposure facilitates arousal.

In the burrows of some hibernating animals the CO_2 concentration may increase considerably, as shown by Williams and Rausch[2] who measured CO_2 concentrations up to 13.5% in semiartificial dens of marmots. High concentrations of CO_2 in the inspiratory air cause a decrease of body temperatures in various nonhibernators and euthermic golden hamsters and have a direct effect on hypothalamic neurons participating in the control of body temperatures, as demonstrated in a previous study[3]. Furthermore, an increase of CO_2 concentration might affect hibernation, as postulated first by Dubois in 1896[4].

The present study in golden hamsters (Mesocricetus auratus, W.) was carried out to elucidate the effect of chronic CO_2 exposure on hibernation.

Material and methods. Experiments were carried out from December, 1981 to April, 1982 in 42 golden hamsters of both sexes weighing 81.4 ± 14.3 g (experimental animals) and 81.1 ± 18.8 g (control animals), respectively. The age of the animals was 21.0 ± 4.6 weeks. The animals were housed in individual cages at an ambient temperature of 5 ± 0.5 °C and an 8:16 h light-dark cycle. 300 ml standard hamster



The results (mean values and standard error) of 42 golden hamsters during 5 experimental months. (* $p < 0.05$; *** $p < 0.001$).

food were offered every 15th day and water was provided ad libitum.

The cages were placed in plexiglas chambers which were perfused with controlled gas mixtures, 50% of the animals (CO_2 hamsters) were exposed to 5% CO_2, 21% O_2, 74% N_2; the control hamsters were breathing air (0.03% CO_2).

Torpor cycles were monitored by continuous measurement of body temperature by means of radio telemetry.

The U-test of Wilcoxon, Mann and Whitney was used for statistical analysis as the data did not follow normal distribution.

Results. The data on the onset of hibernation, i.e. the time from the start of the experiment to the first hibernation bout, showed a high individual variability. On average, however, the CO_2 hamsters started torpor cycles earlier (191 ± 149 h) than the control-hamsters (293 ± 194 h) (fig.). Furthermore, the periods of homoiothermy between two hibernation bouts were shorter in the CO_2 hamsters. The mean duration of these inter-torpor cycles was significantly ($p < 0.001$) shorter in the CO_2 hamsters (26 ± 27 h) than in the control hamsters (35 ± 31 h) (fig.).

A summary of the data obtained during the 5 experimental months shows that the mean duration of torpor cycles was significantly ($p < 0.0001$) shorter in CO_2 hamsters (23 ± 24 h) than in the control hamsters (41 ± 25 h) (fig.). In the CO_2 hamsters 67% (control 29%) of the hibernation bouts were shorter than 20 h and 25% (control 64%) of the torpor cycles had a duration of 30–80 h. Thus, the mean frequency of torpor cycles in the CO_2 hamsters (8.4 cycles per month, SD 3.2) was significantly ($p < 0.05$) higher than in the control hamsters (5.9 cycles per month, SD 2.0) (fig.).

The mean percentage of time spent in hibernation was dependent on the season, with the highest value during January. In every month the control hamsters spent more time in hibernation than the CO_2 hamsters. On average, the CO_2 hamsters spent 14.5% (SD 18.3) of the experimental time in hibernation and the control hamsters 25.1% (SD 20.8) (fig.).

The CO_2 concentration used in the present study did not influence the daily distribution of entrance into and arousal from hibernation. The CO_2 hamsters and the control hamsters did not differ in the duration of entrance- and arousal-time.

Discussion. The finding that the CO_2 hamsters started hibernation earlier than the control-animals and showed significantly shorter inter-torpor cycles indicates that a high CO_2 concentration in the respiratory air favors entry into hibernation. This effect might be related to the observation that in golden hamsters neurons of the thermosensitive preoptic region are inhibited during acute hypercapnia[3]. Additionally, hypercapnia might modify enzymatic pathways, which could lead to a decrease of metabolic rate[5] and thus favor the entry into hibernation.

The results of the present study further show that the torpor cycles of the CO_2 hamsters are significantly shorter than in the control hamsters, i.e. a high CO_2 content in the respiratory air favors arousal. Similar results were reported by Lyman who found that short exposure to CO_2 concentrations above 5% induces the waking process[6]. It seems unlikely, however, that this effect plays a role in the induction of arousal under natural conditions. In the natural burrow of a hibernator the CO_2 concentration will decrease during the torpor bout, since CO_2 loss by diffusion exceeds CO_2 production when the metabolic rate of the hibernating animal is low.

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Adenosine activates a potassium conductance in guinea-pig atrial heart muscle

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Summary. Adenosine shortens the action potential and diminishes the force of contraction in guinea-pig left atria. These effects may be brought about by the activation of a potassium conductance. This assumption is supported by voltage clamp and ^{42}K release experiments.

Adenosine and related compounds exert pronounced effects on the heart; pacemaker activity, force of contraction in the atrium and atrioventricular conduction are depressed (for review, see Burnstock[1]). The effects of adenosine on the heart are very similar to those of acetylcholine, however, they are not blocked by atropine[2]. Studies with adenosine covalently linked to an oligosaccharide have indicated the existence of specific adenosine receptors outside on the cell surface[3]. The subcellular events underlying the actions of adenosine are still poorly understood. Direct membrane effects[4-9] as well as interactions with the sarcolemmal adenylate cyclase[10-12] have been postulated. We report here that adenosine activates a potassium conductance in guinea-pig atrial heart muscle. The evidence for this is derived from electrophysiological experiments and tracer studies with ^{42}K.

Methods and results. Hearts were taken from guinea-pigs and myocardial preparations were mounted for the measurement of the force of contraction (F_c), transmembrane potential and currents, and ^{42}K efflux as described earlier[13-15]. Figure 1a shows the effects of acetylcholine and adenosine on the action potential and F_c in a guinea-pig atrium at maximally effective concentrations. The duration of the action potential was extremely shortened and F_c was almost completely abolished by both substances. In partially depolarized preparations, acetylcholine and adenosine induced large increases in the resting potential. Figure 1b shows the original record of a partially depolarized prep-